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MEDICAL AND SURGICAL.

THE ARTICLES WRITTEN ESPECIALLY FOR THE WORK BY
AMERICAN, BRITISH, AND CANADIAN AUTHORS.

VOL. V.
SUPPLEMENT.

EDITED BY WILLIAM A. EDWARDS, M.D.

ILLUSTRATED.

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PREFACE.

UNTIL this Cyclopædia appeared American Pediatrics was without a standard work in the true sense of the word. There was no work in which was gathered the concensus of the English-speaking Pediatrists. It is true that as early as 1858, in Philadelphia, and 1869, in New York, books were published that have proved to be representative and lasting, but these were the individual contributions of but two writers. It remained then for the Cyclopædia to occupy a place peculiarly its own in pediatric literature. When this work was first thought of Pediatrics was in no way a special department of medicine; indeed, few if any of the colleges had a chair devoted solely to the teaching of diseases of children. That Pediatrics is now fully established as a special department of medicine cannot be gainsaid, and that this work has had much to do with the broadening of the field of Pediatrics cannot be doubted.

In our editorial capacity it has been our endeavor to hold Pediatrics closely allied to general medicine: the majority of sick children are treated by general practitioners, and not by the specialist in the strict sense of the term. As yet the number of men specially devoted to the study and practice of Pediatrics is not large when compared with some other specialties, but no field of medicine can present more conscientious workers or better trained observers. This is particularly noticeable when we consider that Pediatrics as a distinct branch of medicine only began to obtain recognition a few years ago, and that during the last decade some of the most important additions to medical literature here, in England, and on the continent of Europe have related specifically to the diseases of children.

Probably the greatest strides have been those in pathology and etiology. Serum therapy has created an epoch in therapeutics. The results in diphtheria are so brilliant, startling, and life-saving, that the entire world is to be congratulated upon this addition to our armamentarium.

This volume is a continuation of the work which was started as early as 1886 by the lamented Keating and the present editor. From its earliest inception in 1886 until its publication in 1889 the work was shared by

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[illegible] of its, so that it is felt that the supplemental volume will be as nea like its predecessors as it is possible to make it. Each writer is alone responsible for his statements.

The editor desires to thank the contributors for their interest and many instances for kindly advice and encouragement. The work is peculiarly fortunate in that no contributor was removed by death during time of preparation with the single and lamentable exception of the ill trious Pepper, who, fortunately for the readers of this volume, had co [illegible] his manuscript before we were called upon to mourn his irrepara loss.

CORONADO, CALIFORNIA, January, 1899.

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CYCLOPÆDIA

OF THE

DISEASES OF CHILDREN.

CONGENITAL DISORDERS AND DISEASES OF THE NEW-BORN.

By J. W. BALLANTYNE, M.D., F.R.C.P.E., F.R.S.E.

IN this article are gathered together several congenital morbid states of the new-born infant which have not been elsewhere considered under such headings as "Congenital Skin Affections," "Congenital Teeth," "Osteogenesis Imperfecta," "Abnormalities of the Intestine," and "Tumors." The subjects also dealt with in the article on "Diseases of the Fœtus" in volume i. of this work have been avoided, save in those cases in which new and important additions to our knowledge have recently been made.

In considering congenital diseases of the new-born infant it will be convenient first to make some general statements concerning antenatal morbid states, and secondly to discuss in detail certain of the special maladies which may develop *in utero*. In this way a comprehensive view of the whole field of research will be more easy of obtainment.

ANTENATAL PATHOLOGY AND CONGENITAL DISEASE.

The laws which govern the manifestations of antenatal pathology are only now beginning to be understood, and there is still much that is shrouded in mystery. From two directions inquiry is being made into the nature of fœtal disease and death: the steadily increasing interest heredity and its laws is turning the attention of the scientist and thec to the study of antenatal influences; and the stationary condition of population in at least one European country is enhancing the value fœtal life and making its preservation a matter for the consideration of practical physician and the political economist. It may be confider expected that the results of such inquiries will be of service not onl the conservation of antenatal life and health, but also in the elucidation the problems of general pathology and medicine.

Pathological influences may be active during three periods of anten life, and their effects will vary with the time at which they come i operation. They may, for instance, affect the as yet separate reproduct cells, the ovum and the spermatozoon, and impress upon them mor tendencies which show themselves during intra- or extra-uterine life; they may come into play during the *embryonic* stage, before the new organi is provided with functionally differentiated organs, and then their results in all probability purely teratological, *monstrosities*, in contradistinction *diseases*, being produced; or, finally, they may operate during the *fœ* period of antenatal life and cause the evolution of diseases differing on in minor details from those which appear after birth. There is go reason to believe that in all these periods the same morbid causes are work; but the effects differ in so marked a degree because the organis they act upon is so conspicuously different in its structure and functions the pre-embryonic or germinal, embryonic, and fœtal epochs. What pr duces a monstrosity at one time leads to the formation of a disease another; and in this way teratology and fœtal diseases may be regard equally as chapters, and important chapters, in the large subject of gener pathology. I have elsewhere (3)[1] further elaborated this conception antenatal pathology; in the present article my concern is with the fœt maladies rather than the embryonic monstrosities, with antenatal patholog only in that part which most closely resembles postnatal pathology.

Congenital or fœtal diseases (in contradistinction to monstrosities an malformations) differ from adult diseases chiefly in the degree which th morbid process reaches, and this in turn probably depends largely upon th peculiar environmental conditions to which the fœtus is subjected durin intra-uterine life. In the uterine interior the new organism is kept at fairly constant temperature higher than that of the external air, is bathe in a warm liquid medium, is protected from traumatism by the matern structures, and is brought into peculiar and intimate relations with th chemico-vital processes of the mother by means of the placenta. One o the immediate results of the fœtal environment is the possibility of th existence of an amount of disease quite incompatible with the maintenance of extra-uterine life. This characteristic feature of certain antenat morbid states I have termed the *potential mortality of the fœtus* (1); an instance is found in general fœtal dropsy, where the dropsical change is often of a degree much more pronounced than that developed after birth, and yet the fœtus so grossly altered commonly survives till it passes by the act of birth into an environment which does not permit the longer continuance of life. What was possible *in utero* becomes impossible in the extra-uterine state. Further, there exists what may be called a *potential morbidity of the fœtus;* for some organs, such as the lungs, kidneys, and stomach, may be inherently weak, and yet before birth there is no evident sign of disease, but as soon as separation from the maternal economy occurs and a call is made upon these viscera their defective state becomes apparent in disease. The fœtal organs may be very seriously altered, and still life and even health (in a sense) be possible *in utero*, for the placenta performs many of the functions which they afterwards take up. It may, however, be said that the placenta is one of the fœtal organs; this is to a certain extent true; and it may further be said that it is the most vulnerable of the fœtal organs, for placental lesions are quickly provocative of intra-uterine death. The placenta is the great exception to the general potential character of antenatal morbidity and mortality.

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[1] The figures within ( ) refer to the bibliography at the end of the article.

It is an interesting question whether there exist any diseases absolutely peculiar to the fœtus. It is admitted that some maladies—*e.g.*, leukæmia[1]—may in the fœtus reach a degree of intensity not met with in after-life because not compatible with that life; but are there any cases showing pathological characters so peculiar as to constitute special diseases? In a certain sense there are. The fœtal bone-lesions, for instance, are so clearly different from the maladies of the developed skeleton that they are entitled to distinctive appellations and ought not to be loosely spoken of as fœtal rickets. But it must not be forgotten that they are allied also to malformations, for in them pathology is seen affecting structures in a transition state, tissues not yet laid down in their permanent form. Several other morbid states—*e.g.*, so-called fœtal amputations—lie on this border-line between diseases proper and deformities. The safest conclusion seems to be that fœtal diseases, truly so called, differ only in degree from the same maladies in the infant and adult, a difference largely due to the peculiarities of antenatal environment and the preponderating influence of the placenta. What is really peculiar to antenatal pathology is teratology. The cognate question whether there are any diseases with regard to which the fœtus enjoys immunity must, I think, be answered provisionally in the negative. No doubt the unborn infant is well protected from external violence by the structures surrounding it, while the placenta prevents the passage to it of germs and may store up in its own substance mineral poisons (4); but occasionally injuries affect the fœtus, and not infrequently microbes and toxines succeed in forcing the blockade of the placenta or in

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[1] Sänger, M., Arch. f. Gynaek., xxxiii. 198, 1888.

[illegible] gaining an entrance through the amniotic membrane [illegible]

CLASSIFICATION.

[illegible] diseases may be classified in any of the ways used in [illegible] of similar maladies, but for convenience their subdivision [illegible] transmitted, and traumatic may be adopted. In the first g [illegible] morbid states which are provisionally regarded as ari [illegible] independently of the mother's state of health; in the sec [illegible] passed from mother to fœtus; and in the third are [illegible] notably of the nervous system, which are caused by traumat [illegible] during (less commonly before) labor. As our knowledge [illegible] there will be a rearrangement of many of the diseases in the ab [illegible] but for the present we must be content with a provisional group

SOME SPECIAL CONGENITAL DISEASES.[1]

Congenital Anasarca, or General Fœtal Dropsy.—A general drops condition of the fœtus or infant at birth (*vide* Plate I.) is probably t regarded as a symptom of diverse morbid states rather than as a sepa pathological entity, yet in the present state of our knowledge we are c pelled to consider it rather in the latter than in the former sense. It rare occurrence, and is characterized by general anasarca, by the presence fluid effusions in the peritoneal, pleural, and pericardial sacs, and usually œdema of the placenta. The anasarca may be of so advanced a degree as to give the fœtus a truly monstrous appearance, and the effusions in body-cavities may be so great as to cause serious delay in delivery. M of the cases have died during or shortly after birth. The mother of su an infant has usually been a multipara, well advanced in child-bearing li not uncommonly dropsical herself, sometimes suffering from anæm sometimes from renal, hepatic, or cardiac disease, but sometimes in the e joyment of good health. Hydramnios is a common but not an invaria concomitant. Family prevalence has occasionally been noted, and of t eight cases which I have had the unusual opportunity of investigating 9, 10) four occurred in one family, three being the offspring of one wom and one the child of her sister-in-law (8). This observation has the ad tional interest of suggesting a possible paternal factor in causation, and was noteworthy that both the woman and her brother were very anæm The pathogenesis, however, of this morbid state is obscure. Some write have looked for and found a purely fœtal cause in premature closure of t foramen ovale, in aortic stenosis, in absence of the thoracic duct, in cyst degeneration of the kidneys, in a leucocytic infiltration of liver and ki neys, and in fœtal leukæmia; other authors have discovered a matern cause in diseased conditions of the blood (hydræmia, anæmia), in nephriti

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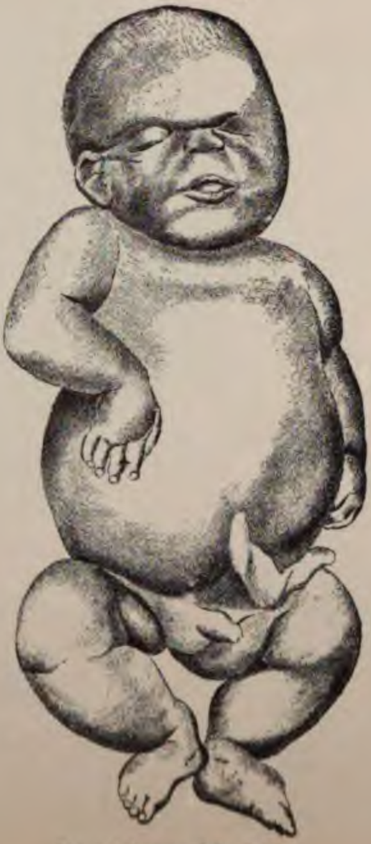
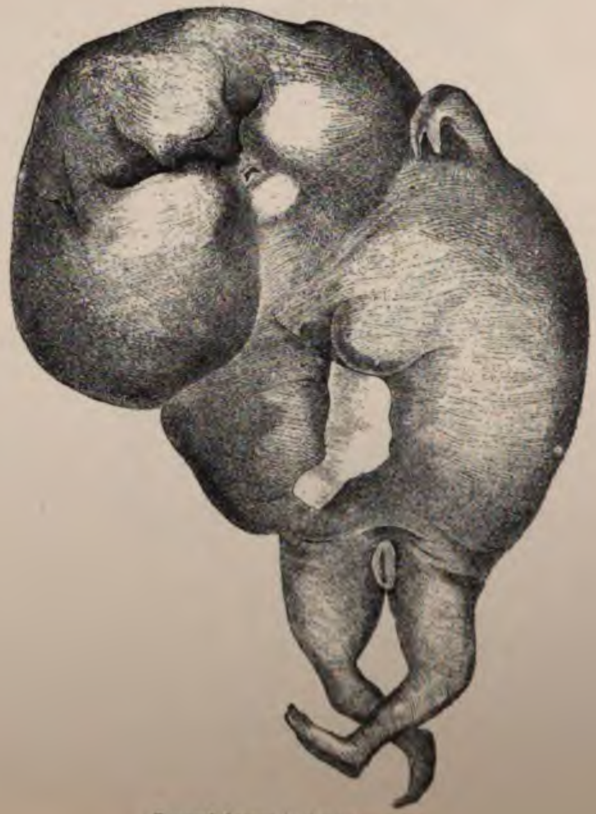
[1] As has already been pointed out, only such congenital diseases as are not fully co sidered elsewhere in this work are dealt with here.

PLATE I.



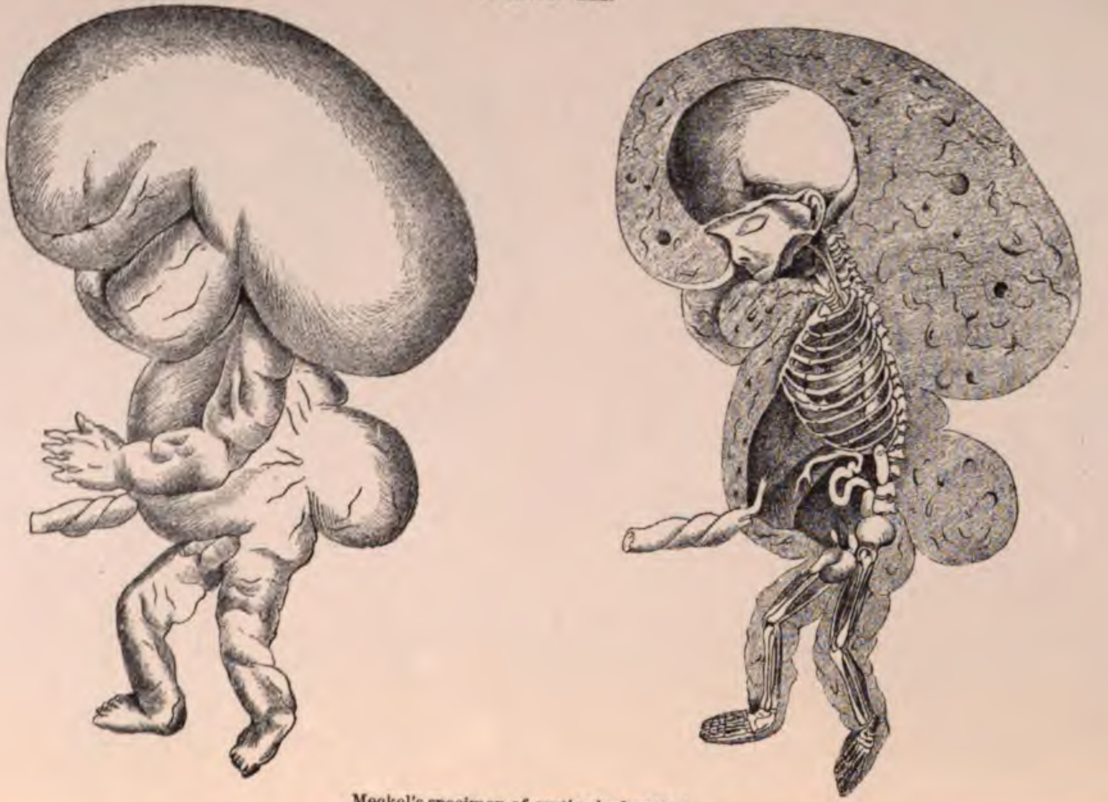
General dropsy of the fœtus.

PLATE II.



General dropsy in the twin-fœtus.

PLATE III.



Meckel's specimen of cystic elephantiasis.

and in malaria. According to Fuhr (7), the first step in the morbid process is the occurrence of endometritis in the mother, leading to placental hyperplasia, to overfilling of the fœtal circulation with resulting obstruction and œdema; the œdema in the placenta itself is due to secondary obstruction in it. The difficulties in accepting any of these theories are great, it being often impossible to distinguish between causes and effects; but it seems clear that the fœtal dropsy is a manifestation of several different pathological processes, and this is quite in keeping with what is known of adult dropsy. In one of my observations the proportion of proteids in the peritoneal effusion was so small as to suggest a blood-dropsy; but that it may also be obstructive or inflammatory in origin seems beyond doubt. In a word, the dropsy is a symptom. The treatment of this malady has not been successful; in the case of one of my observations the mother in a succeeding pregnancy took chlorate of potassium steadily, with the result that the full term of pregnancy was reached, but still a dropsical infant was born. A word may be said here, in passing, regarding the œdematous state in which a malformed twin-fœtus is sometimes found (*vide* Plate II.). This is seen in its most marked form in the allantoido-angiopagous or placental parasitic twins, in which there is an inosculation between the two fœtal circulations in the single placenta. The heart of the one twin atrophies or is absent altogether, and its circulation is carried on, necessarily in an inadequate manner, by that of the other, with the result that œdema ensues. The well-developed twin may show hydramnios, while in the sac of the acardiac or paracephalic dropsical fœtus there is oligo-hydramnios (19).

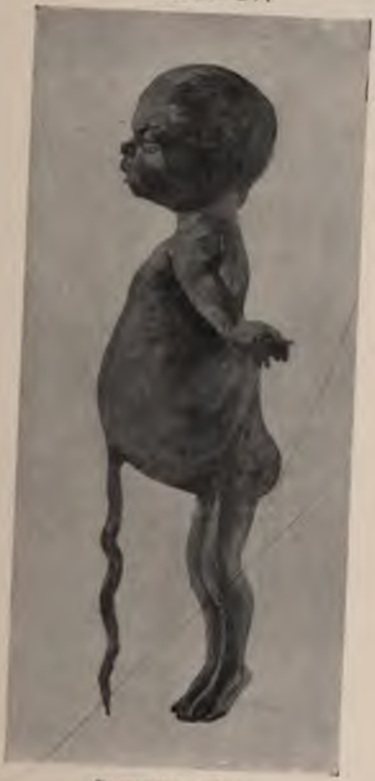
Congenital Cystic Elephantiasis.—This name, with its variants, *elephantiasis congenita mollis* and *hydrops anasarca gelatinosa*, has been given to a disease which in its nature closely resembles general fœtal dropsy, especially that which occurs in the malformed twin. It is characterized by an increase in the subcutaneous tissue and the formation in it of cysts of various sizes with clear serous or curd-like contents. It may affect the whole surface of the body or only a special region,—*e.g.*, the back of the head and neck. The dropsical condition is often gelatinous in its nature, and this peculiarity may be due to the stage of development reached by the subcutaneous tissue when attacked by the morbid process. It is essentially a condition of dilatation or of dilatation and occlusion of lymphatic spaces and vessels, although some have thought that it might be a myxomatous metaplasia of the subcutaneous tissue; and the presence of the large cystic cavities gives to the infant an even more markedly malformed appearance than that seen in general dropsy (*vide* Meckel's specimen, Plate III.). The infant is often born prematurely, and, in the generalized form at any rate, rarely long survives its birth. It is sometimes associated with maternal albuminuria and hydramnios, but these are as likely to be effects as causes of the fœtal condition. The internal organs frequently show grave alterations, such as malformations of the heart, lungs, intestine, liver, and kidneys; but these differ very extensively in the recorded cases, and are

probably accidentally associated states. The real cause of the whole pro
is not yet clear, but in the case of malformed twins it may be confide
anticipated that it resides in the placental relations between the
fœtuses.

Congenital Elephantiasis.—The name "congenital elephantiasis"
been given, without, perhaps, sufficient warrant, to a number of conditi
characterized by more or less localized thickening of the skin and sub
taneous tissues of either a soft or a hard nature (*mollis s. dura*). Sometin
it is associated with subcutaneous fibromata, at other times with more or l
wide-spread areas of nævus-like vascularity. It specially affects the lim
and very closely resembles elephantiasis Arabum as met with in the adu
That it is truly a congenital form of elephantiasis Arabum is held by so
recent writers, and it has been shown to be occasionally hereditary in c
tain families, as in the case reported by Nonne (25). In an interesti
instance, of which I showed a photograph to the Edinburgh Obstetri
Society (32), and which occurred in the practice of Dr. Moncorvo, of R
the mother had several traumatisms during pregnancy, after one of wh
lymphangitis developed, the infant was born with an enlarged right le
and the condition was explicable on the supposition that during the mate
nal lymphangitic attack streptococci passed into the fœtal circulati
through the placenta and set up a lymphangitic process in the leg of t
fœtus. Certainly blood taken from the affected leg after birth show
chains and groups of the streptococci of Fehleisen. In other cases (*e.g.*,[30]
however, no such explanation seems possible, and it would appear that w
must sometimes ascribe the process to the pressure of amniotic bands roun
a limb (31). The malady does not necessarily interfere with extra-uterin
life, and may even be recovered from. It may be treated by elastic com
pressure, electricity, and iodide of potassium, especially if the associatio
with congenital syphilis be suspected.

Congenital Ascites.—Among the fœtal morbid states that lead to dela
in delivery is ascites (*vide* Plate IV.). Sometimes the abdominal distentio
from this cause is excessive, as may be gathered from the fact that in som
of the recorded cases as much as five or six litres of fluid have been foun
in the peritoneal cavity. Usually the infant is still-born, for, even if labo
set in prematurely, the abdominal enlargement is so great as to necessitate operative interference. Recovery has, however, occasionally occurred when the fluid has been drawn off by puncture. Hydramnios is a fairly frequent concomitant condition,—nineteen times in sixty-three cases, according to Fordyce (37). Whilst in the adult ascites is most commonly due to obstruction in the portal circulation and more rarely to peritonitis, the converse is true of the fœtus. The portal circulation is rudimentary until near the full term of antenatal life, and obstruction of it is, therefore, rarely the cause of ascites, but chronic inflammation of the peritoneum is fairly common and is often accompanied by considerable effusion of fluid. Possibly the peritonitis may be occasionally malignant or tubercular in nature, but

PLATE IV.



Congenital ascites.

no such case has yet been put on record. Associated malformations, especially of the genito-urinary organs, are common, and are doubtless due also to the fœtal peritonitis. I have dissected one case in which there was distention of the urinary bladder in addition to the peritonitic ascites, and in this instance there was a possibility that the conditions were gonorrhœal, for the mother died a few days after delivery, from gonorrhœal peritonitis (36).

Fœtal Peritonitis.—In addition to the cases just referred to in which congenital ascites was due to peritonitis, there are others in which the peritoneal inflammation is, at any rate at the time of birth, of the dry type. Such a specimen I showed (45) to the Edinburgh Obstetrical Society some years ago. In it there was a recent dry peritonitis gluing the intestinal coils to each other and to the under surface of the liver, and there were also signs of an older inflammation in the pelvic region, for the right Fallopian tube and broad ligament were firmly adherent to the peritoneal aspect of the cæcum; but there was nothing in the history of the case to explain the origin of this recurring fœtal peritonitis. Doubtless this intra-uterine malady is an important cause in leading to various malformations of the abdominal viscera, although it is also possible that it may be the effect of the malformations in some instances. With regard to the cause of the peritonitis, various opinions have been advanced: that it is always or even often due to syphilis is not the finding of recent investigators; more probably it takes origin in various states, such as traumatism, septic and exanthematous infection transmitted through the maternal organism, and the existence of communications between the intestines and genito-urinary tract and the peritoneal sac. In some instances, as in Palazzi's case (47), no explanation at all is forthcoming.

Congenital Obliteration of the Bile-Ducts.—Among the many pathological results which have been ascribed to fœtal peritonitis must be included the antenatal blocking and obliteration of the bile-ducts, with the development of infantile jaundice, absence from the stools of the typical green meconium, hemorrhages, vomiting, convulsions, and death. J. Thomson (49), who has, more than any other observer, closely studied this process, inclines to the belief that an original malformation of the ducts is the starting-point of the lesion, and there are certainly many facts difficult of explanation by the peritonitis theory. Life may be prolonged up to four or even to six months, but commonly ends in a week or a fortnight.

Distention of the Urinary Bladder.—Cases are on record in which the infant has its advent into the world rendered difficult by an abdominal tumor which is found on dissection to be a distended bladder. In some instances so great is the distention that the rest of the fœtus appears as a mere appendage of the enlarged abdomen. I have met with three cases of vesical distention in new-born infants; in one of these (36) there was a moderate degree of enlargement, with concomitant peritonitis and effusion, and in another (61) there were great distention of the bladder, dilatation of the ureters, hydronephrosis, and an occlusion of the urethra near the meatus. Often the condition is associated with grave anomalies of the genito-urinary organs,—*e.g.*, in Taruffi's specimen (55); but sometimes the only cause would seem to be fœtal peritonitis. In some cases there is hydramnios; but, curiously enough, oligo-hydramnios occurs in others. *Dilatation of the ureters* and *hydronephrosis*, unilateral or bilateral, may be met with apart from bladder-distention, and then depends on some obstruction in the ureter. In this condition life may be prolonged for months and even years, while in the former kind of case it proves fatal in a few days, probably from the effect of the associated malformations.

Congenital Diabetes Mellitus (?).—In 1895 Ludwig (65) reported a case in which grape-sugar was found in the liquor amnii of a diabetic pregnant woman, and, since the fluid was in excess, fœtal diabetes was suggested. Unfortunately for the settlement of the question, the infant was dead-born; but Rossa (66) recently met with a similar case, in which, however, the child survived his birth long enough to have the urine analyzed. In this instance there was no sugar in the infantile urine, although it was present in that of the mother and in her liquor amnii. The occurrence of fœtal diabetes is, therefore, unproved; but the case reported by W. B. Bell (67) in a child aged three months, with diabetic heredity, shows that it is not impossible.

Congenital Pneumonia.—As is well known, a syphilitic infant may be born with a condition of the lungs described as white pneumonia (71); but it is less known that a true septic pneumonia may be present at birth. The infection may pass through the placenta; but it may also gain access to the lungs through the mouth and liquor amnii. In Legry and Dubrisay's case (69) the mother had streptococcic vaginitis, there was premature rupture of the membranes, and the infant died nine hours after birth from pleuro-pneumonia, the same organisms being found in the lungs as in the vaginal secretion. It can scarcely be doubted that here there was direct streptococcic infection through the passage of infected liquor amnii into the fœtal lungs.

Congenital Endocarditis.—Congenital cardiac maladies and anomalies have been fully dealt with by Dr. William Osler in the second volume of this Cyclopædia; but a few words are necessary to chronicle certain recent extensions of our knowledge, more especially with regard to endocarditis. The origin of fœtal endocarditis has always been a vexed question, but Bidone (72) has demonstrated at least one way in which it may arise. He found streptococcic endocarditis in the new-born infant of a woman suffering from erysipelas, and he believes that fœtal infection took place through the placenta. The woman herself developed septic endocarditis and died early in the puerperium. The case is interesting as showing that maternal erysipelas may cause fœtal septic infection; it supports the theory that the streptococci of the two processes are identical. Why there should be a localization of the morbid changes in the cardiac valves, and especially in those of the auriculo-ventricular orifices, does not at present find an evident explanation. It is probable that what has been proved regarding erysipelas is also true in the case of other infectious maternal maladies: the fœtus may be affected by the same disease as the mother (*e.g.*, measles, scarlet fever, diphtheria, etc.), or it may simply take on a form of septic poisoning.

Congenital Typhoid Fever and other Infectious Processes.—As an example of what has just been stated fœtal typhoid fever may be cited. It has recently been demonstrated by several observers (75 to 81) that true typhoid fever may occur in the unborn infant; but Etienne (79) has shown that the typical intestinal lesions are rare, although the presence of the typhoid bacillus is undoubted, while it is more common to find a general septicæmic infection. Probably fœtal typhoid is usually fatal on account of the absence of a localization of the lesions. Chambrelent (81) has proved that the serum of a fœtus with typhoid gives the characteristic reaction (Widal's) just as does that of an adult. Osler (82) has described a case in which typhoid fever in the mother proving fatal during pregnancy seems to have been the cause of a cerebral hemorrhage in the fœtus. The hemorrhage in the centrum ovale was not due to injury in labor, for the mother died undelivered at the sixth month and the fœtus was removed from the uterus post mortem. The bearing of this case upon congenital cerebral paralysis is obvious. It seems clear that we must ascribe a great and varied influence upon the infant to infectious fevers in the pregnant woman, for what has been said of typhoid doubtless holds also for scarlet fever, measles, etc. The experiments of Charrin and Nobécour (83) show that short of actual pathological changes the infants of women who have suffered from tuberculosis, influenza, and fevers are stunted in their growth, as happens also with the young of rabbits who have been injected with bacteria or toxines.

Congenital Tuberculosis.—During the last few years a rapidly increasing literature has demonstrated the correctness of the view that not only is the child born with a distinct predisposition to tuberculosis, but that it is also occasionally present at birth in active form. Hahn (101) gives a useful bibliography of the subject. A very striking case was that reported by Warthin (100), in which there was ectopic pregnancy with tuberculosis of the tubes, placenta, and fœtus. Hochsinger (89) has shown that congenital syphilis and tuberculosis may exist side by side in the same infant, which affords another illustration of the occurrence of mixed infection in the fœtus. It is not known why the tubercle bacilli pass through the placenta in some cases and not in others; but it is natural to ascribe it to the condition of the placental tissue, although the experiments of Charrin and Duclert (103) seem to show that the presence of alcohol in the blood favors the passage of germs. The association of malformations and hereditary tuberculosis is well known; but Hanot (104) makes an interesting suggestion when he states that such a malformation as congenital stenosis of the pulmonary artery may be the sole representative of a tubercular heredity. The anomaly may thus absorb the whole or the greater part of the heredity, and be an ind cation not of tendency to but of immunity from phthisis.

Congenital Prolapsus Uteri.—In conclusion, a word may be said regar ing the rare occurrence of true prolapsus uteri in the infant at birth, c which some seven cases (105 to 110) have recently been reported, and two o which I have examined (109). In all the cases there was also lumbo-sacr spina bifida, and the infant died in a few days or weeks in every instanc It seems necessary to look for an association of causal factors in these case but a nervous influence is undoubted. The fatal issue must be ascribed t the spina bifida, and not to the prolapse. There were usually also ectropio of the rectal mucous membrane and club-feet.

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OSTEOGENESIS IMPERFECTA.

Notwithstanding the accumulation of a large number of observations of cases of congenital bone-disease, and notwithstanding their investigation by competent observers, it is still preferable to employ the somewhat indefinite title given above, viz., *osteogenesis imperfecta*, rather than any of the many names which have been recently proposed. Osteopsathyrosis, chondrodystrophia, periosteal aplasia, achondroplasia, and fœtal rickets are terms which, whilst descriptive enough of certain cases, are quite inappropriate for

[illegible] We have not yet reached the time when a scientific classifica
[illegible] on pathological lines is possible, and we must th
[illegible] a general denomination as *osteogenesis imperf*
[illegible] and used recently by Stilling (1)[1] and oth
[illegible] whether the various conditions met with in
[illegible] are different diseases or simply diffe
[illegible] same disease.

[illegible] stand-point the lesions met with in osteogen
[illegible] In some specimens—*e.g.*, that of Syming
[illegible] the defect is in the endochondral ossification; in otl
[illegible] Muller's [illegible]—it affects the periosteal; but whilst in these c
[illegible] is the leading feature, in other instances, as in one
[illegible] and in a recently reported observation by Ka
[illegible], there is hyperplasia of certain parts of the skeleton. Kaufmann
in his large work describes four kinds of altered growth of cartilage
[illegible] chondrodystrophia malacica; its growth may come to a sta
[illegible] chondrodystrophia hypoplastica; it may grow, but without the f
[illegible] of rows of cells, and so without addition to the length of
bones, or it may grow actively but irregularly, chondrodystrophia hyp
plastica. Its resemblances to true rickets are seeming rather than re
but it must also be borne in mind that occasionally true rachitic chan
are met with at birth (2).

The *clinical* manifestations of osteogenesis imperfecta are less dive
than is its morbid anatomy. There is commonly friability of the bon
with the occurrence of fractures, sometimes very numerous, affecting t
long bones especially, but occasionally also the flat. In both the cases th
I have examined there were numerous solutions of continuity in the lim
bones; but whilst in the second case these were truly fractures, in t
first (Plate I., Figs. 1 and 2) they were rather separations of shaft fro
epiphyses. The stunting in the growth of the limbs (Plate I., Fig. 3)
marked, and doubtless is greatest in the cases in which the morbid proce
began at a very early date in intra-uterine life. Sometimes, as in th
hyperplastic form (Plate I., Figs. 1 and 2), the stunting of the limbs
masked by the enormous proliferation of cartilage in the epiphyses, and i
my first specimen (3) a curious result of the same overgrowth was th
presence of a "tail." (Plate I., Fig. 2.) The head and trunk are com
monly normal in size, but certain peculiarities, such as a deep depression a
the root of the nose and forward bulging of the forehead, due probably t
defective ossification and consequent shortening of the basis cranii, are me
with. Such infants have usually been still-born, but cases are not wantin
in which life has been prolonged for months and even years. The instance
of achondroplasiac dwarfs reported by Porak (4) and J. Thomson (12) full
prove that one at any rate of the forms of osteogenesis imperfecta is no

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[1] The figures within ( ) refer to the bibliography at the end of the article.

PLATE I.

Fig. 1.

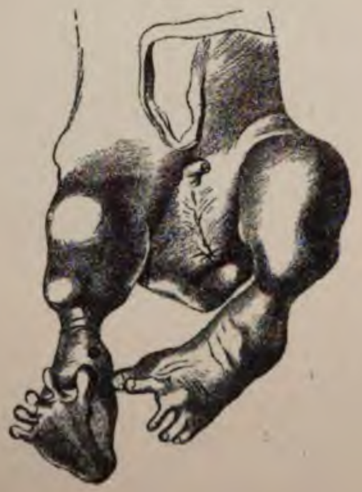


Fig. 2.

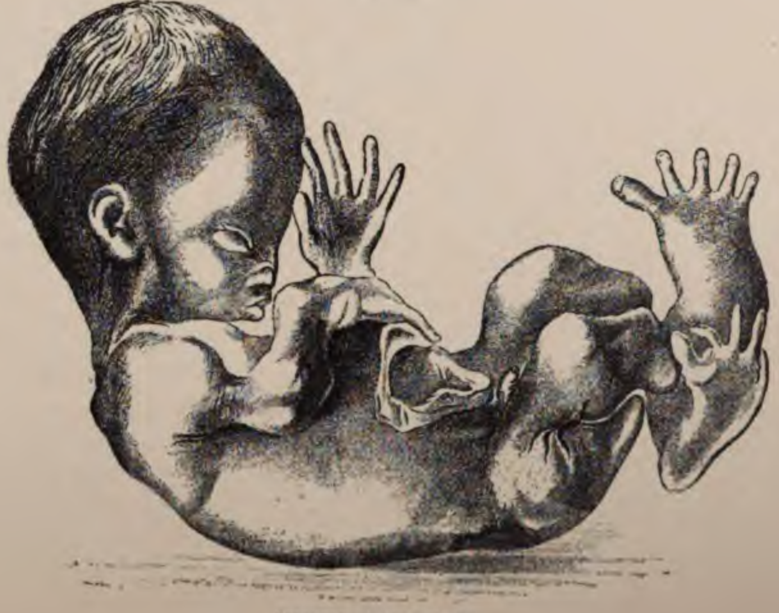
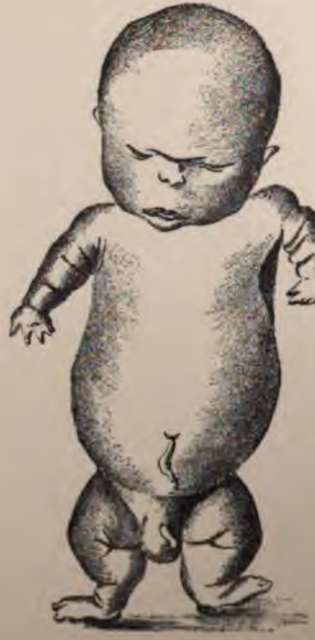


Fig. 3.



Osteogenesis imperfecta.

incompatible with advanced life and physiological activity. The fact, also noted by Porak, that an achondroplasiac mother may give birth to an achondroplasiac infant demonstrates both the possible fertility of the affected individual and the occasional hereditary nature of the disease.

With regard to the causes of these intra-uterine bone-diseases very little is known. A history of maternal privation and traumatism in pregnancy has been once or twice noted, but in the great majority of cases the clinical history is quite unsuggestive. A ray of light is thrown upon the question by some recent experiments of Charrin and Gley (39), who succeeded in producing congenital rickets in a rabbit the offspring of a couple of animals who had been infected with the toxines of diphtheria and blue pus. It may be hazarded that these bone-diseases are among the till recently unsuspected results of infectious processes attacking the organism during or shortly before pregnancy, and that the pathological differences met with are due to the stage in development of the skeleton reached at the time of the action of the pathogenic factor. Possibly if the morbid cause act early enough in intra-uterine life the result may be truly teratological and be represented by such monstrosities as the amelic, phocomelic, and hemimelic, whilst if it is operative later the various types of osteogenesis imperfecta are the consequences.

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CONGENITAL TEETH.

History.—The occasional birth into the world of an infant with teeth already cut was an occurrence well known to the ancients, and we read in Pliny's "Natural History" of a certain Marcus Curius who was on this account surnamed *Dentatus*, while, as Shakespeare well knew, it was commonly believed that Richard III. of England was born with teeth. When, as Weinrichius tells us sometimes happened, the *infantes dentati* were also *barbati* (bearded), no surprise need be felt on learning that the occurrence was regarded as a presage of evil. Far back in the world's history, as the cuneiform inscriptions of Chaldea (6)[1] have revealed to the scholar of to-day, the birth of an infant with teeth already cut betokened long life for the king of the country and success for his armies, but ruin for the home of the toothed infant.

Etiology.—Antenatal cutting of the teeth may be due to various causes. In some instances it would seem that an imperfect development of a tooth, especially a fangless condition, permits its premature extrusion; in other cases the excessive development of the tooth or the too early deposit in it of calcareous matter leads to the same result. The causes, however, may be extra-dental, and may consist in an atrophic state of the gum, in ulceration of that part of it immediately overlying the tooth, or in an intra-follicular inflammation forcing the tooth upward. Possibly several of these factors may be active in the same case. In instances that are doubtless very rare the condition is better described as a displacement, an ectopia of the dental sac, both the tooth and its sac being extra-alveolar. Such a state of matters was reported by Vargas (9) recently in a child which is figured in Plate I.; what was at first regarded as a cut tooth was found to be a tooth in its sac situated outside the gum. Sex would seem to have no influence on the occurrence of congenital teeth.

Pathology.—Prematurely cut teeth are usually incisors, central and in the lower jaw; but in a few instances molars have been noted at birth, and in an infant seen by Thomas there were both incisors and molars to the number of nine. Commonly such teeth are poorly developed; the absence of a root or fang has been often noted, and the enamel has been described as thin and imperfectly formed. In a few recorded instances the teeth have been firmly fixed in the jaw, have been white in color, and have shown a normal state of both dentine and enamel; but in the great majority they were quite movable, rocking to and fro on the gum, lacked the bright ivory appearance of normal teeth, and had a softer feeling than usual. In the cases seen by myself they were thin, discolored scales (10).

Morbid conditions of the dental follicle, of the nature of inflammation, ulceration, and even of gangrene, have occasionally been met with; and prematurely erupted teeth have been in rare instances associated with such

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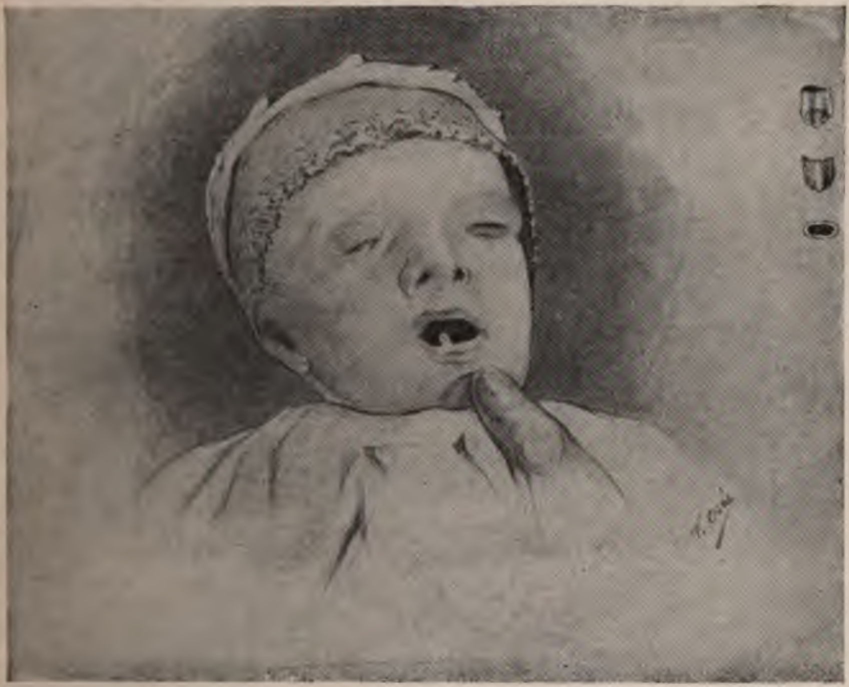
[1] The figures within ( ) refer to the bibliography at the end of the article.

PLATE I



Dr. Vargas's case of extra-alveolar dental sac.

malformations as hare-lip, cleft palate, tongue-tie, and cranial defects. The presence of the tooth has caused an ulceration under the tongue resembling that seen in whooping-cough.

Clinical History.—The history which is usually given in cases of congenital teeth is that at the time of birth the doctor or nurse, having occasion to put a finger in the infant's mouth, discovered the anomaly; or that some hours or days later the mother became aware of the unusual state of matters through the wounding of her nipples by the teeth of the child during nursing. In a remarkable case recently narrated to me by my friend Dr. W. R. Martine, of Haddington, N. B., the infant presented by the face, and the presence of two incisors (one well formed and the other imperfectly developed) was felt by him but not clearly recognized as such during labor: this coincidence of congenital teeth and a face-presentation must be of great rarity.

The pregnancy of which the dentate infant was the result seems generally to have been uneventful, but in a few cases it ended prematurely. In an instance seen by me (10) the mother had influenza shortly before her confinement; and in Martine's case, above referred to, the mother had nursed six persons through scarlet fever in her pregnancy. The inevitable "maternal impression" has been alleged in connection with congenital teeth, and has taken the form of teeth-extraction in pregnancy and of dreams of toothed infants. In a remarkable instance cited by Mattei the infant's mother had also been born with a tooth; and in Limrick's case (5) the woman had two teeth at birth, her second child, a boy, showed the same anomaly, and her sister's first child, a girl, also came into the world with two lower incisor teeth; but in the majority of the recorded examples there is no indication of heredity. A family history of tubercle has occasionally been noted, but the association of this disease with premature development of the teeth has not been clearly established.

Symptomatology.—As has already been stated, congenital teeth are commonly incisors, rarely molars. They may in exceptional instances give rise to no local symptoms, but apparently they act like foreign bodies in the infant's mouth, besides causing injury to the mother's nipples. It is common to read that the little patient was unable to draw the breast properly through imperfect closure of the mouth, and so, getting little milk, began to waste away and was in danger of death. In such cases the removal of the offending teeth usually led to complete recovery, the immediate improvement being by one writer described as "marvellous." In an infant seen by Besnier coryza came on at the age of three weeks; there was frequent sneezing, and as a result the sharp dental borders led to the production of a sublingual ulcer. Sometimes, also, ulcers have been noted on the lips and gums.

It is seldom that the later history of infants born with teeth is forthcoming, so that the general symptomatology is little known. It would seem, however, that the premature teeth, often loosely implanted to begin with, usually fall out or are extracted a short time after birth. In suc instances their places are unfilled until the commencement of the secon dentition; but occasionally the teeth seem to have been supernumerary well as premature, and are replaced by the regular milk-teeth. In oth instances they remain *in situ*, the others of the first dentition growing i around them; in such cases the congenital ones can be recognized by the small size. A very curious after-history was that of an old lady seen b Stocker (1) who had been born with three or four teeth, and who at the a of seventy-five years cut a double tooth! Unfortunately, sufficient recor are not available to establish the association of later anomalies in dentitio with the presence of teeth at birth.

The majority of dentate new-born infants seem to have been delicat and many of them died soon after birth from pneumonia, infantile atroph diarrhœa, etc. In some instances the extraction of the teeth was followe by the disappearance of the dangerous symptoms, but in Magitot's patie their removal was accompanied by hemorrhage, which recurred and ult mately proved fatal. This is, however, the only record of such a disastro result, and probably the infant was a "bleeder."

According to some writers, the presence of congenital teeth is an ind cation of the tubercular tendency; according to others, it is associated wit early closure of cranial sutures and fontanelles; and according to yet other it is accompanied by precocity in intelligence and in development of th body-hair. It is, however, a matter of difficulty to obtain from the recorde cases much support for any of these views.

Diagnosis.—There can be little difficulty in the diagnosis of congenit teeth if the medical man is aware of their occasional occurrence. The may be mistaken for or confounded with a congenital tumor attached to th alveolar border, such as was recently described by Givel (8) and found t be of a sarcomatous or granulomatous nature. Curiously enough, in Givel case the removal of the tumor was followed by the premature eruption of supernumerary incisor tooth.

Prognosis.—Congenital teeth do not, notwithstanding old-fashioned be liefs, give any indication of exceptional mental vigor or the reverse in late life, but through their interference with lactation they may lead to infan tile wasting. In one case their extraction caused fatal hemorrhage, bu the result was practically unique, and probably due to hæmophilia, althoug no hereditary history of that malady could be elicited. In a letter receive recently Dr. W. Atkinson Wood, of Melbourne, tells me of a case (unpub lished) of congenital teeth, in which the teeth sloughed out, the alveola process became necrosed, and the child died.

Treatment.—Where the tooth or teeth give rise to trouble during lac tation, either by wounding the nipple or by preventing complete closur of the mouth, their removal by means of a small pair of forceps is indicated. This is usually easy of accomplishment, for they are loosely placed as a rule, and the danger of hemorrhage from the socket cannot be regarded as other than extremely slight. Where also the teeth rock freely to and fro and are the cause of buccal ulcers, they ought to be extracted; but if they cause no trouble they may best be allowed to remain, for if removed there is always the risk that other temporary teeth may not take their place and so a gap be left until the second dentition. They may fall out of their own accord in some cases, as in one seen by myself, or they may catch upon the clothes and be dragged out forcibly: so it is generally well to extract them at once.

RECENT LITERATURE.

(For literature prior to 1890 *vide* my paper in the *Edinburgh Med. Journ.*, xli. p. 1025, 1896.)

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THE CARE OF THE MOTHER IN PREGNANCY.

BY EDWARD P. DAVIS, A.M., M.D.

THE intelligent care of the mother during pregnancy is not only of great importance to the patient herself, but also goes far to insure the birth of a healthy child.

It has been aptly said that the training of a child should begin with its mother's childhood. The truth of this saying is well recognized by those engaged in the study of pediatrics, and applies with great force to the healthful rearing of children. It is folly to expect weak, ill-nourished, and ill-cared-for women to bring forth healthy and robust children.

We shall best understand how to advise our patients in maintaining a healthful condition during pregnancy if we consider briefly the most frequent variations from the normal which are observed. Earliest in point of time, and usually first in the order of its appearance, comes the familiar phenomenon known as the "morning sickness of pregnancy." This exaggerated reflex, originating in the nerves of the uterus, is transmitted to the abdominal sympathetic, and so to the stomach. It is greatly increased by any abnormality in the shape and consistence of the uterus, or any variation in its position or mobility. In some cases no mechanical cause can be found, and the disorder must be considered as simply a neurosis. For a short time the reflex irritation of the stomach is sufficiently great to interfere with its digestive function, and a transient anæmia is often observed. In healthy women this condition speedily gives place to a plethora sometimes called physiological.

The most important condition in pregnancy, which demands the constant watchfulness of the physician, is the state of the patient's excretions. It is here that a careful study of each woman's peculiarities gives a most satisfactory result. Deficiency in excretion means the gradual accumulation of poisons within the organism, and this influences most profoundly the nervous system, causing in some neuralgia, in others lesions of the skin, while in extreme cases toxæmia and subsequently eclampsia develop. Slight deficiencies in excretion are exceedingly common, and explain many annoying nervous symptoms from which such patients often suffer.

As a consequence of disordered blood-supply and imperfect excretion, the nervous system may present wide variations from a condition of health. Such may take the form of pain, neuralgic, rheumatic, or gouty in character. In others trophic lesions of the skin result, while in some serious disturbances, a disordered state of the special senses, or abnormalities in secretion are at times observed. With abnormal blood-supply and disordered innervation, conditions in the body arise which seem to invite the action of microbes. Hence the acute infections find in pregnant women the most favorable field for the growth of bacteria; the care of the pregnant woman will not be complete unless every safeguard be thrown about her to avoid, if possible, dangerous infection.

The first duty of him who has the patient under his care in early pregnancy is to lessen the disturbances of appetite as promptly and completely as possible. It is of the greatest importance that no exaggerated reflex stimulus, arising from abnormality in the womb, be allowed to interfere with the patient's appetite and assimilation. Should any suspicion be present that the position of the uterus is incorrect, or that erosion of the cervix is present, no time should be lost in thoroughly examining the patient. If necessary, the womb should be placed in the proper position and supported in that way until it shall have risen out of the pelvic cavity. If there be a discharge from the womb, this calls for the exercise of strict cleanliness and for such local treatment as may be necessary. If a very hyperæmic and swollen condition of the cervix be present, this should be remedied by suitable local treatment.

Direct care must be exercised as well as patient watchfulness that the mother has proper food from which to meet the demands upon her strength, in order that the patient's blood-making organs have the proper material from which to derive nourishment: the choice of food, then, during pregnancy is of great importance. During the early months of gestation, when the stomach is most sensitive, small quantities of easily assimilated food must be given. As soon as the patient wakes in the morning, if the desire for emesis be present, it will often be relieved by the expulsion of a small quantity of secretion from the stomach. A short time after this the patient will be able to retain a cup of broth, or a raw egg beaten up with wine, and frequently weak tea or coffee with an abundance of cream. Later, broth, soup, junket, milk-toast, scraped beef sandwich, or gruel will frequently be retained. While milk is a most desirable article of diet, to many it is indigestible and disagreeable; such patients can often take it when diluted with soda, Apollinaris, or Vichy water. The lightest preparations of cocoa are especially useful as furnishing nutritious matter in a simple form. Milk and lime-water, or milk quickly peptonized without heating, may be taken by some who cannot otherwise assimilate milk.

As pregnancy proceeds and the reflex irritation from the womb is lessened, the appetite becomes in many cases better than the average. The instinctive demand for increased nutrition should be met by abundant food. It is not, however, entirely safe to trust to appetite in the choice of nourishment. Excessively nervous patients, or those who have some disorder of the gastric functions, may develop abnormal appetites which would lead them to take articles lacking in nutritive value. The so-called "cravings of pregnancy" must not be regarded as natural, but as exaggerations of some pathological habit. In selecting food for a patient after the fourth month of gestation, meat must be ordered sparingly and with careful selection. So-called "red meat" may be in some cases entirely omitted. In others beef and pork may be spared. In most patients mutton and lamb, with the occasional use of beef, are quite sufficient. Preferably the patient should take fish in season, oysters, the white meat of birds, sweetbreads, and lamb-chops to supply her need for meat; such should be broiled, boiled, or baked, and never fried.

The most useful article of diet in pregnancy, and one which many patients instinctively crave, is fresh fruit. A remark of a patient that she could live upon oranges is characteristic of the acceptability of this class of food. The berries are among the least valuable of fruits during pregnancy. Best of all are apples, oranges, grape-fruit, pears, and lemons; the latter should be used in moderation, as they sometimes exaggerate an abnormal secretion of acid in the stomach. Where fruit can be obtained sound and ripe, it is best taken without cooking; but where this is not always possible, fruit not entirely ripe should be baked or stewed. Grapes are often relished by pregnant patients, and with those with whom they agree form a most valuable article of diet. The patient's appetite for fruit may be freely indulged, provided disturbances of digestion do not occur. The reason for the usefulness of fruit is found in the fact that the acids contained in it and also the acid salts fulfil a useful purpose in rendering soluble the waste products of the body. They also promote secretion and excretion, and thus add greatly to the patient's comfort. They form a natural and harmless laxative, and often render unnecessary the use of drugs.

Vegetables should be chosen with care for pregnant patients. Green vegetables are best, while many women are better without potatoes or other starch-containing vegetable. An excess of vegetable diet during pregnancy is not indicated, as the potassium salts found in some vegetables may increase the quantity of these substances in the patient's urine and further the development of irritating compounds. Salads with oil, vinegar, and salt are much relished by many patients. Where they do not disturb digestion they are useful and profitable articles of food. We refer especially to lettuce, tomato, water-cress, and corn salad, and not to the indigestible forms, such as lobster, potato, shrimp, or mixed salads.

Bread is sufficiently important in the dietary of the pregnant woman to demand attention. Flour which contains starch only should be discarded. Graham flour or other unbolted flour should be employed. Bread should not be fresh, and hot bread should not be eaten. In women of vigorous

In choosing beverages for the pregnant woman, her previous habits and history must be considered. It is very rarely necessary to prescribe alcoholic drinks; unfortunate examples of inebriety in which the habit was formed during pregnancy are sufficiently common to warn us in this regard. Where a woman before her pregnancy has been accustomed to take wine or beer, this may be continued in moderation without injury. The free use of tea and coffee during pregnancy is a distinct disadvantage to mother and child. Over-indulgence in tea is more common among pregnant women than is the excessive use of coffee, and frequently aggravates heart-burn and other gastric disturbance. There can be no question that the excessive of both of these substances favors deficient elimination and increases danger of eclampsia. The best of all beverages for these patients is soft n, feebly acid drinks, as very dilute lemonade or buttermilk, or water ining carbonic acid. A pint of water may be taken before retiring, pint in the morning, to great advantage. Many patients experience atest comfort from the free use of Apollinaris, effervescing Vichy, his water in allaying the suffering caused by excessive acidity and orn. Most pregnant women enjoy cool or cold water as a beverage. lly a small amount of hot water taken before meals diminishes the discomfort and indigestion from which these patients suffer. rged with carbonic acid gas serves a most useful purpose in lk more palatable, and thus enabling some patients to take both ter, which is so desirable.

ost forms of cocoa are sometimes relished by patients who beverage with meals. To some this is constipating, which is while others find in cocoa a pleasant substitute for meat. heavy for such patients, and should be avoided.

of a strictly regulated diet for pregnant women is not d. It is not uncommon, in dealing with infants, to obcating appendicitis, from which she made a difficult recovery. Her digestion and assimilation were permanently impaired, and her child, born after this attack, had from birth a profound toxæmia.

Next in importance to the proper selection of food for the pregnant woman is the accurate observation of her excretory processes. The constipation which is so constantly seen in pregnancy predisposes to toxæmia, as does the mechanical obstacle which the enlarged uterus presents to the free discharge of urine. As toxæmia depends upon failure of the action of the kidneys, liver, intestines, lungs, and skin, the physician must assure himself that these special organs are doing each its part in the interest of the patient.

In studying the urine in pregnancy, the amount, specific gravity, percentage of solids and urea present, and microscopic sediment are of first importance. Of secondary value is the presence or absence of serum albumin and traces of sugar. It is usually possible to ascertain the amount of urine passed without serious inconvenience to the patient. This should be systematically recorded at intervals of ten days or two weeks, if possible, during the entire pregnancy. If the specific gravity be taken, and its last two figures multiplied by 2.33, the result will give an approximation of the amount of solid matter, which is a useful clinical index. The percentage of urea should also be determined and similarly recorded. If sediment be present, it should be thoroughly examined for casts or epithelial *débris*. If these precautions be taken and the urine be examined every two weeks during gestation, the number of cases of eclampsia developing will be very greatly diminished. A superficial examination, however, made largely to ascertain the presence of serum albumin, is practically worthless.

To promote the proper action of the kidneys, suitable diet, the free use of water, warm baths at night, and the occasional use of calomel in small but frequent doses are most successful. An absolute milk diet for a few days will often relieve the patient very greatly. But few, however, are willing to continue this, and hence fruit and bread should be added to the milk. If the specific gravity of the urine remains low, and the percentage of urea be less than one and five-tenths, the patient requires strict supervision. A sudden and rapid fall in the excretion of solids calls for active measures and rigid supervision of the diet.

Constipation will be largely obviated if the patient consent to follow such a diet as has been described. For habitual use, cascara and small doses of mineral waters are the best laxatives. Fruit, however, is so far superior that it should be employed, if possible, in place of drugs. The patient's clothing should be carefully regulated, corsets being laid aside, proper waists substituted, and all pressure removed from the abdomen. Evidence of failing action of the liver must be carefully sought, and is cause for the proper use of suitable treatment. The skin should be kept active by daily baths, and if elimination is lacking and the patient's nervous system is irritated and excited by retained toxines, the warm bath at night acts as a most useful sedative. An abundance of fresh air will permit the lungs to do their share in the removal of excrementitious matter.

Attention must be forcibly drawn to the fact that the early symptoms of toxæmia occur in the nervous system, and that many of the minor ailments of pregnancy are not the inevitable consequence of this condition, but are symptoms of mild toxæmia. Such are neuralgias, headache, perversion of temper, sleeplessness, apprehension, and melancholy. The mistake is often made of prescribing bromides and chloral for these conditions, when a thorough study of the patient would reveal the fact that what she needs is increased excretion to restore her to health.

Exercise during pregnancy has an important function in increasing elimination, improving appetite and digestion, and fostering the patient's assimilative power. The best exercise is walking upon a level or driving upon smooth roads. All violent forms of exercise, straining, lifting, climbing of stairs, use of the sewing-machine, and all violent motions, should be omitted during this period. Exercise should preferably be in the open air, and extremes of heat and cold should be carefully avoided.

Equally important for the pregnant woman is abundant rest. Not only should the usual night's rest be taken, but during the day the patient should seek the opportunity to lie down for a short time. In cases of passive distention of the veins in the lower extremities this will assist greatly in preventing the development of a varicose condition. Strict watch should be kept over the patient that no trivial matter be allowed to interfere with her rest.

The nervous system of the pregnant patient has long been considered unduly susceptible to external irritation. Alternations of joy and despondency are not infrequent in this condition, while in those who have a tendency to nervous disorders this liability is greatly increased. There can be no question of the positive duty of those who have the care of pregnant patients to spare them as much as possible all forms of depressing influence. Agitation of every sort, especially sudden shock and fright, is to be carefully avoided. The influence of the latter upon the unborn child has long been a subject of much discussion. There is sufficient evidence to cause us to believe that at certain periods of development the fœtus can be profoundly influenced by a great shock to the mother's nervous system. Children may be born so deformed that the deformity shall distinctly reproduce the object of the mother's fright. While this is denied by some who reason from biological data, still the practitioner of medicine will find abundant examples in the literature of the subject, and may witness such himself, in which at least a marked coincidence is present.

Those women who have a tendency to nervous disorders should be watched over with especial care when in the pregnant state. Thus, a choreic patient, an epileptic, or one who has suffered from intractable neuralgia will usually find her disease aggravated by pregnancy. Those who have frequent obstinate headaches will often suffer more severely w in the pregnant condition. In those who are predisposed to melanch and mental disease, pregnancy may bring about the very trying condit of puerperal mania.

It is of great importance to the comfort of the mother and the w being of her child that measures be taken to secure the freest possi growth of the fœtus and its mechanical accommodation. This requires removal of all constriction from the patient's abdomen, and the arran ment of her clothing in such a way that pressure shall be entirely abs and that exercise may be unconstrained. If the clothing has not been arranged that its weight is supported from the shoulders, it should be adjusted at once. If the patient's abdomen is not pressed upon by clothing, no obstacle to the growth of the fœtus will be present from t source.

MORTALITY OF EARLY LIFE.

By A. BROTHERS, B.S., M.D.

ACCORDING to Tanner, a century ago "the London workhouses presented the almost incredible result of twenty-three deaths in every twenty-four infants under one year of age; this frightful devastation being allowed to go on for a long time almost unnoticed, as it was deemed beyond the reach of remedy." A half-century ago Meissner wrote that "almost in all parts of civilized Europe it has been proved by statistical data that more than one-third, and in some countries nearly one-half, of all births are lost during the first year of life." Coming down to a quarter of a century ago, we find that in England and in the United States about one-fourth of all deaths was made up of children under one year. In 1865 J. L. Smith stated that in New York City from one-third to one-fourth of all the infants born died under the age of five years, and about 1870 Tanner, in England, concluded that one-fourth of the newly-born died before the end of the fifth year.

At the present time, in New York City, more than ten per cent. of newly-born children die before the age of one month. This death-rate corresponds with that of most large European cities. (Eröss.) Nearly one-fourth (twenty-three and eight-tenths per cent.) of all deaths is still made up of children under one year;[1] and if we include still-births at term, the proportion is increased to twenty-seven per cent. Extending the period of life to five years, we find that forty-one and nine-tenths per cent. of all deaths occur under this age. These figures prove conclusively how little advance we have made in the last quarter of a century in controlling the fearful death-rate of our children during the first years of life.

More than fifty thousand children are born annually in New York City. More than six per cent. of the births at term are reported as "still-births." These figures, however, do not begin to express the loss of fœtal life in the earlier months of pregnancy, when the vast majority of cases are not reported. At term, my former studies show that more than fifty per cent. of all still-births can be traced to two causes: 1, protracted labor resulting in asphyxia of the child; 2, compression of the umbilical cor This article does not include infantile mortality occurring during labor, bu it would be well to bear in mind that progress in the art of obstetrics ma in the future, serve to diminish the death-rate in the newly-born. Of on hundred children born alive but dying before the age of five days, mor than one-half succumb to congenital feebleness or prematurity. Of on hundred and fifty deaths under one month, forty-two per cent. were trace to the same causes.[1]

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[1] I am indebted to Drs. M. Cisin and R. S. Tracy for valuable assistance in the preparation of statistics for this article.

ASPHYXIA NEONATORUM.

Normally the child *in utero* is in a condition of apnœa, in which there i an interchange of oxygen and carbonic acid gas. If, previous to birth, a interruption of this process takes place, as from compression of the cord separation of the placenta, etc., an attempt at inspiration ensues, with th aspiration of mucus, blood, and other foreign substances into the air-passages. As no air enters the child's lungs, a condition of "intra-uterin asphyxia" results. If, on the other hand, the child is born normally, bu is unable to initiate the act of inspiration, we are then dealing wit "postnatal asphyxia." This may occur in cases of feebleness from prematurity or certain congenital pathological conditions.

Intra-uterine asphyxia is divided into two grades. In the first grade the reflexes are still present, and the finger introduced into the child's mouth with the object of removing mucus or foreign matters will excite efforts at swallowing or choking. In the treatment of this condition, after cutting the cord a little blood is allowed to escape, and then it is tied. The child is placed in a warm bath at a temperature of about 100° F., and then rubbed dry with warmed napkins. If this does not suffice, cold water is dashed over the baby, and it is again thrust into the warm bath. To these procedures systematically applied—removal of mucus, bath, friction—may be added rhythmical traction of the tongue, flagellation, or other methods of external irritation. These measures will usually revive the infant in a few moments, unless it passes into the second grade of asphyxia.

The second grade is characterized by the absence of the reflexes. In such cases the finger in the child's throat fails to provoke a reaction. Attempts, therefore, to induce respiration by external irritants alone are useless. We must introduce oxygen into the system, so as to excite the medulla. Laborde's method of rhythmical traction of the tongue is again indicated. Or, seizing it by the feet, the child may be suspended a minute with the head downward. The best results, however, after clearing the air-passages of mucus are obtained from the various methods of artificial respiration. Personally I have always favored the method of Silvester, by which the arms are drawn upward and everted, to simulate inspiration, then turned downward, at the same time compressing the thoracic wall, in imitation of the act of expiration. On the Continent the method of

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[1] Infantile Mortality during Childbirth, etc., P. Blakiston, Son & Co., 1895.

Schultze is very much in vogue. In this the child is swung in the air so as to double its body on itself, simulating expiration, and again swung back, so as to hang perpendicularly downward on the operator's fingers, to imitate inspiration. Occasionally catheterization of the trachea or faradization of the phrenic nerve may be substituted. Whatever method is used, it must be alternated with the warm bath and friction.

In postnatal asphyxia we are dealing with premature or delicate children who have not the power to institute respiration. Violent methods of resuscitation, like that of Schultze, must be avoided. In the resuscitation of premature children, after warmly wrapping them up in a blanket, resort may be had to catheterization of the trachea. This may be followed by the warm bath and friction. If these measures fail, a cautious trial of the Silvester method may be made. It must be borne in mind that some of these cases have congenital causes of obstruction—such as malformation of the organs of circulation and respiration—which cannot be overcome.

PREMATURITY.

Under the heading "Prematurity," which usually includes most cases of "congenital debility," are placed fifty-six and eight-tenths per cent. of all deaths occurring under four weeks, and, according to the recent researches of Erõss, ten per cent. of all deaths at all ages. The claim has been made (Gilbert) that thirty-five per cent. of children born at the seventh month and eighty-five per cent. at the eighth month can, with proper care, be saved. With Rotch, I believe these figures to be very much exaggerated in the present state of our art. Still, much can be done. Previous to labor the mother suffering from syphilis, tuberculosis, malaria, or the contagious diseases ought to be under appropriate medical supervision with a view towards protecting the interests of the child *in utero*. During labor the various conditions arising from dystocia (maternal or fœtal) must be properly met with an eye to the welfare of the child. Asphyxia deserves special notice, as it may at times be confounded with conditions suggestive of congenital debility.

Our practical knowledge of the subject of prematurity is very limited and highly unsatisfactory. Three things must be borne in mind in the management of these weaklings. The body temperature must be equal to that in the uterine cavity. Therefore the prematurely born child must be kept in surroundings having a temperature of about 100° F. Secondly, these children must be properly fed. Thirdly, they must be spared all possible injury during handling.

A great deal of ingenuity has been expended in the accomplishment of the first object. In private practice—particularly among the poorer classes—we resort chiefly to cotton packings kept warm by hot bottles and superheated rooms. In institutions the same object is more scientifically accomplished by the use of incubators like those of Tarnier, Winckel, Rotch, Marx, and others. Unfortunately, even with their aid, most of the children are lost because many of them are tainted with congenital disease, and l cause of the impossibility of securing a food which shall be free from t risk of irritating the delicate gastro-intestinal tract.

The feeding of such babies, therefore, is a most important factor in t problem of their preservation. These infants must receive their nouris ment in small quantities, given to them with the aid of special "feeder The most appropriate food is milk modified according to the rules l down by Rotch. When this is not practicable, cow's milk boiled must diluted with barley- or oatmeal-water in proper proportion (about one three).

If the child increases in weight, it is well next to put it to the breast a nursing woman. It is to be remembered, in this connection, that t weight of premature infants of the same age varies at birth. All childr ought to be weighed at regular intervals, so as to note the normal pr gressive increase in weight.

The handling of these babies ought to be intrusted only to a m painstaking and conscientious nurse. The tissues being necessarily fragi it is quite important that the child suffers no unnecessary injury. Wher some authorities (Runge) advise several baths daily, others (Rotch) adv little bathing. If, besides these measures, the baby is kept in a darken room free from noise, we are doing all that our present knowledge justif towards the welfare of these human mites.

PYREXIA OF THE NEWLY-BORN.

Whereas fever in the newly-born may be due to inflammatory cond tions, such as bronchitis, pneumonia, meningitis, etc., we meet with o form in particular which seems to be little known of in the profession an which has been called "inanition fever." In this form of pyrexia— pointed out by McLane and Holt—the baby develops fever simply fro insufficient supply of nourishment. I have observed a case recently which a newly-born infant presented a temperature elevation ranging b tween 101° and 104° F., and which spontaneously subsided on the fu establishment of milk in the mother's breasts. In such cases it is well examine the breasts, and in the absence of a sufficient supply of milk add the bottle or procure an efficient wet-nurse.

The most frequent cause, however, of pyrexia of the newly-born is du to sepsis,—usually emanating from the navel-wound. Eröss found fort three per cent. of all newly-born to be suffering from an elevation of ten perature. Rösing, however, in one hundred observations, found onl twenty-one per cent. of the children to have fever. Doktor, again, foun as many as forty-five per cent. of the cases with fever. In fifty per cen of the cases Eröss has traced the fever directly to the navel-wound. Rösin from his series of observations, reduces this figure to twenty-five per cen By devoting himself personally to the supervision of the daily dressing o the navel-wound, Doktor reduced the proportion of cases to eleven an three-tenths per cent. Keilmann confirms these facts, and believes that the omission of the daily bath still further tends to reduce the proportion of the cases. In this connection it has been pointed out that, among the poor, the bath-tub is also used as a wash-tub for the cleansing of soiled napkins and clothes: so that we ought to keep an eye on this as a possible source of infection during the baby's daily bath.

We can only allude, in passing, to cerebral hemorrhages, traumatisms, melæna, trismus, erysipelas, and a few less frequent conditions, as factors of varying degrees of importance contributing to the death-lists at the earliest stage of life.

DIGESTIVE DISTURBANCES.

During my dispensary practice I found that out of one thousand consecutive children brought for treatment, two hundred and eighty-seven, or nearly one-third, were suffering from digestive disturbances. During the year 1895, two thousand eight hundred and thirty-nine children under five years died in New York City of diarrhœal diseases. As the total number of deaths was eighteen thousand two hundred and twenty-one, we have the amazing proportion of fifteen and five-tenths per cent. attributed to this one group of cases alone. Before the age of two years—when the vast majority of children are affected—more than three-quarters of the deaths (seventy-six and two-tenths per cent.) occur before the end of the first year. The group of diarrhœal diseases include cholera infantum, enteritis, gastritis, diarrhœa, gastro-enteritis, and dysentery.

As stated, the largest number of deaths occurs before the termination of the first year of life. It will be conceded that at this age most experiments in feeding are made with children entirely, or in part, deprived of the breast. Seldom do children kept exclusively at the breast acquire digestive disturbances of a serious nature. It is well known that the "colics" experienced by most infants during the first three months of existence are not of much moment in breast-fed children. It is the effort to substitute for maternal milk some artificial food, by ignorant mothers or nurses, which may be followed by serious disorders of the infant's delicate gastro-intestinal tract.

In my own experience among the poorest classes, children exclusively brought up on the bottle from birth are usually hopeless cases. And even in more intelligent and wealthier surroundings the greatest difficulty is sometimes met with in tiding over the first two years of life. Normally a baby should gain regularly in weight a half-pound each week. At the end of five months it ought to show double the birth-weight. But how often do we meet these puny artificially fed babies several months old, and no larger than when first born! With the first hot spell gastro-enteritis sets in and their little lives are swept out of existence.

During the first year of life these gastro-intestinal disturbances are attributed by the laity to "teething;" during the second year, to the "second summer." As just alluded to, the first set of cases originates in efforts substitute in whole or in part for mother's milk. The second set corr sponds to the usual age at which weaning takes place and during which t child is permanently deprived of the breast. Among the poor and ignora errors in feeding are prevalent at both periods. From five months onwa babies are given "just a taste" of raw fruit, beer, fish, nuts, candies, mea etc., with the presumed object of making them strong. The resulti gastro-enteric disturbance is then usually misinterpreted and attributed dentition, or to a cold, or to the second summer.

INFLAMMATORY DISORDERS OF THE ORGANS OF RESPIRATION.

We shall be able only to consider the two most frequent disorders o the respiratory organs in childhood,—namely, bronchitis and pneumoni The latter condition may be met with in two forms,—broncho-pneumoni and lobar pneumonia. According to Jacobi, the former is encountere twice as frequently as the latter. Lobar pneumonia is now recognized an acute infectious disease, due to a distinct micro-organism. There ca be no question that in the near future a similar bacteriological etiology fo broncho-pneumonia will likewise be established, particularly in diseases lik measles, whooping-cough, and influenza. Still, the prevalence of thes disorders, chiefly during the colder months of the year, must be borne i mind in the consideration of their causation.

In looking over the records of several hundred consecutive cases o children under two years brought to the dispensary for treatment, I fin that thirty-four per cent. were due to these respiratory affections. Pneumonia is, of course, the most serious of these conditions. According t Meigs and Pepper, under the best care the mortality of pneumonia in children less than five years old is over six per cent. In institutions an among the poor I believe the mortality to be much greater. During th first year of life the death-rate from these affections is nearly double that of the second year.

"Taking cold" is a potent factor in the production of these pulmonary disorders. In the near future this will probably be regarded as a means of expression for the lowering of the vital forces by which pathogenic organisms are enabled to effect an entrance into the system. At all events, as the warmer months are responsible for a preponderance of gastro-intestinal diseases, so the colder months show an increased prevalence of respiratory disorders.

By a little intelligent supervision many of these inflammatory affections of the air-tracts and pulmonary tissues can be avoided. Infants and young children must be warmly clad during the colder months. From an early period of life children can be kept out of doors during certain portions of the winter. In dry, clear weather, without blowing winds, even young infants may be sent out daily for a short time, provided they are warmly wrapped in clothes. With increasing age the period out of doors can be lengthened. Night air, damp weather, and strong winds are, in particular, to be avoided. In-doors, young infants must not be permitted to crawl around on uncarpeted floors, and undercurrents of air must be excluded from the nursery. Older children—as is frequently the case among tenement-house children—must not be allowed to run about barefooted in the cold season. Jacobi advises that every child, while well, should be armed against the results of exposure by regular invigorating ablutions and frictions with cold water. In wealthier surroundings most of these precautions can be carried out. It is among the ignorant and poor that preventive measures are most difficult to inculcate.

Usually the largest number of recoveries will follow good nursing and intelligent efforts directed towards maintaining the vital energies.

DISEASES OF THE NERVOUS SYSTEM.

We propose briefly to refer to cases of convulsions, meningitis (simple or tubercular), and cerebro-spinal meningitis. Under two years of age eight hundred and thirty-two deaths out of a total of thirteen thousand eight hundred and thirty-seven—or six per cent.—were attributed to these complaints. They were about equally distributed between the first and second years of life. In certain years, owing to epidemic influences, the mortality is very much increased. During the past three or four years the death-rate from these diseases has been about the same.

A large number of cases with convulsions can be traced to reflex disturbances originating in the gastro-intestinal tract. Only a small proportion—contrary to the views of the public at large—is attributable to dentition. Traumatism is an important etiological factor in many of the cases of meningitis. Epidemic influences account for the prevalence of cases of cerebro-spinal meningitis during certain years. In tubercular meningitis we have, of course, a distinct pathogenic element.

The tubercular form of meningitis is practically hopeless. Still, I have known the diagnosis to be made in a few cases by eminent physicians and the children are alive to-day. Hence every case must be treated, and vigorously, in the hope of an error in diagnosis.

The treatment of every case of convulsions is important. "Every convulsion has its danger from interfering with the cerebral circulation, and must be terminated as quickly as possible." (Jacobi.)

CONTAGIOUS DISEASES.

In looking over the records of private cases during a series of years I find that nearly one-half of all deaths under two years were due to the three contagious diseases measles, scarlet fever, and diphtheria. During the year 1895 more than twenty thousand cases of this group of contagious diseases were reported in New York City. When it is remembered that a vast number of cases are never seen by medical men at all, and that many others are never reported, it will be readily seen why double this number would more nearly express the truth. Although we know that the mild cases are as contagious as those of a more severe character, yet how frequently are such children in the street, at school, or in public vehicles before the period of contagion has elapsed! How seldom does a disease like measles or whooping-cough, once developed in a tenement-house, fail to involve every floor, or even every family on a floor!

Measles.—This disease is very variable in its occurrence, especially when modified by epidemic influences. Thus, the mortality of measles in Munich, according to Möller, during the first and more especially during the second half of the decennium from 1880 to 1890, while almost treble the average for the preceding decennium, declined considerably in the succeeding years. The statistics of the Riverside Hospital for 1889 show a mortality for measles of seven and nine-tenths per cent. In certain epidemics the mortality of this disease, according to Jacobi, may reach thirty-three per cent. When we recall how lightly the public at large regards measles, it is surprising that facts of this sort, based on careful observations on both continents, make so little impression. The truth of the matter is that so many mild cases get well of themselves. But is the laity, as a rule, competent to distinguish between mild and severe cases? Again, it must be remembered that quite a proportion of cases die later of complications and sequelæ long after the original attack of measles has been forgotten.

Recent statistics show that of eleven hundred and fifty children who, under two years of age, died of measles, scarlet fever, or diphtheria, three hundred and five, or more than one-quarter, were credited to measles. These deaths from measles were nearly equally distributed between the first and second years of life. Until the laity can be taught that measles may at times be nearly as fatal as scarlet fever or diphtheria, we cannot hope to diminish infantile mortality from this disease.

Scarlet Fever.—Although the death-rate from measles is very variable, and may for a short time exceed that from scarlatina, in a long series of years the latter is far the more fatal. (Meigs and Pepper.) During the second year of life scarlet fever kills nearly four times as many children as during the first year. The disease is likewise subject to epidemic influences. In New York City, in 1889, there were twelve hundred and forty-two deaths; in 1895 there were four hundred and sixty-eight deaths. During the year 1889, at the Willard Parker and Riverside Hospitals, the mortality reached twelve and three-tenths per cent. Therefore, as in measles, the death-rate is larger in certain years. The disease is less contagious than measles, but more serious, as a rule. The prognosis involves the future as well as the present. Broncho-pneumonia is frequently a fatal complication of measles; nephritis may prove fatal long after an attack of scarlatina.

Diphtheria is one of the most terrible scourges of early life. During the year 1895 more than ten thousand cases were reported in New York City. As large numbers of the mild cases are overlooked or diagnosticated as "follicular tonsillitis," "catarrhal croup," or "simple laryngitis," or are wilfully suppressed, we can readily understand how the disease must have been much more prevalent. In comparing the mortality of diphtheria with that of scarlet fever I find that four times as many deaths are caused by the former disease. Under five years of age the mortality of diphtheria at certain public institutions in New York City at times reaches from forty to fifty per cent. We must bear in mind, however, in considering the statistics of institutions, that these percentages exceed the average because only the most severe cases are sent thither. Mild cases are usually treated at home, even among the very poor.

In 1895, in New York City, nearly two thousand deaths were ascribed to diphtheria or croup. We include the latter condition because fatal croup in childhood, in the vast majority of cases, is synonymous with diphtheria of the larnyx. The milder conditions, known as "catarrhal croup" or "false croup" or "catarrhal laryngitis," are seldom reported to the Bureau of Contagious Diseases.

The prognosis in diphtheria depends to a great extent on the site of the exudate. Hence all statistics of this disease ought to specify distinctly the region involved. Pure tonsillar diphtheria is usually a very mild disease with a good prognosis; about ninety-five per cent. recover. Nasal and laryngeal diphtheria are virulent manifestations of the same disease, which if left to themselves would probably not yield more than from five to ten per cent. of recoveries. The statistics of diphtheria under two years show that thirty per cent. occur during the first and seventy per cent. during the second year of life.

Whooping-Cough.—We cannot close this subject without referring to this universal plague of childhood. According to Jacobi, twenty-five per cent. of all cases under one year of age die. In New York City the records of the Bureau of Vital Statistics show that about five hundred children under two years die annually of pertussis, and that of these sixty-five per cent. are under the age of one year. This ought to prove how serious an ailment whooping-cough may be. The popular notion—often supported by medical advice—that, being a protracted disease, it is not amenable to treatment, is radically wrong. Many children can certainly be saved by judicious, timely, and persistent treatment.

FEEDING IN INFANCY AND EARLY CHILDHOOD—WEANING.

By THOMAS MORGAN ROTCH, M.D.

In the article on Feeding which was published in the first volume of the "Cyclopædia of the Diseases of Children," in 1889, the general principles underlying this important subject were thoroughly dealt with. In the present article the endeavor will be made to supplement the original article and to state only what may be considered as new and progressive. On referring to the original article it will be noticed that the general principles connected with the growth of healthy infants, and the question of feeding directly from the breast, as well as from the bottle, were discussed. There has not been any especial advance made in the subject of breast-feeding during the last eight years. In regard to feeding from the bottle, no marked changes have been made regarding the amounts to be given at the different ages and the intervals of feeding. The subject of artificial foods was thoroughly discussed in the original article, and the decision among those who have made a special study of infant-feeding is that their use should be abandoned as unworthy of the intelligence and of the knowledge of the physician of the present day. On the other hand, the idea of cream mixtures, which, we believe, practically originated with Dr. Frankland, and the formula for which was published by him in the *Manchester Guardian*, February 24, 1854, has been a basis for progress, and it was in this general direction that research and advances in the subject of feeding were for a time carried on. Dr. Frankland's mixture contained about 3.50 of fat, 6.50 of sugar, and 1.66 of proteids. Beidert later pursued the same line of investigation, and Dr. J. F. Meigs, of Philadelphia, for many years fed infants on a mixture of milk, cream, lime-water, water, and milk-sugar. Later, in 1882, Dr. Arthur V. Meigs published a paper on the analyses of milk in which he still further developed the mixture which his father had used by making it in such a way that a chemical analysis showed it to contain fat four per cent., sugar seven per cent., and proteids one per cent. The principle which Dr. A. V. Meigs promulgated was that human breast-milk is practically unvarying, and contains fat four per cent., sugar seven per cent., and proteids one per cent. In the investigations, however, which have been carried on during the last few years certain facts have been conclusively proved which show that Meigs is in error as to the unvarying analysis of human milk. It is in its variety, and not in its stability, that its success lies. The chief advance which has been made in the subject of feeding of infants of late years consists in the scientific modification of cow's milk, making it possible for the physician to think and write in percentages, and to effect the composition of these percentages by means of milk-laboratories. While we still affirm that human breast-milk, when of good quality and containing the percentages adapted to the digestion of the especial infant, is the best food for human beings in the first year of life, yet we have learned to recognize that good human breast-milk covers a much wider range of percentages than was formerly supposed, and that to feed an infant invariably on four per cent. of fat, seven per cent. of sugar, and one per cent. of proteids shows a lack of appreciation of what nature is continually pointing out to us,—namely, that there is a great variety of good human breast-milks, and that while a number of healthy infants may thrive on an equal number of breast-milks varying noticeably in their percentages, yet on interchanging these infants, by giving one the milk which has agreed with another, serious digestive and nutritive disturbances may arise.

The following table shows the truth of this opinion, and has been sustained by the most recent works on human breast-milk analyses, such as that of Adriance:

HUMAN BREAST-MILK ANALYSES.

(Mothers healthy and infants all digesting well and gaining in weight.)
(Harrington.)

CONSTITUENTS.	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	XI.	XII.	XIII	XIV.
Fat	5.16	4.88	4.84	4.37	4.11	3.82	3.80	3.76	3.30	3.16	2.96	2.36	2.09	2.02
Sugar	5.68	6.20	6.10	6.30	5.90	5.70	6.15	6.95	7.30	7.20	5.78	7.10	6.70	6.55
Proteids	4.14	3.71	4.17	3.27	3.71	1.08	3.53	2.04	3.07	1.65	1.91	2.20	1.38	2.12
Mineral matter	0.17	0.19	0.19	0.16	0.21	0.20	0.20	0.14	0.12	0.21	0.12	0.16	0.15	0.15
Total solids	15.15	14.98	15.30	14.10	13.93	10.80	13.68	12.89	13.79	12.22	10.77	11.82	10.32	10.84
Water	84.85	85.02	84.70	85.90	86.07	89.20	86.32	87.11	86.21	87.78	89.23	88.18	89.68	89.16
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00

On examining these analyses it will be observed that the percentage of the proteids varies greatly, and in only one instance approaches one per cent. closely. In a number of these cases, which were all under the writer's supervision, where the infant, which was thriving on one of these milks, was changed to another, it ceased to do well and suffered from acute indigestion. This again opens the question of the various artificial foods, and whether it is worth while to use them. There are only certain food-elements which can be made use of, no matter what food as a whole is demanded. The artificial foods, if well made, should be unvarying. If they vary, we must at once give up any idea of feeding with exact percentages of the different constituents of the food. Even if they do not vary, do we know enough about the exact percentages of these constituents to calculate how, in the completed whole, as mixed for the patient, we can give just such percentages as we deem best for the individual? That is, the elements of the food may be there, but we do not get the combinations in any near approach to exactness.

Our investigations teach us that variety and comparatively exact combinations constitute the true principle of successful feeding and a resulting perfect nutrition. It is better, then, to put aside entirely not alone the uncertain conglomerations of food-elements represented by artificial foods, but also all kinds of routine cream mixtures, and to substitute for these a certain and practical method of modifying the elements of cow's milk to suit the individual needs of the infants we are feeding. This is the only intelligent and practical method of dealing with so difficult a question as malnutrition.

There is very little question that, by the careful mathematical calculations which are made by means of the Walker-Gordon laboratory tables, practically exact combinations of especial percentages of the constituents of food can be made. What is of equal importance is that by these tables we enunciate the great principle in feeding,—namely, that variety to suit the individual digestion can be obtained by a simple prescription of a few figures; also, that an almost unlimited number of different combinations of the constituents of food can be ordered by the physician each in a few minutes, and put up by the laboratory in as short a time. Changes in the percentages of the constituents of milk may be made by home modifications, but not so well as by the laboratory methods. There are those who hold that their own peculiar way of arriving at correct results in home modification is the best, disregarding the fact that to obtain an exact combination of the constituents of the food needs skilful mechanical training, as well as a correct calculation of figures, and that the personal equation of mother and nurse at home is often a serious drawback to the successful mixing of the food, for this mixing cannot be, in the vast majority of cases, as well done by mother and nurse as by the trained clerk in the laboratory. To the success of home modification there are many obstacles which may arise. For instance, on four successive days in New York, at the same reputable dairy, the request was made to give, for purposes of home modification, for a sick infant, an unvarying cream. This cream, obtained from an honest, conservative dealer, was tested each day, with the result of finding a variation in the percentage of fat from 10.13 to 28.34, which, of course, would at once preclude any chance of obtaining an unvarying percentage of fat in the mixture as given to the patient. This uncertainty, from the varying percentages of daily creams, may be expected to arise when the physician endeavors to carry out an exact modification of milk at home. It has been found that the "top milk process" (as it is called) is also very uncertain. Milk set for the raising of cream by gravity, whether in an open pan, a jar, or a Cooley can, will vary with differences in the atmosphere or in the medium in which it is set. With great attention, though, the night or evening milk can be creamed in ten hours, with some degree of regularity in percentage. But this approximate exactness calls for personal attention every fifteen minutes during this time. Open pan and jar setting is uncertain in results, so that when debarred from access to a scientific laboratory the physician cannot hope to attain any degree of exactness by home modification. The percentages of the sugar and the proteids will also vary from day to day in about the same way, though to a less degree than does the fat. A predetermination of the milk each day is indispensable to exactness in home modification, and this is expensive and tedious.

It is exceedingly important to obtain a safe milk for infant-feeding. A safe milk is hard to obtain from the farm or dairy, and is especially hard to obtain in the private stable. A safe milk is a matter of the producer's conscientious work. The veterinarian must be constant in attendance to guard against such a danger as tuberculosis. Typhoid fever, diphtheria, scarlet fever, and other infectious diseases are easily transmitted by rinsing water, by the milker's hands and clothes, and by other common sources of dairy infection. These are common sources of danger, and yet perhaps not so dangerous as more subtle sources which lie outside of the observation of the physician who has charge of the home modification. The rôle which the streptococcus pyogenes plays in infant pathology is very uncertain, and its virulence varies in any given instance. Yet it is beginning to be believed that this coccus becomes highly pathogenic in certain company, and that milk containing these cocci, which are quite commonly present in average dairy milk, becomes at times especially dangerous to the consumer. The experiments of Nivens in one hundred and sixty cases, of Kruger (1890), of Guillebeau in seventy cases, of Karluiske, Escherich, Longard, Adamitz, Stokes, Bullock, Tonarelli, Cérenville, Taval, and especially Booker, all connect severe diarrhœas in infancy and acute gastro-enteritis with the presence of this organism.

Of especial interest in this connection are the observations of Stokes and Clement on a herd of seventy cows who were infected by the hands of a strange milker, who had one of his fingers infected when working on a large dairy farm in York, Pennsylvania. A complete autopsy made on one of these cows showed that there was no general infection, but that the septic process was limited to a somewhat purulent inflammation of the milk-ducts. Dr. Stokes also traced the source of an epidemic of diarrhœa which broke out in a school of seventy girls to milk from cows infected with this local sepsis. These opinions are to be taken for what they are worth in the individual mind of the physician, but the fact cannot be ignored that in nearly all farm, dairy, or private stables the milk produced very commonly contains large numbers of these organisms. The fecal matter from the cow is a constant source of danger, and is present in nearly all the milk of commerce, while a direct pus contamination often occurs.

Taking all these matters into consideration, it may be said that while with great care and perfect supervision home modification may be done with more or less exactness and safety, it entails a large amount of personal responsibility on the part of the physician. On the other hand, when the infant is fed from the laboratory by prescription, both the primal milk employed and the accuracy of the work required in filling the prescription are guaranteed.

The ground which this article is intended to cover comprises what may be called the three nutritive periods of child-life. The first nutritive period comprises the first twelve months of life, during which time the infant thrives best on a combination of fat, milk-sugar, proteids, mineral matter, salts, and water. Towards the close of this period—namely, during the eleventh or twelfth month of life—the amylolytic function of the infant has become almost fully developed. In accordance with the rule regarding the use of the different functions,—namely, that a function should not be taxed before it is developed, but that when its development is almost completed it should be brought into use,—we should in the latter part of the first year begin to use that function of the digestive tract by means of which the amylaceous elements of the food are converted into sugar, and when we begin to add these amylaceous elements to the infant's food we enter upon another period in its feeding. This transitional period can be called the second nutritive period. The third nutritive period has arbitrarily been made to begin at about the thirtieth month of life. At this time the infant's digestive functions have become accustomed to a greater variety of food, and are especially prepared for the digestion of proteids in the form of meat.

FIRST NUTRITIVE PERIOD.

The first nutritive period may be divided into (1) maternal feeding, (2) direct substitute feeding, and (3) indirect substitute feeding. As has been stated, the first and second of these divisions have already been sufficiently dealt with in the original article, and, in fact, no noted advances have been made in them. The third division (indirect substitute feeding) is where the greatest advances have been made.

As so much is continually said about the expense of preparing an infant's food, it is well to state that in the light of our recent experience those physicians who demand a cheap food for infants do more towards injuring the cause of proper food than any other class we meet with. It is the people of moderate means, and even the poor, who most appreciate the advantages of feeding by exact methods, and it is more apt to be the rich, aided and abetted by physicians ignorant of the rudimentary laws of physiology and development, who are continually preaching economy in regard to the most important factor in the problem of perfecting the human race through its nutrition. People of this same class uphold far greater expense in less important branches of medicine. A good thing costs, and should be paid for. Money is well spent in developing in the best way those who must make and use that money in the future. The importance of this self-evident truth should be impressed upon the people by physicians. Antitoxine is not withheld from the man of moderate means because it is expensive; its employment is best for the patient, and he gets it, whatever his means may be. The infant, with its low vitality and its high mortality-rate, should have the best means for reversing these rates in the midst of its struggle for life. Let not the millions spent in charity be given entirely to the treatment of disease. Let some of it go to the cause of prophylaxis in the early periods of development and to the proper management of the nutrition, which in the young will do more to withstand disease, when it comes, than pounds and quarts of drugs.

The question of indirect substitute feeding is reduced practically to some modification of cow's milk, and, as the milk of all animals must be modified for the human infant, it is as easy to deal with cow's milk as with any other. A few words as to the primal milk supply will perhaps be of great use to one who is studying the subject of infant-feeding. First, having decided that the cow is the animal which can best provide us with material for substitute feeding, we must consider whether any especial breed is better adapted than others for accomplishing this purpose. To do this we should first examine chemically and microscopically the elements of the milk of those breeds which can be employed best throughout the civilized world. It has been found that the breeds of cows from the Channel Islands are more liable, when transported from their home to countries where the climate is more severe, to contract diseases, such as tuberculosis, than are the animals represented by the Durham, Devon, Ayrshire, and Holstein breeds. The percentage of fat is decidedly higher and that of the proteids somewhat higher in the Jerseys and Guernseys than in the breeds which have just been spoken of. It is for future research to determine whether there is a qualitative as well as a quantitative difference in the milk of the Jerseys and Guernseys and that of the more common breeds, but at present it would seem wiser in choosing our medium of modification to select the milk of the hardy breeds of cows.

Some of the marks which distinguish the breeds best adapted for infant feeding are:

1. Constitutional vigor.
2. Adaptability to acclimatization.
3. Notable ability to raise their young.
4. Freedom from intense inbreeding.
5. A distinctly emulsified fat in the milk.
6. A preponderance in the fats of the fixed over the volatile glycerides.

A cow whose milk is to be used for purposes of infant-feeding should be properly housed and well cared for, as the domestic cow is an animal peculiarly sensitive to her surroundings, and her product is correspondingly liable to be thrown out of equilibrium. The milk product of a herd of healthy cows is much less liable to the variations so injurious to the infant's digestion than is the milk of any one cow. For cows to be used for the purpose of infant-feeding a barn is needed where each cow shall have at least one thousand cubic feet of fresh air. The food should be kept where it cannot be contaminated. The manure should be as carefully removed from the barn as if it were a human dwelling. Large, dry, sunny exercise yards should always be provided for her. Her food should always be brought to her and selected with great care. Pure water should be provided, and suitable cups or troughs containing pure running water should be in her stall. The bedding should be fresh and free from mould or from any soil productive of bacterial growth.

The feeding of the cows on the farms connected with the milk-laboratories has for its object the production of an even, nutritious, digestible milk and the careful avoidance of over-stimulation of the lacteal secretion. For this purpose a somewhat wider ration than that employed for the production of milk to be used in butter-making, but somewhat narrower than that employed for the production of beef, has been found to be best. The ratio which has been demonstrated to produce the best milk for infant-feeding is the mean between these two,—namely, one nitrogenous part to five and a half or six non-nitrogenous parts. A constant use of this ratio in the combination of many fodders and grains appears to have produced a reasonably large supply of milk of fair richness, but without over-stimulation such as would be shown by a disturbance of function.

The cows must be kept clean by grooming and the necessary washing, the precaution always being taken to rub the moistened parts dry. The milkers should be dressed in sterilized suits and caps. Their hands and arms should be thoroughly scrubbed before milking, and wiped on sterilized towels. The hands in milking should be kept dry. The milk should be drawn into sterilized pails and carried immediately from the barn to the milk-house, which should be a sufficient distance from the barn to be free from odors. Though we cannot prevent some few bacteria getting into the milk during the milking-time, yet it is possible to reduce the number so greatly as to make the milk practically sterile for the purpose of infant-feeding, particularly if the second half of the product of the udder alone is used and milked into sterile tubes. The first half probably contains many bacteria, which, entering from without, have reached the lower portion of the teat.

After the cows are milked the milk should be carried quickly from the cow to the milk-house. To prevent the milkers from going into the milk-room, the milk should be poured by means of a block-tin pipe through the wall of the milk-room into a large tank, which is also the mixer for the milk of the entire herd. In the space of a few minutes, by means of an ice-jacket, the milk is cooled from 33.88° C. (93° F.) to below 4.44° C. (40° F.). This is to remove the heat, which is conducive to bacterial growth, rapidly. The milk, on its way to the tank, passes through a filter composed of four strata of sterilized cotton.

The milk-room is practically clean from a bacteriological stand-point, for the walls and floor are kept wet with clean water and all dust is excluded. The milk is drawn into sterile jars, in which it is transported. The jars are then sealed, packed in ice, and in a few hours delivered at the laboratories where the milk is to be used for substitute-feeding.

After this treatment of the milk, repeated bacteriological examinations made on its arrival at the laboratory have given the uniform result that it has proved to be comparatively sterile, and at times it has contained either no colonies of bacteria, or only one or two.

No antiseptic, without danger to the infant, can be used about the cow, while all the mechanical devices heretofore tried to take the place of manual milking have inevitably tended to impair the lacteal function of the udder.

The experiments on the biology of the milk of the herd used in connection with the Walker-Gordon Laboratory in Boston have been made by Professor Ernst and Dr. Jackson, and the results obtained are shown in the following table. The specimens examined were taken from the mixed milk of the entire milk of the herd:

Bacteriological Examination of Milk from the Entire Herd. Milk showed Six Hours after Milking Sixty-Eight Thousand Colonies.

SPECIMEN.	HEATED TO	MINUTES.	DEVELOPED BACTERIA.
Whole milk	75° C. (167° F.)	10 and 20	0
Modified milk	75° C. (167° F.)	10 and 20	0
Whole milk and modified milk . .	65.55° C. (150° F.)	10 and 20	Numerous

In striking contrast with these results, obtained by experimenting with the entire milking, are some special experiments made on this same milk by Dr. Austin Peters and Dr. A. K. Stone, at Mr. Gordon's suggestion, to decide whether it was possible to obtain a practically sterile milk at any part of the milking. The manner of performing the experiments was as follows:

Dr. Peters was dressed in a freshly boiled white suit and cap, and had his arms and hands thoroughly washed with a 1 to 1000 bichloride of mercury solution. The cow's udder, teats, flanks, sides, groins, and abdomen were washed with the same solution and dried with a freshly boiled cloth. The milking was then done by Dr. Peters into bottles which had been carefully sterilized at the bacteriological laboratory. The result was as follows:

Of the four cows milked for this experiment, and selected without special choice, the bottle marked 1 in each of the following sets of figures in this table represents the milk of the first half of the milking and drawn by the hand of the milker directly into the sterile bottles. Number 2 in each set of figures represents milk drawn through a sterile canula directly into the bottle, while Numbers 3 and 4, respectively, represent milk drawn by hand after more than one-half of the udder had been emptied. A bacteriological examination of the milk in these bottles gave the following results:

	COLONIES.	COLONIES.	COLONIES.	COLONIES.
1	141	167	19	53
2	0	0	1	2
3	0	6	0	2
4				

The results of the examination showed, first, that the milk obtained from the first half of the milking contained a comparatively large number of micrococci and fine bacilli of the same general appearance respectively; secondly, that the milk drawn through the sterile canula was practically sterile, and that the milk drawn in the second half of the milking by hand was so uniformly sterile as to awaken the suspicion that the isolated colonies might have been the result of the manipulation between the "cow and the plate."

These experiments at once provide us with a means of procuring a milk practically sterile but not sterilized. This experiment also seems to prove that the bacteria which are found in cow's milk do not necessarily come from external sources, whether they be of the cow herself or her surroundings, but may also come from some part of the milk tract between the udder and the end of the teat. These conclusions are made with reference to healthy cows.

Infectious mammitis, to some extent, seems clearly to be carried by the hands of the milkers from cow to cow. This also points to the fact that bacteria may find their way to the ducts through the teats.

These experiments are of great practical importance when it is considered that while under certain circumstances it is impossible to obtain the advantages of such a farm as this and the modification of milk by means of laboratory processes, yet it may be of great necessity to the infant, on account of sickness, to be fed with a sterile fresh milk not sterilized. This could, of course, be accomplished on any farm with any cow by means of ordinary care in the milking and by such rules as were carried out by Dr. Peters. The major part of the bacteria present in the milk are such as cause the usual acid fermentation which we recognize in the common souring of milk; but there are many species of bacteria which ought to be prevented from gaining access to the milk, arising from mouldy hay, straw, or fodder, partially decayed roots, and the natural decay of the wood-work of the barn and adjoining buildings. These latter varieties, which are found to be especially inimical to the preparation of substitute foods, cause in some cases the alkaline fermentation and other abnormal conditions of milk. Every barn apparently has its own set of bacteria, and the flora in America do not exactly resemble the analogous European species which have so often been described.

It is very important that certain precautions should be taken to prevent the use of cows which are affected with tuberculosis. It is probable that three per cent. of the cows whose milk is used for food are tuberculous. Where tuberculosis is actively developed in the cow, the disease can usually be detected by a skilled veterinarian by means of the physical examination which is employed in cows. But, as it is a disputed question at present as to when the milk of a tuberculous cow becomes affected, it is wiser to adopt all measures of precaution known to science. Of these measures the one which is most efficacious in detecting even the incipient stages of tuberculosis is that which is used on the farms connected with the laboratories. The cows employed for the production of the primal milk supply for the milk-laboratories have been subjected to the test for the diagnosis of tuberculosis. This is known as the "tuberculin test." The method of making this test is as follows:

At about nine o'clock P.M. the temperature of the cows is taken per rectum with an ordinary clinical thermometer. The temperature in healthy cows may vary from 37.7° to 39.7° C. (100° to 103.5° F.), according to the age, the weather, the condition of pregnancy, or the period of the day. As soon as the temperature of the individual cow is recorded, each one receives a subcutaneous injection of from two to three cubic centimetres of a ten per cent. solution (one cubic centimetre of Koch's tuberculin to nine cubic centimetres of a one-half per cent. solution of carbolic acid in sterilized water), the proportion being adapted to the weight and vigor of the especial cow. This fluid, for convenience and uniformity, is introduced in the upper part of the right shoulder. After an interval of eight hours—that is, at five A.M.—the temperature is again taken per rectum, and this procedure is repeated at intervals of three hours until two P.M. At five A.M. the temperature should in healthy cows be slightly lower than that found on the previous evening. Subsequently the temperature should not rise above that of the first record at nine A.M. No rise in temperature occurs in a cow which is free from any tubercular affection. Where the temperature rises to 41.1° to 42.2° C. (106° to 108° F.), it indicates disease and marks the cow as tuberculous, though even a lower reading sometimes marks the presence of the disease in cows whose normal temperature was low. No water should be given to the cow during the period of the experiment, because it is found that the temperature, as soon as the water reaches the stomach, is lowered to or nearly to normal, according to the amount and temperature of the water. This test is a very delicate one, and records the presence or absence of the slightest tubercular infection, even if the disease has not previously affected the cow in any way which can be detected by an ordinary physical examination. At the point of inoculation there are marked tenderness and heat in cows that are tuberculous for many hours after the conclusion of the test, while in cows that are healthy the skin is not irritated by the use of the syringe. The accuracy of the test is now conceded, and the percentage of mistakes is less than one.

MILK-LABORATORIES.

Allowing that infants need a great variety in their food, and that nature should be copied as closely as possible, we must have some means of preparing a food not only for the many, but for the individual, and when introducing new methods of preparing a substitute we must recognize the necessity of providing for many possibilities. In this age of scientific rational medicine physicians all over the world demand, first, means of saving time, and, secondly, exact methods of work, which in themselves soon become time-savers. In every branch of our art the tendency is growing year by year to systematize the detailed and laborious work of the individual for the common practical use of the profession at large. With this end in view the writer has given his professional assistance to the establishment of the system of milk-laboratories already referred to, where the materials used are clean, sterile, and exact in their percentages. These laboratories have been placed under the control of educated, intelligent men in whom we have the same confidence that we have conceded to the pharmacist, and we can now write directions for an infant's food and send them to these laboratories, just as in the treatment of disease we write our prescriptions for the division of one drug or the combination of several. The means for prescribing a diversity in the elements of milk according to the idiosyncrasy of the digestion we are dealing with are supplied by these laboratories, equipped with special machinery, and controlled by educated milk-modifiers. Bear in mind that the chain of facts in regard to substitute feeding, which has already been referred to in the original article, is important in all its parts, and that if broken the value of the whole chain may be lost. One end of this chain is at the milk farm, in the cow-house, and in the milk-house. It then comes to the laboratory, and we must so manage that its continuation shall extend unbroken to the infant consumer.

The milk-laboratory is intended to stand in the same position to the profession as does the apothecary shop. That is, certain combinations of the constituents of milk written for on a prescription by the physician should be sent to the laboratory, just as he sends his prescriptions for drugs to the apothecary shop. Thus, what we require of the laboratory is not any especial food, or knowledge of infant feeding, but merely to furnish accurately to the patient what the physician has prescribed.

The establishment of these milk-laboratories has marked a new era in preventive medicine, and has made possible the scientific feeding of infants. It is probable also that the medical treatment of the various abnormal conditions arising in infancy is in the future to be largely dietetical rather than by means of drugs.

In Boston in 1891 the first milk-laboratory in the world for the exact modification of milk was established under the writer's supervision, and opened to the public as the Walker-Gordon Laboratory. Physicians, as well as all others who are interested in the welfare of the human race, must acknowledge how much is due to Mr. G. E. Gordon, Mr. George H. Walker, and Mr. J. H. Waterhouse, whose combined efforts made the establishment of milk-laboratories possible. During the last few years the author of this article has been able to test the value of laboratory feeding in many thousands of cases, his experience being controlled in the practical use of this new system of milk-modification by over two thousand physicians. These laboratories have been established already in Boston, New York, Philadelphia, Baltimore, and Chicago, under identical management.

As milk is one of the best means for the cultivation of bacteria, the milk-laboratory should be situated in a healthy locality. It should be as free as possible from contaminating influences, should be kept absolutely clean, and every aseptic precaution against the harboring or development of pathogenic organisms should be employed. From the moment that the milk is delivered from the farm at a temperature of about 4.4° C. (40° F.), it should be watched over and cared for with scientific accuracy during the whole process of the modification which it undergoes in the laboratory. The milk-rooms should be cool and free from dust, and isolated, as far as possible, from all sources of contamination.

There should also be a separate sterilizing room, where the returned packages and all articles received from the homes of the consumers should be directly brought from the street or wagons, and immediately sterilized in an apparatus reserved for this purpose.

The modifying materials used in the laboratory should be carefully kept for use in glass vessels and at a temperature of about 4.4° C. (40° F.), to prevent the growth of bacteria. The reason for this is that milk modified from materials free from bacteria is better for the infant than milk in which the bacteria have been destroyed by heat. Therefore the utmost care is necessary in all parts of the process and in every department of the laboratory.

A special room should be provided for the milk-modifiers who are to put up the mixtures required by each prescription.

The office of the laboratory should be entirely separate from these work-rooms, so that customers coming to leave their orders should not go near the materials used for modification and thus possibly contaminate them.

It is necessary also that all odors should be excluded from the work-rooms, as milk absorbs odors very quickly.

The employees of a laboratory, whether they be in the office or in the work-rooms, should be intelligent and interested in their work, and should be free from disease.

The milk from the farm should be delivered in the milk-room within a few hours from the time of the milking. On its arrival its temperature, which was 4.4° C. (40° F.), should be held by means of ice below 7.22° C. (44° F.). On its arrival it should be immediately placed in tanks of ice-water.

The milk of the laboratories, as a result of the especial manner in which the cows have been fed and cared for, and their selection according to the proper breed, may be said to have an almost uniform percentage of its elements. Even at those times of the year when the percentages of the different elements of milk commonly vary from changes in the pasturage and in the habits and surroundings of the animals, the milk of the cows employed, which have their food supplied to them in stated rations at one time of the year as well as at another, is not subject to the elemental variations which occur in the milk of ordinary cows.

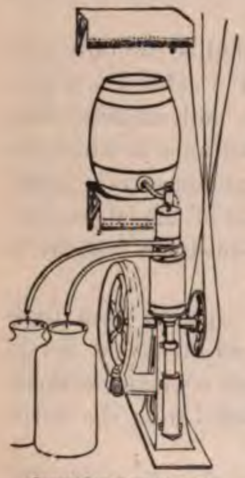
Separating-Room.—The separating-room is arranged and cared for in very much the same way as is the milk-house at the farm. The walls are of white tile, and the ceilings are of material which can be washed and scrubbed. The floor is of asphalt, impenetrable to water, and is kept thoroughly moistened and free from every kind of dust and dirt.

In addition to the precautions against pathogenic germs, which I have already explained, the air of the separating-room is kept fresh and pure by means of a ventilator, consisting of a large steel fan, which revolves at the rate of two thousand times a minute, and by the force of its current carries away any flies or particles of dust which may come within its reach.

In the separating-room is a machine called the centrifugal separator. (Fig. 1.) This separator is made to revolve six thousand eight hundred times a minute, and works with such searching effect on the milk that only a small percentage of fat (0.13) remains in the separated milk.

The utility of the separator, however, does not consist wholly in its absolute withdrawal of the fat from the milk, and in its providing cream as fresh as to time as is the separated whole milk; it accomplishes two other very important results: first, by its great centrifugal force it separates from the cream and separated milk certain particles of foreign matter which are contained in the milk of healthy and clean cows, and thus provides at once a practically clean milk, a most important result from a bacteriological point of view; secondly, the resulting cream contains a certain definite percentage of fat, the importance, of course, of this being in its stability, and not in its special percentage.

Fig. 1.



Centrifugal separator.

There can also be in the separating-room a still for freshly preparing each day distilled water.

The ventilating-fan, the separator, the water-still, and the sterilizer, which will presently be described, can be run by steam apparatus.

Modifying-Room.—In the modifying-room the milk is tested. The materials for preparing the food are brought to this room from the different rooms when needed, and the modification of the milk is here completed.

To be doubly sure that the chemistry of the milk is what we suppose it to be from the uniform nature of the primal milk-supply, we take advantage of the knowledge which we have concerning the changes most likely to take place in certain elements of the milk. The percentage of the proteids, of the sugar, and of the mineral matter in the milk of a herd cared for as just described, and where uniformity in the feeding is the rule, is not apt to be appreciably affected. But the percentage of the fat in individual cows differs from day to day, and thus slightly affects the percentage of the fat in the milk of the herd. The fat, then, being the element by which we know whether each milking gives a uniform product, we test this element by means of what is called the Babcock milk-tester. (Fig. 2.)

The peculiar feature of this method of ascertaining the percentage of fat in milk consists in placing test-bottles containing acidified milk in a centrifugal machine, by the rapid revolution of which the milk is made to separate quickly and completely. The milk is acidified in order that the proteids, casein, and fibrin may be changed to soluble acid albumins, which offer less resistance to the rising and aggregation of the fat-globules.

Approximately equal volumes of milk and commercial sulphuric acid are mixed in a test-bottle with a long graduated neck. A pipette, delivering about 17.5 cubic centimetres of milk, and a measuring cylinder for the acid, are used. The bottles are whirled for several minutes at a temperature of 93° C. (200° F.) in a horizontal wheel making from seven to eight hundred revolutions per minute. The separation of fat by gravity alone is not complete even when the bottles are left standing for several hours. By centrifuge, however, a perfect separation is accomplished in a few minutes. If whirled at once, no heat need be applied, as that caused by the strong acid and milk is sufficient. After whirling, the bottles are filled to the neck with hot water, returned to the machine, and whirled for one or two minutes longer, after which they are filled with hot water to about the seven per cent. mark, and the machine is again turned for a short time, the temperature being kept up by means of a lamp or by filling the jacket with hot water. The fat separates and its percentage is noted while still liquid, preferably at about 65° C. (150° F.), the reading giving the percentage of fat directly without calculation, and being easily taken to 0.1 per cent.

This daily testing of the fat enables the modifier to preserve the accuracy of his material and to correct any variation in the percentage of the cream as it comes from the separator.

Many analyses of the milk of the herd used at the laboratory farms have been made, with the following result:

Fat	4.00
Sugar	4.30
Proteids	4.00
Mineral matter	0.65
Total solids	12.95
Water	87.05
	100.00

With the proper modifying materials the modifying clerks combine eac infant's food according to the prescription before them, and pour it into th glass tubes from which the infant is to be fed. These tubes are especiall devised as the most practical for general use, are adapted both for transportation and for use as nursing bottles, and are easily cleansed.

There are two sets of clerks. One set is engaged in modifying th milk according to the prescription. As soon as the tubes are filled by th modifying clerks they are passed on to the stoppling clerks, who immediately seal them with aseptic non-absorbent cotton especially prepared fo this purpose, and place them in baskets adapted as to their compartments to the number of feedings ordered for the special infant. The tubes are kept on tube-racks within easy reach of the modifying clerks. Each basket has its own label attached to it, with the address of the person to whom it is to be sent.

The rule of absolute cleanliness is carried out in every possible detail, from the table on which the materials are combined to the dress and hands of the clerks. (Fig. 3.)

When the milk has been separated, recombined according to the prescriptions, stoppled, and placed in the respective baskets, the baskets are taken from the modifying-room to the sterilizing-room. They are here placed in a sterilizer. The sterilizer is so arranged that the steam which passes through it can be regulated so as to produce any degree of heat required up to 100° C. (212° F.). This is accomplished by a regulator attached to the steam-pipe, and the man in charge of the heating of the food, by placing his hand on the regulator and watching the thermometer which is fitted to the sterilizer, can subject the baskets and the tubes in them to whatever degree of heat is ordered, and, of course, for any length of time required. (Fig. 4.)

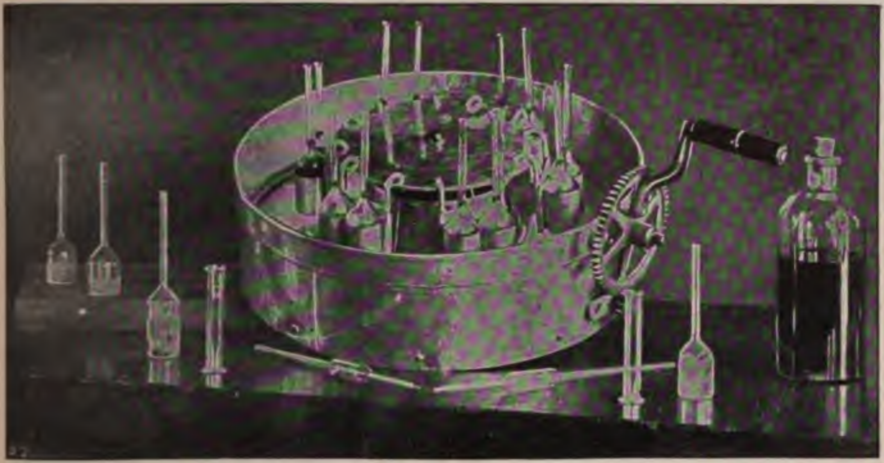
After the food has been heated, the baskets are taken out of the sterilizer and placed in the cooling-tank, where the temperature of the food is reduced to 13.3° C. (38° F.).

The baskets are then placed in the delivery wagon, which quickly conveys them to their various destinations. (Fig. 5.)

When the baskets are delivered at the homes of the consumers the baskets and tubes of the previous day are returned to the laboratory, and are taken directly from the street to the wash-room, which is entirely shut off from the rest of the laboratory. Here, in order to carry out absolutely the aseptic precautions, the baskets and everything which has been returned to the laboratory are placed in a special sterilizer belonging to the wash-room. The bottles after being sterilized are thoroughly washed in tubs in a solution of soda and water. All the tags and stoppers are destroyed after sterilization. The baskets are of woven willow, and are easily kept sterile.

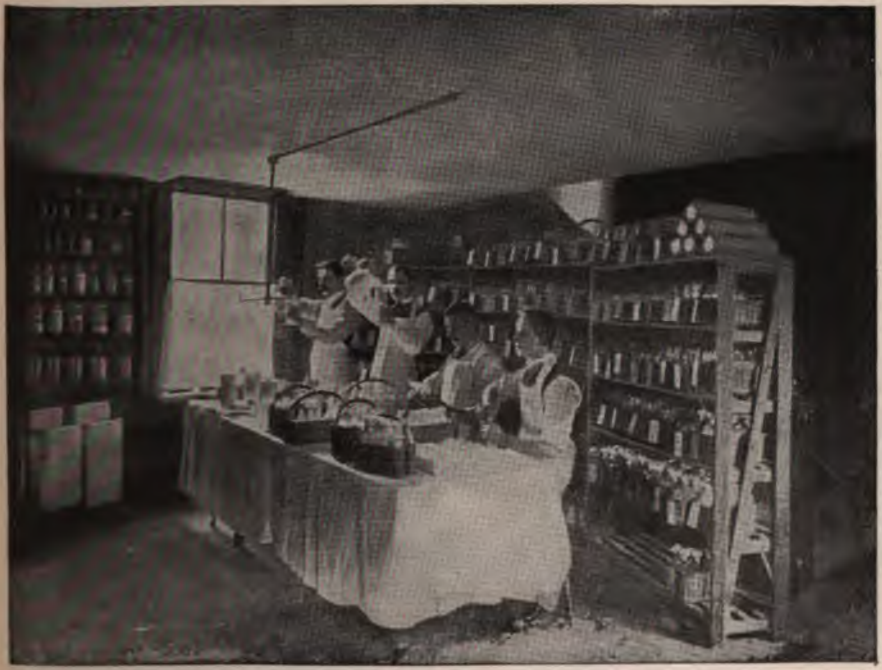
In this way, always guarding against possible infection of all kinds, the laboratory enables us to make use of the chemical and bacteriological

FIG. 2.



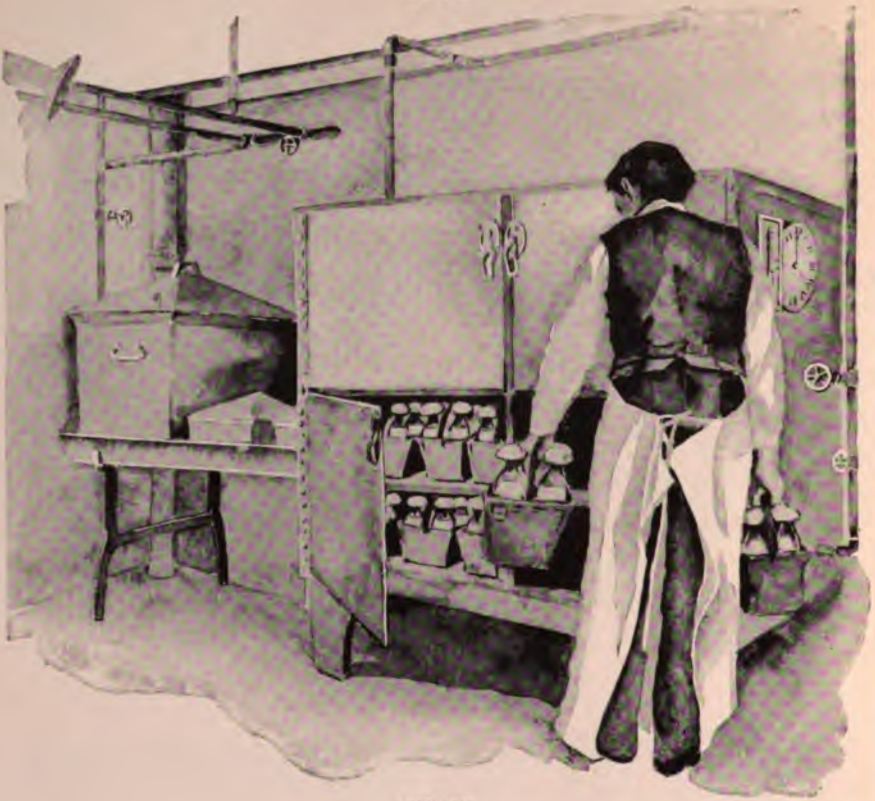
Babcock milk-tester.

FIG. 3.



Modifying-room.

FIG. 4.



Sterilizer.

FIG. 5.

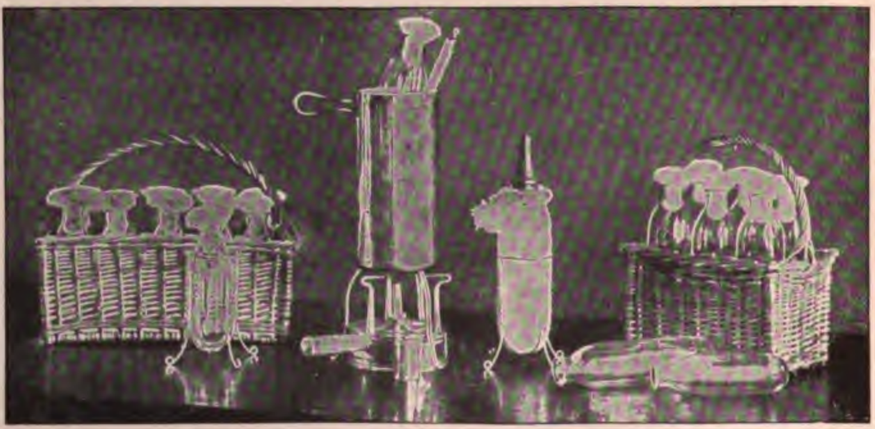


Illustration showing package employed for modified milk, with nursery apparatus for warming. In the left of the picture is the basket holding ten tubes of a capacity of six ounces each. In front of the basket is a four-ounce tube in a wire stand. In the middle of the picture is a tin apparatus for warming the milk at the time of feeding. An alcohol lamp is shown beneath the warmer, and a tube of milk and a thermometer for testing the temperature of the milk are in the tin warmer. Next to and to the right of the tin warmer is a tube with a capacity of eight ounces. It is enclosed in a white worsted cozy, has the rubber nipple in place, and is supported in a wire stand. In the right of the picture is a basket containing six tubes with a capacity of eight ounces each. In front of this basket are an eight-ounce tube and a four-ounce tube.

knowledge which we have acquired in connection with the feeding of infants, and fulfils the requirements of that system of substitute feeding which up to the present time has proved to be the best.

Modification.—Supposing that it is desired to prescribe some modified milk for an infant four months old, with normal digestion and of normal weight and development. A prescription can be written as follows on a milk-prescription blank, just as you would write a prescription for a drug:

MILK-PRESCRIPTION BLANK.

PER CENT.		REMARKS.	
℞ Fat	4.00	Number of feedings . .	7.
Milk-sugar	7.00	Amount at each feeding .	135 c.c. (4½ oz.).
Proteids	1.50	Infant's age	4 months.
Mineral matter . . .		Infant's weight	14 pounds.
Total solids		Lime-water.	5 per cent.
Water		Heat	75° C. (167° F.).
	100.00		

Ordered for ....

Date 189

Signature ....

The percentages written for in this prescription are—fat, four; sugar, seven; proteids, one and a half; reaction, slightly alkaline; the number of feedings, seven; the amount at each feeding, one hundred and thirty-five cubic centimetres (four and a half ounces); the tubes to be heated for twenty minutes at a temperature of 75° C. (167° F.).

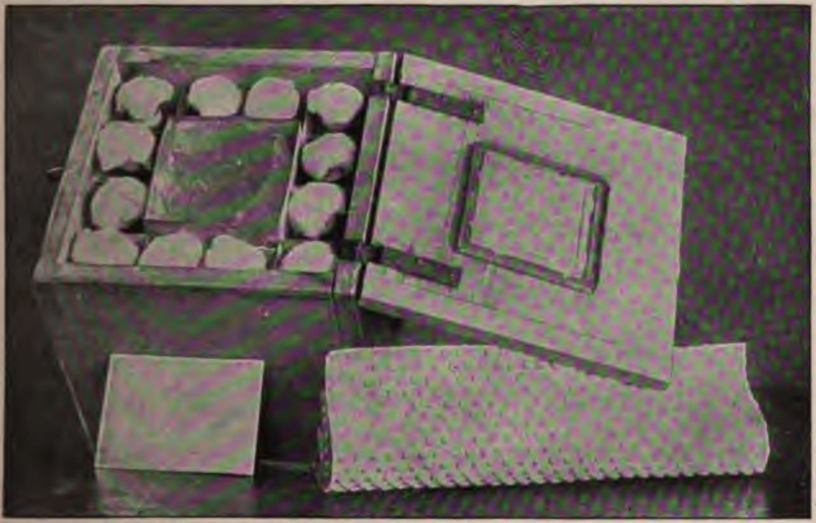
In regard to the question of the reaction, it can be left to the milk-modifier, as we leave to him the carrying out of other directions contained in the prescription. If the milk brought to the laboratory on the special day when we are sending our prescription is sufficiently alkaline for an infant's digestion when normal, no lime-water need be added. If, on the contrary, the milk has its usual acid or amphoteric reaction, the milk-modifier will make it slightly alkaline, in accordance with our prescription and according as the milk of the special day has a greater or less acid reaction. Lime-water is the best material to use for this purpose, and the least likely to do harm. If, however, the infant's digestion is not normal, and we wish to prescribe a precise amount of lime-water, we can do so by writing for whatever percentage we choose, as we do for the other elements of the milk. In modifying the milk which comes from the farm connected with this laboratory, as a rule, one-twentieth part of lime-water (five per cent.) is sufficient to make the reaction correspond to that of normal human milk. By referring to the following table it will be seen what the percentage of lime-water should be in order to obtain a greater or less degree of alkalinity.

[illegible]	Reaction.
[illegible]	Strongly alkaline.
[illegible]	Still strongly alkaline.
[illegible]	Slightly but distinctly alkaline, and corresponding to women's milk.

The hydrate of lime is said to be soluble to the extent of one part in seven hundred and seventy-eight parts of water at a temperature of 15.5° C. (60° F.). This would make one ounce of lime-water contain rather more than [illegible] (one-half grain) of CaO_2H_2 (hydrate of lime). The temperature of 75° C. (167° F.) is sufficient to kill the developed bacteria which would be of any harm to the digestion of the infant, and at the same time is below 77.2° C. (171° F.), the point at which coagulation of the proteids is supposed to take place. We thus obtain a practically pure fresh milk [illegible] and sterile. We therefore write in our prescription 75° C. (167° F.). If, however, the milk is to be sent a long distance, if the weather is hot, or if the milk-supply has to last for more than twenty-four hours, a higher degree of heating can be used according to the wish of the prescriber. Thus, 100° C. (212° F.) is a temperature used for these purposes in the laboratory. Where, again, we wish the milk to be absolutely sterilized, as may be the case when we are preparing it for an ocean voyage or for a trip across the continent (Fig. 6), not only a high degree of heat, 100° C. (212° F.), but also two or three heatings with intervals of twenty-four hours are necessary for this complete sterilization, and this can be called for in our prescription. The length of time during which the milk should be heated, as a rule, can be left to the judgment of the superintendent of the laboratory. Heating for ten minutes is often sufficient to kill the developed bacteria and to make this especial milk practically sterile. Experience, however, has proved that during transportation the milk is often exposed to temperatures conducive to the further development of bacteria, and that practically the bacteriological results which we obtain in the laboratory do not entirely hold when the milk is exposed to these varied conditions of transit. As a rule, therefore, from twenty to thirty minutes is the proper time to heat mixtures of modified milk sent from the laboratory.

The prescription when written by the physician is sent to the clerk in the office of the laboratory. The clerk copies it into a book which records each day's feeding of each individual infant. The clerk then translates the physician's prescription into such a form as can be readily understood by the modifying clerk. Of course, this form may vary in different parts of the world according as the metric or the apothecary system is in use. In the work of these laboratories, although the prescriptions are written by the physicians in the metric system, it has been found more convenient when delivered to the patrons of the laboratory to have the amounts expressed in ounces and drachms. The office clerk, after translating the metric percentages into ounces and drachms, copies it on a blank which is called the Modifying Clerk's Formula.

FIG. 6.



Ice-box, holding twelve tubes. Receptacle for ice in centre of box. Laboratory prescription-blank in front of box, and packing-paper under end of open lid.

This formula is then placed in the hands of the modifying clerk, who combines the different elements of the prescription by means of the elemental materials which have been brought into the modifying-room from a different part of the laboratory, and which have been already described. Physicians have been requested to write their prescriptions within certain limits as to the percentages of the fat, sugar, and proteids, and to allow the mineral matter for the present to regulate itself. The limits which up to the present time the laboratory has found it necessary to place on the prescriptions for the milk-modifiers, and within which the modifying clerk is supposed to put up the prescription, are as is shown in this table:

Fat	From 0.03 to 36.00
Sugar	From 0.87 to 20.00
Proteids	From 0.22 to 4.00

The results obtained from combining the modifying materials have so often been proved to be practically correct that we can assume that when we write a prescription we shall obtain a product which in its various elements comes within a fraction of one per cent.

The following table will be of use to those who wish to write for certain percentages of fat, sugar, and proteids:

PRACTICAL LIMITS OF MILK-MODIFICATION WHICH CAN BE ACCOMPLISHED IN THE LABORATORY.

I.

Low Fats.

Fat	0.03	0.04	0.08	0.12—16
Sugar	2.00	3.00	4—5	6.00—7.00
Proteids	0.75	1.00	2.00	3.00—4.00

II.

Low Sugars.

Sugar	0.87	1.40	2.12	3.50—4.30
Fat	2.00	3.00	3.50	4.00
Proteids	0.75	1.00	2.00	3.00—4.00

III.

Low Proteids.

Proteids	0.22	0.34	0.45	0.53
Fat	2.00	3.00	4.00	4.50
Sugar	2.00	3.00	4—5	6.00—7.00

In I. the lowest percentages of fat which can practically be used at the laboratory have been combined with the various percentages of sugar and proteids; in II. the lowest percentages of sugar which can be combined with these various percentages of sugar and proteids; and finally in III. the same calculation has been made for the proteids.

Other materials can also be obtained at the laboratory on the physician's prescription for older infants and children, notably preparations of oats, barley, and wheat.

When a physician orders cereals to be prepared at the laboratory he can thus obtain exact preparations as to the percentages of the constituents of any cereal food. This is accomplished by employing an analysis of the special cereal ordered, and with distilled water as a diluent regulating the time the heating should be maintained with "live steam" around the porcelain crocks which are used for this purpose.

The question having arisen whether the emulsion of milk which is used for modification is interfered with or destroyed by modification, the following specimen of milk strongly magnified and photographed will be of interest:

Fig. 7 represents a drop of milk the percentage of which is as seen in the following analysis:

ANALYSIS.

Cow's Milk.

Fat	4.04
Sugar	4.55
Proteids	4.15
Mineral matter	0.71
Total solids	13.45
Water	86.55
	100.00

Fig. 8 is a drop of a mixture which has been so treated as to represent the same analysis, and which in fact is the original whole milk that has been separated and recomposed.

The photographs show that the emulsion of the recomposed milk is quite as good as that of the original milk from which it was separated. There is not much question that the separation and recomposition of milk are not injurious so far as the emulsion is concerned.

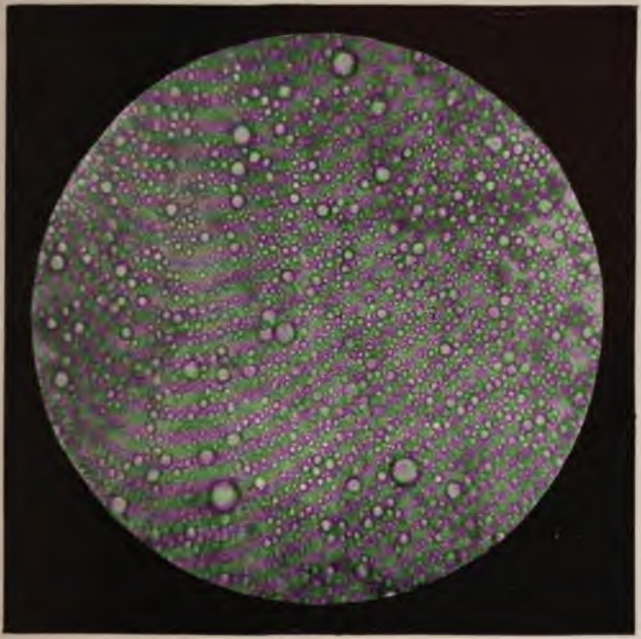
As in direct feeding from the breast the food which the infant receives has the same temperature at the end of the feeding as at the beginning, 36.6° to 37.7° C. (98° to 100° F.), this provision of nature should be copied and the temperature of the food not allowed to vary during the time it is being taken. To accomplish this end a white worsted cozy can be used. The cozy is warmed at the same time that the milk is being heated, and the tube when placed in it is prevented from cooling. Thus the infant receives a food of unvarying temperature throughout the whole of the feeding.

For transportation an ice-box (Fig. 6) can be used in hot weather, and will always prove of great practical utility.

This box admirably serves the purposes of an express-box and of a home refrigerator. The ice is packed in a metal compartment in the middle of the box, and the tubes are placed, each in its own compartment, around the sides of the ice-receptacle.

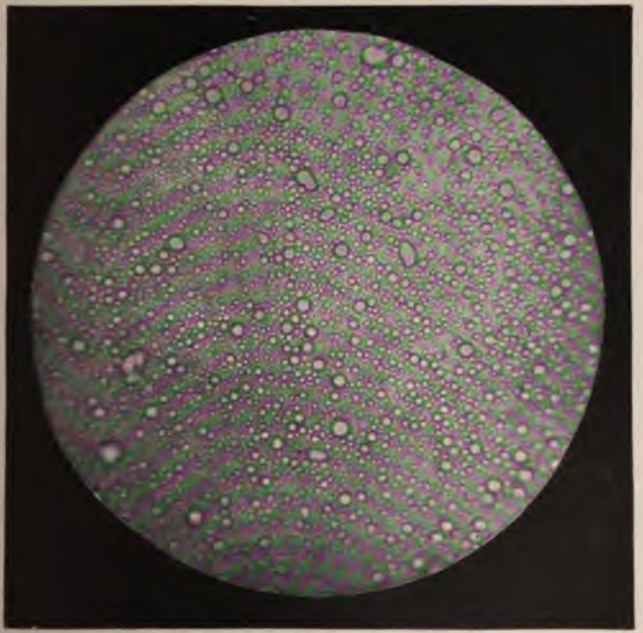
The following prescriptions which the writer has sent to the laboratory at different times will give a very fair idea of the simplicity and precision of substitute-feeding:

Fig. 7.



Cow's milk.

Fig. 8.



Cow's milk separated and recomposed.

PRESCRIPTION.

A girl six years old; duodenal jaundice (functional).

℞ Fat	0.50
Milk-sugar	6.00
Proteids	3.00
Lime-water	10.00

Send 12 tubes, each 4 ounces.

PRESCRIPTION.

A boy six weeks old; healthy.

℞ Fat	3.00
Milk-sugar	7.00
Proteids	1.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

12 tubes, each 2 ounces.

PRESCRIPTION.

A boy six months old; healthy.

℞ Fat	4.00
Sugar	7.00
Proteids	2.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

8 tubes, each 6 ounces.

PRESCRIPTION.

A girl four months old; proteid digestion weak.

℞ Fat	4.00
Sugar	7.00
Proteids	0 75
Lime-water	5.00
Heated to	75° C. (167° F.)

8 tubes, each 4 ounces.

PRESCRIPTION.

A boy six months old; sugar digestion weak.

℞ Fat	3.00
Sugar	4.00
Proteids	2.00
Lime-water	5.00
Heated to	75° C. (167° F.).

8 tubes, each 6 ounces.

PRESCRIPTION.

A girl four months old; summer diarrhœa. Food has to be sent to a distant town by express.

℞ Fat	2.00
Sugar	5.00
Proteids	1.00
At time of each feeding add lime-water	8 drachms.
Heated to	100° C. (212° F.)

20 tubes, each 1 ounce and 1 drachm.

In this last case the diarrhœa had not been sufficiently studied to determine whether it was putrefactive or fermentative, so that a safe general prescription was sent to begin with. The lime-water had to be introduced at each feeding on account of the 100° C. (212° F.) heating necessitated by the hot weather and the distance to be sent. If the lime-water had been introduced at the laboratory and heated to 100° C. (212° F.) with the food, a reaction would have taken place between the lime and the sugar and the mixture would have turned brown and have had a peculiar taste.

Feeding of Average Infants Born at Term.—When an infant is born at term, is of normal weight and development, and is healthy, the quality of the food to begin with should usually be as shown in the following prescriptions:

Prescription.

For the first twenty-four to thirty-six hours of life.

℞ Milk-sugar, five per cent. solution, in sterilized distilled water.

Prescription.

First week.

℞ Fat	2.00
Sugar	5.00
Proteids	0.75
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription.

Second week.

℞ Fat	2.50
Sugar	6.00
Proteids	1.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription.

Third week.

℞ Fat	3.00
Sugar	6.00
Proteids	1.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription.

Four to six weeks.

℞ Fat	3.50
Sugar	6.50
Proteids	1.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription.

Six to eight weeks.

℞ Fat	3.50
Sugar	6.50
Proteids	1.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION.

Two to four months.

℞ Fat	4.00
Sugar	7.00
Proteids	1.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION.

Four to eight months.

℞ Fat	4.00
Sugar	7.00
Proteids	2.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION.

Eight to nine months.

℞ Fat	4.00
Sugar	7.00
Proteids	2.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION.

Nine to ten months.

℞ Fat	4.00
Sugar	7.00
Proteids	3.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION.

Ten to ten and a half months.

℞ Fat	4.00
Sugar	5.00
Proteids	3.25
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION.

Ten and one-half to eleven months.

℞ Fat	4.00
Sugar	4.50
Proteids	3.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION.

Eleven to eleven and one-half months.

℞ Unmodified cow's milk.

At about the tenth or eleventh month at first one and then two meals can be given daily of equal parts of oat jelly, prepared at the laboratory, with plain cow's milk heated to 75° C. (167° F.) and a little salt added according to the infant's taste at the time of the feeding. Freshly prepared barley or wheat can, if preferred, be given with milk at this age.

In the twelfth month the infant can be accustomed to taking a little bread one day old with its milk, and to be fed from a spoon, so that by the time it is a year old it is taking bread and milk for its breakfast and supper, and oat jelly and milk for the three middle meals.

Although exact rules for the modification of milk in health and disease cannot as yet be given, yet one cannot help feeling from the large number of cases which have been treated in this way that certain impressions which have been derived from watching carefully the results of this treatment may be of some use to those who are working in this field of medicine. For instance, in regard to the treatment of a difficult proteid digestion, it has been found that instead of predigesting the proteids it is better to reduce them first to a minimum, and then gradually, as the proteid digestion becomes stronger from not being overtaxed, to increase the proteid percentage until the amount necessary for nutrition is attained. Thus, beginning with the proteids 0.25 for all ages, whether infant, child, or adult, to increase up to 1.50 for infants, 2 to 3 for children, and 3 to 4 for adults.

According to the writer's experience, it is seldom wise to increase the fat above four per cent., which is the usual limit found in breast-milk, especially during the first eight or ten months of life in the feeding of healthy infants. Where, however, fat is indicated, as in those cases in which cod-liver oil is usually prescribed, a decided increase in the fat up to five or six per cent. is at times of value.

It is also noticeable that since it has been found possible to accomplish the exact and careful modification of milk at the laboratory, using the many different combinations at our command, our experience does not accord with that of those who believe that certain individuals cannot take milk, or that milk is contra-indicated in certain diseases. All foods contain practically the same constituents, and milk, or milk mixed with starch, comprises all these constituents. The individual does not reject milk, or suffer from malnutrition caused by the administration of milk. Milk is not contra-indicated in an especial disease; on the contrary, it is some element, or constituent, or combination of the constituents making up the milk wherein the fault lies. We might just as well at once, and better, too, because we can do it more exactly and more simply, find out by changing our modification whether it is the fat, or the sugar, or the proteid, or the starch digestion which is weak or diseased, or whether these elements as a culture-ground will encourage bacterial growths in the individual. We can then remedy the trouble by not overtaxing the especial function until it has had time to recover its normal power. The great mistake which those make who experiment with foods is that they look upon the different foods and their action and effects in health and disease as a whole, and not at the details which make up this whole. Milk, as a whole, is a mild and simple dinner made up of the same food-constituents as are placed on our tables, excepting that in it we have the starch predigested into sugar. One individual will thrive best on an excess of vegetable food, another on fats, another on meat, and this idiosyncrasy of the individual in early life and in disease in later life can be met by simply modifying these food-constituents in the milk.

In a fairly large experience derived from the care of infants who have been fed from the first days of life among people who have had sufficient means to give their children the best hygienic surroundings, I can state that I have never yet seen an infant who during the first ten or twelve months of its life was carefully fed on nothing but the milk from these laboratories, where the percentages of the milk constituents were carefully changed, enter upon its second year without firm flesh and an average development. I have found the teeth to be sound and to come at the usual age. The functions of sitting, walking, and standing and the amylolytic function all appear and develop normally. I have followed these children into their third, fourth, and fifth years, and have found them strong, ruddy, with good bones and teeth, and with digestions which permit them to be fed on a general mixed diet of all the food elements.

It has long been noticed that in breast-feeding an infant is often found to digest well the milk of one woman, and to suffer from serious digestive disturbances when another woman's milk is substituted for it. For this reason in selecting a wet-nurse we not infrequently have to try one nurse after another until one can be found in whose milk the proportions of the fat, sugar, and proteids are suited to the infant's digestion. So it is with older children; if cow's milk is not well digested the different constituents of the milk should be changed in their proportions and in their combinations until proper percentages and combinations have been found. It must be remembered that prescribing so much milk and so much cream is a very indefinite method, and one in the use of which it will be found that most varying percentages will result, and often entirely unlike what we suppose we are prescribing. The only exact method by which we can obtain the percentages of the different elements which we believe will suit the individual case, and which can make our own experience valuable in comparison with that of others, is to prescribe directly a definite percentage of the fat, sugar, and proteids. This method is, in fact, exceedingly necessary in scientific infant feeding.

We are not in position to-day to say exactly what are the best modifications of milk to be used in all the manifold instances in which some modification is evidently indicated; we have learned that to modify milk is a wise and valuable adjunct to our treatment, and in certain instances what combinations of the constituents of the milk are best for the individual case. Looking at the subject as a whole, however, we are strongly impressed with the value of the opinion that different individuals, both in health and in disease, require different combinations of the constituents of the milk to suit a special case, from the fact that with the ignorance of the prescriber working against the prescription, still the result is that in a vast majority of instances the patients have done well. The position which it seems fair for us to take is, that the principle of the modification of milk is scientific, is practical, is right, that in the milk-laboratory we have one more instrument of precision to aid us in our work, and that in all probability the failures are not to be attributed to the principle and the instrument, but to us, who, acknowledging the principle as a whole, and using the instrument, are as yet ignorant of the details which make up that principle, and do not do the instrument justice.

When any special modification of milk obtained from the laboratory does not suit an individual infant or child, and the treatment with this is not successful, each of the elements of the milk must be carefully changed and different combinations of these elements tried until the individual idiosyncrasy in the especial case is discovered.

Quantity of Food at Different Ages.—The following table represents the average amount of food taken at different periods during the first year by three hundred and forty-one infants who were fed from the milk-laboratory. They were all well and strong, of average weight, and steadily gained during the year. They received only stated amounts of food carefully ordered by prescription at the milk-laboratory, and were watched with the greatest care to see when they were evidently hungry enough to have the total amount of their food increased. Of course, the opportunity for exact work is almost unlimited where one has a milk-laboratory at his command, and it therefore seems that this method of determining exact gastric capacity is an unusually good one, and one which has never been thoroughly carried out before.

THREE HUNDRED AND FORTY-ONE INFANTS FED AT THE MILK-LABORATORY.

AGE.	NO. OF CASES FOR EACH AGE.	AVERAGE AMOUNT OF FOOD AT EACH FEEDING.	
		C.C.	Ounces.
Birth	45	29.4	0.98
Four weeks	76	70.5	2.35
Eight weeks	84	96.6	3.22
Twelve weeks	97	118.8	3.96
Sixteen weeks	87	137.0	4.57
Twenty weeks	86	158.4	5.28
Six months	73	171.3	5.71
Seven months	56	185.4	6.18
Eight months	54	208.5	6.95
Nine months	45	226.2	7.54
Ten months	33	238.8	7.89
Eleven months	28	242.0	8.07

(In this table the same infant has, of course, been recorded a number of times at different ages.)

FEEDING OF PREMATURE INFANTS.

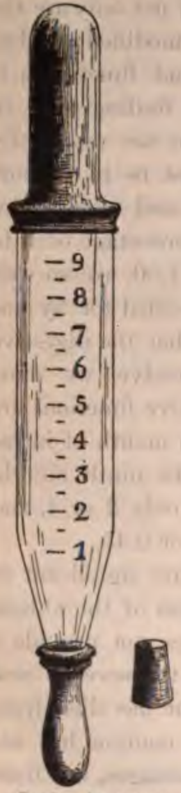
The most satisfactory example of the value of feeding with exact percentages representing the constituents of food, and not prescribing food as a whole, is shown in the feeding of premature infants. Here it has been found of such vital importance that the digestive function of the infant should not be overtaxed, that the possibility of providing for the undeveloped premature infant's stomach a milk with its percentages mechanically correct is of unquestioned value. The quantity which should be given to the premature infant is, of course, very important, and has not been spoken of in the previous article. This amount has, however, been deduced from careful measurements of fœtal stomachs, just as we have calculated the amount which should be given to the infant at term and during the first year of its life. It will be found by referring to the investigations which have been made on this subject that it is safer to begin with four or five cubic centimetres (about one gramme) and gradually to increase the amount up to a point where our very imperfect knowledge on this subject, derived partly from the weight of the infant, makes us believe that its stomach is full, than to begin at once with the larger amount. It is absolutely necessary that we should avoid distention of the stomach, as this may prove fatal. In this early period of development not only are the lungs and the heart immature, and in need of a carefully modified air, but the gastro-enteric tract is also undeveloped in its size and functions; it needs smaller quantities of food and shorter intervals of feeding than in the stage of development met with at birth. Its functions are essentially weak, are not ready to be used, and, if used, as they must be to support life, they are easily overtaxed, as has been proved over and over again. If we but recognize that while the fat digestion with its percentage of 3 to 4, the sugar digestion 6 to 7, the proteid digestion 1 to 1.50, are so well developed in the average infant at term, and so well provided for by human breast-milk with its percentages corresponding to what the digestive functions of this stage of development are ready to receive, we must realize, on the contrary, that the premature infant's digestive functions are in an earlier stage of development, and that at the seventh month of intra-uterine life the fat percentage, instead of being 3, as at the ninth month, may be only 1, the sugar digestion, instead of 7, may be only 3 or 4, and the proteid digestion, instead of 1 or 2, may be only 0.25 or 0.45.

These rules the writer has tested clinically, and they are significant as showing that nature, while providing the best food by means of the human breast for the stage of development met with at term, does not provide a suitable food for the stage of development met with in the seventh and eighth months of pregnancy. Many a premature infant has died from being put to the breast of its own mother, for the human mamma has not been made to modify its milk constituents into low percentages, but from long ages of use and inheritance has been taught to modify in the higher percentages adapted to the tenth, eleventh, and twelfth, or, rather, first, second, and third months of life. In this class of cases, therefore, there seems to be no question that the careful modification of cow's milk, by dealing with low percentages, gives the premature infant a far better chance for life than does human breast-milk. Any one who has followed a premature infant from the day of birth, giving it up to the time of weaning nothing but carefully combined percentage feeding, seeing it digest and thrive first on 0.50 of fat, 4 of sugar, 0.25 of proteids; then on 1 of fat, 5 of sugar, 0.50 of proteids; then on 1.50 of fat, 5.50 or 6 of sugar, 0.75 of proteids; then on 3 of fat, 6.50 of sugar, 1 of proteids; then on 3.50 of fat, 6.50 of sugar, 1.50 of proteids; then on 4 of fat, 7 of sugar, and 2 of proteids in the sixth month, cannot help feeling that at least we are approaching the time when an exact system of dealing with nutrition is to be hoped for.

In connection with the food which is given to the premature infant, it has been found that where the infant swallows or sucks with difficulty a device suggested by Dr. Breck will often be of practical use. (Fig. 9.)

It is simply a glass cylinder twelve centimetres (four and three-fourths inches) long and two and four-tenths centimetres (one inch) in diameter. The cylinder is graduated to two cubic centimetres (one-half drachm), and holds thirty-six cubic centimetres (nine drachms). It is shaped at one end so as to have a small rubber nipple fitted to it. The large end is covered by a rubber cot. The rubber cot, which has no holes, acts as an air-reservoir, and by simply introducing the small perforated nipple into the mouth and gently pressing the rubber cot the food is slowly forced down the infant's throat without choking it and without the infant's having to suck or apparently to use any effort. To fill the tube the rubber nipple and cot are removed, a rubber stopper is introduced into the small end of the cylinder, and the required amount of food is poured in at the large end. This method of feeding is especially desirable for a weak premature infant in a brooder, because it entails no loss of strength on the part of the infant, and can be easily managed by the right hand of the nurse while her left hand supports the infant's head. This method is far preferable to that of gavage, which is not so easily managed by the nurse and causes more exhaustion to the infant.

FIG. 9.



Feeder for premature Infants (reduced one-half).

MIXED FEEDING.

Where the infant is not thriving satisfactorily, but where it is not deemed advisable to wean entirely, it is better to feed partially from the breast and partially from the bottle.

There are certain points to be considered in mixed feeding. First, if the mother's milk is agreeing with the infant, the substitute food should be of the same composition. Second, if the mother's milk is fully digested by the infant, but is lacking in certain nutritive qualities, the absence of which prevents the infant's nutrition from being normal, we should, after the first week, alter the composition of the substitute food so as to make it fulfil the requirements of nutrition by increasing the percentage of that special element in the substitute which is deficient in the composition of the maternal milk.

The times at which the substitute food should be given will depend upon the number of feedings which are found to be necessary in addition to the maternal feedings, and we should carry out the same principle in this mixed feeding that I have laid down for the general management of human breast-milk. If the mother's milk is lacking in quantity we should make the intervals between her nursings longer and introduce one or two substitute feedings, according as the age of the child requires shorter or longer intervals. If, on the contrary, the mother's milk is abundant, but either too strong or too weak, we should make the intervals of her nursings correspondingly long or short. In this way, with an accurate knowledge of the percentages which exist in the mother's milk, and with our power to change these percentages in substitute feeding, we can usually in a week or ten days regulate the substitute feeding of the infant to such a degree that the mother's milk will also agree with it, and the infant will thrive again.

WEANING.

In the original article I have explained how much better it is to wean slowly than rapidly. This is very strongly exemplified when we make use of the laboratory methods of modification for the purpose of weaning. If the infant is thriving and is digesting its mother's milk well, it can be given a substitute food from the laboratory with the percentages corresponding to those of the milk which it is receiving from its mother. After a few days, if this food is agreeing with the infant, the percentages of the different constituents can be changed with the object of gradually combining them in such a way as to correspond to the percentages of the elements of unmodified cow's milk. This is easily and precisely accomplished. For instance, supposing that the infant is receiving from its mother a milk in which the percentage of fat is 4, sugar 6.50, and proteids 2, the percentages in the modified milk can first be made to correspond to these percentages. After a few days, if this is digested well, the fat can be made 4, sugar 5.50, and proteids 2.25. If this is digested well, a few days later the sugar can be decreased to 4.50, the proteids increased to 3, and the fat kept at 4. In a few days more, if this food is digested well, plain cow's milk can be given heated to 167° F., with lime-water sufficient to make it alkaline. If this still agrees with the infant, cow's milk unheated and unmodified can be given.

Thus, in no way can weaning be accomplished so well and so satisfactorily as by slowly changing the percentages until they have approached each other so closely that the digestive functions of the infant cannot help being ready to receive unmodified cow's milk.

What the writer hopes in the future to accomplish is to have the diarrhœal cases so differentiated that they will be fed and treated by a reduction of the special constituent of the milk which appears to offer a culture-ground for the development of especial flora in the intestines.

Under all circumstances it is important to carry out strictly the many details which are necessary if we wish to obtain satisfactory results in the feeding of cases of difficult digestion.

HOME MODIFICATION.

On reviewing the history of the evolution of substitute feeding from the various artificial foods prepared by the non-medical capitalist and administered under the authority of the nurse-maid, to the preparation of a substitute food prescribed on scientific principles by educated physicians and prepared by skilled clerks, there is not much doubt that the home modification of milk for infants will finally fall into disuse. This has been the history of the propounding of drugs at home or in the physician's office. It is highly probable that the home modification of milk will cease to exist, as the propounding of drugs has already done, except at great distances from medical centres or among partially educated physicians. For the purpose of home modification, however, until laboratory methods are better understood, a few directions will be of value to the country practitioner concerning the modification of milk in the home of the patient in connection with the modification of milk in the laboratory. It should be thoroughly understood, however, that the writer believes that laboratory preparations are far better than home modifications, and that by laboratory modification only can the problem of infant-feeding in the future be solved.

Certain materials and apparatus are needed for home modification.

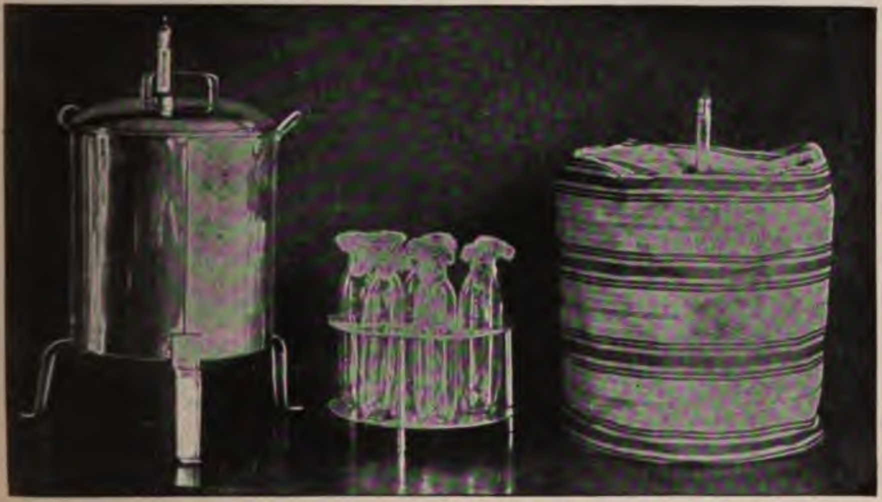
For purposes of heating the food at different temperatures what is called the home sterilizer can be used. (Fig. 10.)

This is simply a tin can supported on legs so that it can be heated by a lamp, or, if preferable, the legs can be removed and the can be placed on a stove. It has a lid to which is fitted a thermometer, by which the degree of heat within the can is indicated.

The tubes for holding the food, varying in number according to the number of feedings which are required in twenty-four hours, are placed in a rack which can be lowered into the sterilizer and immersed in the water, which is made to rise as high as the level of the milk in the tubes. The tubes are stoppled with aseptic non-absorbent cotton-wool, which has been found in the laboratory to be the most practical method. For the purpose of retaining the heat after the flame has been removed, the sterilizer is covered with a thick cozy, through which the thermometer from the lid passes and indicates the degree of heat retained within the sterilizer.

An eight-ounce glass graduate divided into half-drachms is most useful,

FIG. 10.

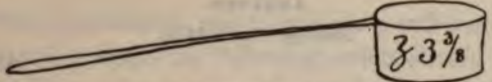


Sterilizer and thermometer. Rack for tubes. Sterilizer covered with cozy after removal from heat.

though not absolutely necessary, for measuring the materials which make up the food.

The milk-sugar can be bought in bulk and the proper amount added by means of the sugar-measure. (Fig. 11.)

FIG. 11.

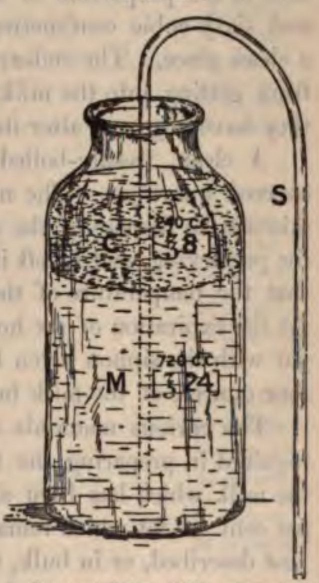


Sugar-measure.

This measure obviates the expense of having the milk-sugar put up in packages by the apothecary, and is sufficiently exact to regulate the sugar percentages in the mixtures of which I shall speak presently. It is well to remember, however, that a pound of milk-sugar contains four hundred and sixty-four grammes (seven thousand grains), and that if it is preferred to order the sugar in packages of 13.5 grammes (3⅜ drachms) directly from the apothecary, in place of using this measure, thirty-five packages can be made from the pound, and a package of milk-sugar can be used instead of a measureful.

A glass graduated jar (Fig. 12) and a glass siphon tube one-quarter to one-half inch in diameter, with the end out of which the milk is to flow at least six inches longer than that which is to be inserted in the jar, should be provided. To operate the siphon, fill it with boiled water, close the longer end with the finger, invert the siphon, and place the shorter end in the milk. Then withdraw the finger, and the water followed by the milk will run out from the long arm of the siphon. Do not use the mouth to start the flow of the milk through the siphon under any circumstances.

FIG. 12.



Jar containing milk, cream, and siphon. C, cream; M, milk; S, siphon.

The home-modifier is to be told that precautions must be taken to follow all directions to the minutest detail, or otherwise a uniformly correct result will not follow. It must be explained that the milk from a herd of cows is preferable to that of one cow, for many reasons, but especially because the elemental percentages are less likely to vary in the mixed milk of a herd than in the milk of the individual, and because the mixing lessens the deleterious effects on the milk arising from occasional disturbance of health in an individual member of the herd. The cows should be of a common breed and such as give a moderately rich milk. The milk should be drawn with clean hands. The udders and teats of the cows should be cleansed, and the cows should be milked in as clean a place as possible. The milk should be thoroughly strained. The milk will then be fairly uniform in its elemental percentages and comparatively free from bacteria and foreign matter. The composition of this milk will usually correspond to the following analysis:

ANALYSIS.

Average Cow's Milk

Reaction	Slightly acid.
Specific gravity	1029—1033
Water	86—87
Total solids	14—13
Fat	4.00
Sugar	4.50
Proteids	4.00
Total ash	0.70
Chlorine	13.45
Sulphur	0.41
Phosphoric acid	27.98
Lime oxide and alumina	0.44
Lime	23.17
Magnesia	2.63
Potassium	53.00
Sodium	4.49

The milk is then set in a vessel containing ice and water with some salt, in the proportion of five grammes (one teaspoonful) to nine hundred and sixty cubic centimetres (one quart) of water, and the vessel is set in a clean place. The endeavor should always be made to prevent impurities from getting into the milk, in preference to trying to eradicate them after they have begun to alter its normal composition.

A clean, freshly-boiled cotton cloth should be thrown over the uncovered quart jar. The mouth of the jar is kept open for about fifteen minutes, to dispose of the animal heat. The jar is then sealed tightly, as for preserving, and is left in the ice-water for six hours, care being taken that the temperature of the water does not fall below 1.66° C. (35° F.). At the expiration of six hours siphon out carefully from the bottom of the jar with the siphon seven hundred and twenty cubic centimetres (twenty-four ounces) of the milk into a clean glass vessel.

The various materials are thus ready for any combinations which are required in preparing the food for an especial infant. These materials are the milk which has been siphoned from the jar, the cream containing ten per cent. of fat which remains in the jar, the sugar, either in packages, as just described, or in bulk, to be used when needed with the sugar-measure, some fresh lime-water, and some clean drinking-water which has been boiled for five minutes.

The writer has arranged in the following tables the figures by means of which the various combinations can be made which are likely to be needed, and which correspond somewhat to the prescriptions which have been shown in connection with laboratory modification.

Supposing that the following combination is required:

Fat	0.25
Sugar	4.00
Proteids	0.25
Lime-water	5.00

To obtain this combination and to provide a sufficient quantity of food to last for twenty-four hours the following orders should be given.

Set enough milk to raise cream sufficient for the mixture required. For each twenty ounces, or part of twenty ounces, use the following formula:

Cream	½ ounce.
Milk	1 ounce.
Lime-water	1 ounce.
Water	17½ ounces.
	20 ounces.
Milk-sugar	2 measures.

The milk-sugar is to be thoroughly dissolved in the water before the other materials are added.

In like manner in the following combinations the percentages have been reduced to ounces:

TABLE.

Fat	1.00	Cream	2 ounces.
Sugar	5.00	Milk	2 ounces.
Proteids	0.75	Lime-water	1 ounce.
Lime-water	5.00	Water	15 ounces.
			20 ounces.
		Milk-sugar	2 measures.

TABLE.

Fat	2.00	Cream	4 ounces.
Sugar	5.00	Milk	None.
Proteids	0.75	Lime-water	1 ounce.
Lime-water	5.00	Water	15 ounces.
			20 ounces.
		Milk-sugar	2 measures.

TABLE.

Fat	2.00	Cream	4 ounces.
Sugar	5.50	Milk	1½ ounces.
Proteids	1.00	Lime-water	1 ounce.
Lime-water	5.00	Water	13½ ounces.
			20 ounces.
		Milk-sugar	2¼ measures.

TABLE.

Fat	2.50	Cream	5 ounces.
Sugar	6.00	Milk	None.
Proteids	1.00	Lime-water	1 ounce.
Lime-water	5.00	Water	14 ounces.
			20 ounces.
		Milk-sugar	2½ measures.

TABLE.

Fat	3.50	Cream	7 ounces.
Sugar	6.50	Milk	1 ounce.
Proteids	1.50	Lime-water	1 ounce.
Lime-water	5.00	Water	11 ounces.
			20 ounces.
		Milk-sugar	2½ measures.

TABLE.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Milk	None.
Proteids	1.50	Lime-water	1 ounce.
Lime-water	5.00	Water	11 ounces.
			20 ounces.
		Milk-sugar	2¾ measures.

TABLE.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Milk	2½ ounces.
Proteids	2.00	Lime-water	1 ounce.
Lime-water	5.00	Water	8½ ounces.
			20 ounces.
		Milk-sugar	2½ measures.

TABLE.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Milk	5 ounces.
Proteids	2.50	Lime-water	1 ounce.
Lime-water	5.00	Water	6 ounces.
			20 ounces.
		Milk-sugar	2¼ measures.

TABLE.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Milk	7½ ounces.
Proteids	3.00	Lime-water	1 ounce.
Lime-water	5.00	Water	8½ ounces.
			20 ounces.
		Milk-sugar	2 measures.

TABLE.

For weaning.

Fat	4.00	Cream	8 ounces.
Sugar	5.00	Milk	7½ ounces.
Proteids	3.00	Lime-water	1 ounce.
Lime-water	5.00	Water	8½ ounces.
			20 ounces.
		Milk-sugar	1 measure.

TABLE.

For weaning.

Fat	4.00	Cream	8 ounces.
Sugar	5.00	Milk	8 ounces.
Proteids	3.25	Lime-water	1 ounce.
Lime-water	5.00	Water	3 ounces.
			20 ounces.
		Milk-sugar	⅞ measure.

TABLE.

For weaning.

Fat	4.00	Cream	8 ounces.
Sugar	4.50	Milk	12 ounces.
Proteids	3.50		20 ounces.

After the various materials have been mixed in the proportions indicated in the tables, the mixture is prepared for the "home sterilizer." The requisite amount of food for one feeding is poured into each of the tubes. They are stoppled with cotton-wool, care being taken to have a reasonably tight stopple in and a dry neck to the tubes. The tubes are then placed in the rack and lowered into the sterilizer, and the water in the sterilizer is adjusted to the level of the milk in the tubes. Heat by means of a lamp or stove is then applied to the sterilizer, which is watched with the cover off until the thermometer shows that the water-bath has reached a point of 77.2° C. (171° F.). As soon as this temperature is reached the sterilizer is removed from the heat, the cover put in place, and the cozy over it. The thermometer should mark a temperature of between 75° and 77.6° C. (167° to 172° F.) for thirty minutes, at the expiration of which time the tubes are to be removed from the sterilizer, carefully cooled by allowing ice-water to run over them, and then kept in a cool place, preferably the ice-chest, until needed.

SECOND NUTRITIVE PERIOD.

During the eleventh and twelfth months of life the amylolytic function of the infant has become almost fully developed. In accordance with the rule regarding the use of the different functions,—namely, that a function should not be taxed before it is developed, and that when its development is almost completed it should be brought into use,—we should in the latter part of the first year begin to use that function of the digestive tract by means of which the amylaceous elements of the food are converted into sugar. Preparations of oats or barley mixed with the milk are of great value in the feeding of this transitional period, and are the best means of testing the infant's amylolytic function. The high percentage of sugar which up to this transitional period has been given in the modified milk should now be reduced to that which normally exists in average unmodified cow's milk, since the starch in the additional articles of food now given will be converted into and provide the necessary amount of sugar. In this connection also it will be noticed that the proteids may be markedly increased in amount, as the power to digest proteids is much increased during the latter part of the first year. In a normal infant with normal digestive functions a considerable percentage of starch can be digested and absorbed with benefit in the eleventh and twelfth months. Preparations of oats or barley mixed with milk are to be introduced into the infant's food at this period. There is a larger percentage of starch in oats than in barley. It is also more nutritious in every respect, as it contains a considerable percentage of fat. The starch in oats, however, takes a somewhat longer time to be converted into sugar than does that of barley, so that in the case of an infant whose amylolytic function is not fully developed or is somewhat weak, preparations of barley will be better to begin with because they do not introduce so high a percentage of starch into the food, and also because the starch will be more readily converted into sugar. Preparations of oats seem to be the best form of food to be added to the modified milk when the infant has reached a period at which it needs a change in the character of its food.

When the infant has reached the second or fourth month of its life it should normally be able to digest four per cent. of fat in its food. This percentage of fat corresponds to that which exists in average cow's milk. It is natural to suppose that at the eleventh and twelfth months a still further increase in the amount of fat which is provided in the infant's food is required as well as the new element, starch. This fat is supplied in considerable quantity from the oats. We have, therefore, in preparations of oats, both for purposes of weaning and for establishing a new regimen of diet for the infant, a food which in combination with cow's milk satisfies completely the demands which the digestive functions at this period are making for a perfect nutrition.

Oats.—For the preparation of oat jelly, the following method should be employed:

One hundred and twenty grammes (four ounces) of coarse oatmeal are allowed to soak in a quart of cold water for twelve hours. The mixture is then boiled down so as to make a pint, and is strained through a fine cloth while it is hot. When it cools a jelly is formed, which is kept on ice until needed. Different proportions of this jelly can be used, but usually it is best to begin with equal parts of jelly and cow's milk. When needed, this mixture is warmed and a little salt is added.

Barley.—Barley water is made by boiling one hundred and fifty grammes (five ounces) of granulated barley in a quart of water until the volume is reduced to a pint, and then straining.

If a barley jelly is to be made, one hundred and twenty grammes (four ounces) of barley flour are to be employed, and the same process is gone through with as for the preparation of oat jelly. The resulting jelly is treated in the same way with milk as has been directed for oat jelly.

It is well to speak here of a few other preparations.

Wheat.—Wheat can be prepared by the same method as that described for oats and barley.

Peptonized Milk.—For peptonizing milk the following rules are the most practical and simple:

In a clean glass jar containing four ounces of cold distilled or boiled water, dissolve one gramme (fifteen grains) of bicarbonate of sodium and 0.25 gramme (four grains) of pancreatine (extractum pancreatis), to which add twelve ounces of whole milk. Set the jar in a vessel of water at a temperature of 41.6° C. (107° F.) for from seven to ten minutes. Cool immediately, and keep on ice until used.

To peptonize modified milk, an amount of the powders should be used corresponding with the percentage of the proteids in the mixture, taking the standard of the whole milk to be represented by four per cent. of the proteids.

Sweet Whey.—Sweet whey is best made by the following method. For each pint of whey needed, take one quart of whole fresh milk, heat it to 37.7° C. (100° F.), and add eight cubic centimetres (two drachms) of the essence of pepsin, or one square inch of rennet. When the proteids have been precipitated, break the curd finely with a fork and pour off the fluid, straining it through two thicknesses of boiled cheese-cloth. This removes such of the proteids as are coagulable by acids. Place this strained liquid in a clean porcelain pot, and raise the temperature to the boiling point by a stove or a lamp, but do not allow it to boil. Strain this hot liquid through a cloth as before. This removes the proteids coagulable by heat. Cool the resulting fluid slowly to a temperature of 10° C. (50° F.), and keep it on ice until needed.

The second nutritive period may be reckoned to last from the twelfth to the twenty-eighth or thirtieth month of life. That is about the second half of the period which we are in the habit of calling infancy. It also includes the time when the last four teeth of the first set appear. In this second nutritive period the element of variety in the food is important. It is undoubtedly important that the actual nutritive values of the food which it is best to give the infant in this period be considered, but it is of much more consequence that special attention be paid to the variety. Foods should be given which, while containing a fair percentage of nutritive elements, yet differ in the combination of these elements to such a degree that they fulfil the requirements of this period of life. It is best to increase gradually the variety of articles of diet from the twelfth to the twentieth month, always adapting the food to the special infant. Thus, some infants may be able to digest and assimilate proportionately large quantities of starch; others may both need and digest larger proportions of the proteids or of sugar than the infants first spoken of.

Between the twelfth and thirteenth months I am in the habit of giving an infant five meals during the day. At this time it is well to accustom it to take its food from a spoon and as soon as possible to omit feeding from the bottle. The five meals should be arranged in the following manner:

For breakfast, bread and cow's milk slightly warmed.

For lunch, equal parts of oat jelly and cow's milk, warmed, with a little salt added according to the infant's taste.

This meal of oat jelly should be repeated in the middle of the afternoon.

In the middle of the day broth of some kind, either chicken or mutton, carefully prepared so as to be free from fat on its surface, can be given with some bread.

The fifth meal should be given in the latter part of the afternoon, and should consist of bread and milk.

In some cases it is impossible to make infants swallow bread for a long period after the usual time of from twelve to thirteen months. At times it is not until they are from two and one-half to three years old that they can be induced to take bread. In these cases we must feed them according to our judgment of the individual case.

When the infant is from fourteen to fifteen months old, some thoroughly boiled rice can be added to the broth in the middle of the day, and if it digests this well it can also have bread given with this meal.

When the infant is sixteen months old, it can have a small amount of butter on its bread. When it is from seventeen to eighteen months old, it can have a thoroughly baked white potato, mixed with butter and salt, added to its mid-day meal of broth. When it is from nineteen to twenty months old, eggs can become part of its diet.

There are not many fruits which should be given to the infant in its second year. A baked apple can be given at the evening meal when the infant is from fourteen to fifteen months old; or, for variety, the apple can be made into a simple sauce, never, however, having the sauce made with much sugar. When peaches are in season, a ripe peach can often be given with benefit, especially if the infant is inclined to be constipated. Other fruits should be avoided, as they are not necessary for the infant's nutrition, and at times produce serious trouble.

This is the diet which is sufficient for the infant during the second nutritive period. It is important for the subsequent integrity of the infant's digestion and general nutrition that the parents should insist that no other articles of food be employed, except such as are similar to those which I have spoken of,—namely, the cereals in a variety of forms, according to the taste, judgment, and knowledge of cooking which exist in the special household. For instance, preparations of wheat and barley cooked in various forms can be given in place of oatmeal. Bread also in different forms may be given. The crust of French bread is easily digested, and is supposed to have less starch in proportion to its gluten than the usual home-made bread. It is well to begin with some form of bread of this kind when we are getting the infant accustomed to take starch in the form of bread. If it is constipated, Graham bread and preparations of rye will also be found useful. Fresh bread should never be given, and bread one day old is the preferable form.

The infant should never be given cake or candy, even to taste. I think that it is necessary to state this very decidedly, because it is an erroneous view which is held by most mothers that it can do no harm to give occasionally to an infant in its second year of life, or to a young child, a little candy or a little cake. This may be true so far as the immediate effect these articles may have on the digestion is concerned, but it is of far more importance that the infant should not have its taste perverted from those articles of diet which are best for its nutrition. These new articles appeal more strongly to its sense of taste, and allow it to know that there is something which tastes more agreeable than the food which it is accustomed to have. When an infant has acquired a relish for cake or candy it will cease to enjoy the food by which its development will be best perfected. It is, in fact, kinder to the infant never to allow it to have cake or candy. When these articles are withheld it will continue to have a healthy appetite and taste for necessary and proper articles of food.

The question is so often asked, What is the best method of preparing simple broths for infants? that perhaps it may be well to know how these broths are made.

Chicken Broth.—A fowl weighing about five pounds should be boiled for twelve hours. The fluid should be strained while hot through a fine sieve. It should then be allowed to cool in an earthen jar for twelve hours in the ice-chest. The resulting jelly can be used in full strength or diluted with water. When the jelly has been thoroughly cooled, the fat can be either partially or entirely removed from the top.

Mutton Broth.—A shoulder of lamb, when it can be obtained, otherwise of mutton, weighing from five to seven pounds, is treated in the same way as is the fowl for the preparation of chicken broth.

THIRD NUTRITIVE PERIOD.

The third nutritive period may arbitrarily be made to begin at about the thirtieth month of life.

At this time it is well to begin to accustom the child's digestive functions to a still greater variety of food. In summer the more easily digestible vegetables, such as squash, young peas, and young beans, can be given. The variety of fruits can also be increased at this period, but they should be cooked. The principal change which is to be made in the diet to which the infant has been accustomed is a very decided increase in the proportion of the proteid element of its food. This is accomplished by giving the child meat. The quantity of meat which should be given towards the end of the third year should be small at first, and given at intervals of a day or two. Meat as a regular article of diet for each day is not, as a rule, required until the child is between three and four years old. The kinds of meat which should be given in this early period of childhood are chicken, mutton chop, roast beef, and beefsteak. These meats should be cut into small pieces, and a little salt added according to the child's taste. It is well, during the latter part of the third year and the first half of the fourth year, to give the child an egg on one day and meat on the next.

When the child has reached the age of five or six years we should allow it to have a somewhat more varied diet, but during the whole period of childhood up to the age of puberty the closest attention should be paid to the regulation of the kind and the amount of food, and any deviations from the foregoing rules are to be deprecated.

PICA, OR DIRT-EATING, IN CHILDREN.

BY EDWARD H. SMALL, A.M., M.D.

CHILDREN at times acquire the habit of eating various substances which are "not only wholly indigestible, but are devoid of all attractiveness of taste, and sometimes are perfectly disgusting. They prefer unnatural articles to proper food, which they have to be coaxed to eat." (Thompson.)

To this habit the name of pica has been given, from the Latin word for magpie, on account of that bird's propensity for picking up any or everything within reach. It has also been called dirt-eating, abnormal appetite, geophagia, allotriophagia, chthonophagia, fames canina, cachexia Africanorum, malacia, mal d'estomac, etc.

The disease is not confined to children, but is seen among older persons as well, notably in the so-called clay-eaters of the Southern States, in some parts of South America, the West Indies, Italy, and Africa. It is found among the insane, idiots, and imbeciles. It is akin to the capricious appetite of pregnancy.

The condition is not very common, but it is necessary to have it in mind, for, as Dr. Corrigan says, many of its symptoms resemble those that usher in tubercular meningitis, hydrocephalus, or mesenteric disease. This writer includes in the name pica the habit some children have of stuffing things into their noses.

The habit may be found in children of any age who are suffering from some serious ailment, as rickets, bronchitis, worms, anæmia, or tuberculosis. It generally disappears as the children recover from these diseases.

Or it may arise in perfectly healthy children, as chance offers, and usually disappears in a year or two without anything special having been done to prevent it. By far the greater number of cases are in the last class.

As soon as most children are able to use their hands they are apt to put all sorts of things into their mouths, and in this way the habit may be started. Usually this tendency is soon stopped, as the results are not pleasant. In children with pica it is kept up. The practice of giving babies various articles to put into their mouths to keep them quiet, as the nipple of a nursing-bottle, a piece of cloth, or a sugar-teat, creates in them a desire to have something in the mouth always, no matter what.

Some think that the craving for wall-plaster and chalk is due to a lack of lime in the system. The other abnormal cravings cannot be thus explained.

It has been thought that it is an affection of the pneumogastric nerve or some other form of local neurosis. It is not, however, merely a disease of the stomach, though the stomach may be partly at fault. This craving is rather to be regarded as "a perverted instinct; a minor psychosis; a little localized insanity of one of the lower corners of the mind." (Thompson.) It should be classed with such other bad habits as sucking the thumb, biting the nails, and masturbation. This last habit in children is not to be looked upon as a local neurosis merely, though it might have been started by some local itchiness; no more is pica to be regarded only as a local neurosis of the stomach.

The things which children eat are as various as the substances to which they have access. Generally but one or two unnatural things are taken, but again the child devours whatever can be reached.

Coal, cinders, earth, paper, wall-plaster, chalk, tile, slate, clay-pipe, matches, raw starch, cork, egg-shells, soft soap, bar soap, black lead, asphalt, dirt off the road, toys, mud licked from boots, hair picked from their own heads, have each and all been eaten by children with pica. In some parts of the South, as Alabama, adults as well as children eat the clay found along the banks of streams. The children who eat this clay persist in the habit throughout life, thus differing from those eating other substances, who usually stop the habit at about four years of age. In the West Indies, South America, and Africa it is kept up during life.

Pica is met with in children of various degrees of malnutrition, although some children seem to be perfectly healthy and well cared for. Children with pica have been described as thin, white, weakly, undergrown, flabby looking, pale, hollow-eyed, with dirty complexion, pigeon-breasted, anæmic, and tubercular; they grow old early; their faces soon lose the bright glow of youth. In many cases diarrhœa is met with, or difficulty in defecation, owing to pieces of hard substances, as coal, becoming impacted in the rectum or anus. At times impaction in the larger bowel occurs, and this causes grave symptoms. In some otorrhœa or some other minor trouble arises.

One writer describes the characteristic physiognomy of pica, and two cases are claimed to have been diagnosed by this alone. He says a child with pica "has not the rosy tint of healthy childhood: it lacks clearness, and is dull and unhealthy looking. It is hollow-eyed, with a hungry and unhappy look. It is very difficult to describe exactly, but it is certainly recognizable."

Children with pica are seldom seriously ill as the result of this habit, while in adults death occasionally occurs, as in the West Indies. Few instances of death in children have been recorded. In case of children suffering from constitutional diseases, pica disappears as the accompanying disease improves, or usually in any case when the child is three or four years old.

The habit is difficult to stop, especially in neglected children.

All substances for which the child seems to have a craving should be kept out of his reach. His digestion should be carefully looked after; an abnormal condition of the stomach is sometimes one of the causes of the habit. As a number of these cases arise in children weakened by disease, care should be taken to get the child's health in as good condition as possible.

Change of scene and ideas has a favorable effect. It diverts the mind, and may cause the habit to cease.

When the child is suffering from the presence in the stomach or bowels of substances eaten, these should be got rid of by purgation. If there is impaction in the intestines or obstruction in the rectum which purgation will not relieve, proper surgical measures should be used.

ADVANCES IN THERAPEUTICS.

BY H. A. HARE, M.D.

THE treatment of functional disorders and organic diseases as they are met with in early life requires even more care and thought than does the therapeutics of more advanced years, for while it is true that the child possesses regenerative and recuperative powers far in excess of those of the adult, it is also a fact that minor ailments more seriously disorder the entire system of the child. Further than this, the delicacy of the organism of the child is so great that the physician must not only be careful in selecting his remedies, but equally cautious in deciding what the proper dose of the remedy should be. It is this question of dosage which we believe is disregarded to an extent which oftentimes prevents the accomplishment of the good we desire to achieve. In many instances it will be well, upon employing well-tried remedies in the ailments of children with failure, to regard that failure as rather a result of an error in judgment as to the dose than of an error in judgment as to the remedy.

It should not be forgotten that the extraordinary activity of cell life in the child renders the use of drugs more dangerous, because this activity is more readily perverted or entirely set aside by the introduction of powerful disturbing agents than is the more staid activity of the cells of the body in adults; and, further, that the greater activity of the circulation of the blood and lymph and the ready excitability of the vaso-motor system in children lead to the absorption of most remedies with extraordinary rapidity, and sometimes to their equally rapid elimination. For these reasons the rule in the medication of children should be to use the smallest possible amount of medicine capable of producing the desired effect, and to give this amount not in one or two doses, but in small divided doses frequently repeated. Particularly is this necessary in the ordinary employment of drugs which are desired to act as cardiac or nervous sedatives, and there is little doubt that a part of the success of some irregular practitioners depends upon their custom of dissolving some powerful remedy in a considerable quantity of water, and directing that a teaspoonful or a very minute dose of the real medicine be taken every fifteen minutes till the entire quantity has been ingested. The same principle, in a modified form, obtains in the use of the so-called alterative remedies, such as mercury and arsenic, both of these drugs producing the best result, as a rule, if given to a young child in ascending doses. On the other hand, the alimentary canal and its closely associated glands usually require larger doses of purgatives to affect them in proportion to the size of the individual than does the digestive apparatus of adults, and for this reason fairly large and active doses of purgatives are usually necessary. As a rule, large and concentrated doses of remedies are to be avoided and to be replaced by the graded doses named above.

Again, so far as treatment by drugs is concerned, the influences of every remedy should be closely watched, for children are even more apt to develop idiosyncrasies to drugs than are adults. Finally, the writer wishes to utter a protest against the use of poultices in the treatment of pneumonia, bronchitis, and pleurisy occurring in children. There is no evidence that they do any real good, and not only are they harmful if the greatest caution is not exercised, but they also aid in maintaining the fever and in rendering the discomfort of the patient unbearable.

With the use of climates, baths, Swedish movements, and massage, all of which are in many instances by far the best remedial agents in the management of the diseases of children, this article cannot deal, but the physician should remember them as offering extraordinary benefits, particularly in the more chronic or subacute ailments of young patients; nor should the all-important matter of diet be overlooked, for too frequently drugs are ordered when a proper regulation of the methods of feeding and the character of the food would at once produce relief or cure. All these questions are considered most ably elsewhere in this Cyclopædia. With these remarks as to the general therapeutics of the diseases of children, the writer refers the reader to Dr. Bartholow's article on this subject in volume i., and will devote the remaining space at his disposal to the consideration of some special therapeutic points of interest.

A subject of very great importance from the aspect both of preventive and of remedial medicine is that of the use of the various antitoxins, of which, of course, that of diphtheria is the most useful and widely employed; while the employment of the antitoxins of pneumonia, tetanus, and streptococcus infection are of some promise, but as yet not well enough tried to be generally resorted to by the general practitioner. Again, the use of serum derived from animals or man in the advanced stages of a given disease in persons just entering upon their illness is worthy of note, as is also the employment of the culture fluids of certain micro-organisms in the treatment of malignant growths. Nor should we forget in this connection the moderately successful results obtained by the injection of serum of healthy animals naturally immune to the disease affecting the patient. Further, we have had opened for therapeutic purposes a large field in which have been introduced the use of certain glands in the cure of disease, notably the thyroid in cretinism and myxœdema, and the so-called animal extract methods, many of which are utterly futile and based on an erroneous conception of the influence of non-glandular organs on the body.

The extraordinary results obtained by the use of the antitoxin of diphtheria will, of course, be exploited by the author of the article on that disease. It suffices in this article that attention be called to the collection of statistics gathered about two years ago by William H. Welch, and the valuable data collected and analyzed by the Committee of the American Pædiatric Society in the spring of 1896 and 1897, both of which show beyond doubt the great value of this method of treatment of an otherwise extremely fatal disease.

The chief fact to be emphasized in regard to the application of the diphtheria antitoxin is the prime necessity of injecting it as early as possible. In no illness is delay in treatment more dangerous, and in no disease does the withholding of the remedy so materially decrease the prospects of its achieving good. Again, it is to be distinctly remembered that the antitoxic serum in no way destroys the Klebs-Loeffler bacillus, although its use prevents the spread of the false membrane and seems to render the presence of the bacillus in the throat harmless. Thirdly, the dose of antitoxin must be in direct proportion to the dose of toxine. One dose of antitoxin does not necessarily do the work, but the size of the doses and the frequency of their repetition must be governed by the degree of toxæmia to be antagonized. It should be the rule of the physician in suspicious cases to resort to the antitoxin treatment at once, without waiting for a bacteriological diagnosis to be made, because if the lesion be due to the streptococcus or other organism producing an exudate, the child will neither be benefited nor injured by this treatment. Further, let it not be forgotten that pure diphtheritic infection is comparatively rare, and that in a certain proportion of cases the deaths which take place result from the toxines of other micro-organisms than the bacillus of Loeffler, which cannot be antagonized by the pure antitoxin of diphtheritic infection. Finally, neither antitoxin nor any other power for good can regenerate dead cells or repair hopelessly damaged vital parts if employed after such changes have occurred.

The statistics as to the value of the use of antitoxin serum in croupous pneumonia are scanty, and by no means favorable, at least in the sense in which the antitoxin treatment of diphtheria is favorable.

In regard to the use of the antitoxin of tetanus, there seems reason to believe that some benefit can be obtained by its use, although the disease is so rare in man that the number of cases in which it has been tried is necessarily limited, and these have to be analyzed with care in respect to their results, for it is a well-known fact that whereas the mortality of severe acute tetanus is over ninety per cent., that of chronic tetanus, or that form which lasts for days, has a mortality of only about fifty per cent., or, in other words, half of these cases would recover whether tetanus antitoxin were used or not. According to Hewlett, the average mortality of all cases of true tetanus is about seventy-five per cent., yet in sixty-one cases collected by this writer which were treated by tetanus antitoxin the mortality was only thirty-six per cent. In this list of cases, however, the chronic cases were not separated from the acute ones, and, further, it is claimed that some of the acute cases in Italy treated with antitoxin which died have not been reported. On the other hand, Haushalter asserts that out of forty-four cases of tetanus treated by Tizzoni's antitoxin there were twenty-six cures and eighteen deaths, or a mortality of forty per cent. He also points out that other treatment should be used to aid the antitoxin. Mawson has collected thirty-one cases, of which, however, only twenty-two were completely reported. An analysis of these shows that they can be divided into four classes: (1) cases in which the symptoms commenced to abate immediately after injection and then steadily disappeared, nine; (2) those which remained *in statu quo* for a short time after injection and then gradually improved, six; (3) those in which no further muscles became involved in spasms after commencement of treatment, though occasionally an aggravation of certain other symptoms (as trismus and difficulty in swallowing) occurred, two; (4) those ending fatally, notwithstanding treatment, five. Mawson therefore thinks that there is no doubt that the antitoxic serum has a favorable effect in certain cases of tetanus, and those not always of the mildest form.

On the other hand, Washbourne has called attention to the results obtained by Kantnack in collecting statistics on this subject. From these statistics it would appear that the antitoxin treatment is useless in acute cases with a short incubation period and rapid onset of spasms, while the chronic cases, with a long incubation period and slow onset of spasms, often recover; but, as we have pointed out, the chronic cases frequently do well with other methods of treatment. A definite opinion cannot be formed until a much more extensive trial has been given to the remedy. It must be remembered that in tetanus there is no characteristic lesion at the spot of infection, and a diagnosis is arrived at only when the disease is far advanced; consequently, treatment is commenced at a late stage, and analogy with the experiments conducted upon animals renders the prospect of success not very hopeful.

The treatment of septicæmia by the use of antistreptococcic serum so far promises more than the two remedial attempts just named. Much depends, of course, upon the true cause of the septic manifestations. If they are due to the streptococcus infection, the use of the antitoxin is, of course, of some possible value; whereas if the toxæmia is due to other micro-organisms, it is useless. Further, the limited statistics regarding the use of the antitoxin are, to say the least, encouraging.

Antistreptococcic serum has also been used by Bondot and others in the treatment of erysipelas, and this author asserts that its employment causes marked amelioration in the symptoms.

On the principle that many of the evil symptoms of scarlet fever are due to a toxæmia induced by the streptococcus in addition to the specific micro-organism, Marmorek has employed the antistreptococcic serum in no less than ninety-six cases of this disease. Of these ninety-six cases five died,—four from diphtheria and one from pneumonia. In every case there was a most favorable influence exerted on the swollen cervical glands and the albuminuria was decreased. No serious effect was produced by the injections, but, on the contrary, general improvement. Baginsky has also used this serum in fifty-seven cases of scarlet fever, of which only forty-eight are suitable for analysis. In these forty-eight cases there were seven deaths,—namely, 14.6 per cent., the usual mortality during five previous years varying from 22.6 to 34.4 per cent. The death-rate among two hundred and thirty-eight other cases not treated with serum and belonging to the same epidemic amounted to 24.9 per cent. The cases treated with serum were not of a less severe type than the others. This author concludes that Marmorek's antistreptococcic serum is worthy of a further trial in scarlet fever. The dose of the serum varies with its strength and the severity of the infection, but usually from ten to forty cubic centimetres are used, injected into the loose tissues of the belly wall.

Rapporte has employed antistreptococcic serum containing 0.5 of a one per cent. carbolic acid solution in the treatment of scarlet fever in sixteen cases; in four of these the disease was not grave, and he thinks that the use of the serum prevented the aggravation of the infection; in two cases with symptoms of great infection the serum did not exercise much action; these two patients died. Of the ten other cases to which the serum was given for the purpose of overcoming the symptoms, no less than two succumbed. He also found that the serum did not exercise any material influence over the ordinary course of the temperature, and concludes that its use is not satisfactory. A marked streptococcic infection in addition to the infection of scarlet fever seems necessary in order that this treatment shall be useful.

The use of tuberculin in the treatment of tuberculosis in man has fallen into what is practically an entire and deserved oblivion. It is rarely used for diagnostic purposes in adults, but its employment in children either for diagnosis or for treatment is unjustifiable. Further than this, the employment of the antitubercular serum, prepared with the same idea as is the antidiphtheritic serum, has given very uncertain results, and the injection of serum from animals usually immune to tuberculosis, but otherwise unprepared to resist injections, has not given promise of sufficient good to permit its general use. It is true that a tubercular antitoxin has been prepared from horses which had had their natural immunity increased by inoculations with tubercular virus, but its employment did good only in cases of pure tuberculosis in which the lung was infected by no other organisms than this bacillus. As nearly all tuberculous lungs are so infected, there was little result in the way of good. The dose of this tubercular antitoxin is usually about two or three cubic centimetres, which may be increased to ten cubic centimetres.

The larger doses, however, nearly always produce a marked febrile reaction, which may last for some days, but this does not seem to arise from any absorption of toxic materials from the diseased lung, but rather from an influence directly exerted on the general system by the antitoxin. Sometimes albuminuria develops as a transient symptom. The change for the better under these injections often does not manifest itself for some three or four weeks, and an absence of fever is often the first sign of improvement. In much the same manner that skin eruptions follow the use of diphtheria antitoxin they also follow the injection of the antitubercular serum. They possess no particular significance. So far as the writer is aware, this method of treatment has been used too little in this country to justify its acceptance as a definite therapeutic advance, and the same feeling of uncertainty concerning it seems to exist abroad. On the other hand, Maragliano asserts that his statistics of this method of treatment are that out of four hundred and twenty-two cases a cure was produced in 26 per cent., amelioration in 45.5 per cent., no improvement in 25.5 per cent., and aggravation or death in 8.25 per cent. He also states that this treatment succeeds best in the apyretic forms of tuberculosis. Paquin has tried this method with asserted good results. Richet and Hericourt, who were among the first to use antitubercular serum in this disease, have reported several cases benefited by its employment, and De Renzi has reported twenty-two cases with improvement in every case.

Typhoid fever has also been treated by antitoxic serum to a limited extent, notably by Chantemesse, who in three cases found marked improvement to follow its use. In twelve cases of enteric fever treated by Börger the results were variable. Some benefit was thought to occur in four cases. The remainder were not benefited, and Börger, while unable to support the method, thinks it harmless, even if useless. The best results occurred in the cases in which the serum was used early.

In this connection it is interesting to note that Fränkel and Manchet have treated fifty-seven cases of typhoid fever by intra-muscular injections of sterilized cultures of typhoid bacilli, which, while they produced a fever, in the end favorably modified the disease. Rumpf has treated thirty cases by sterilized cultures of the bacillus pyocyaneus with the same results; of these cases two died. Kraus and Buswell did likewise in twelve cases, and as two cases died they naturally conclude that the method is useless.

Single instances of the use of the antitoxic serum of various diseases have been recorded in whooping-cough, measles (Weisbecker), scarlet fever, typhus, small-pox, and cholera. Kinyoun has reported the use of blood-serum of a vaccinated heifer in the treatment of small-pox. He believes that such treatment modifies the severity of the disease. He tried it in two cases, and one of them died. The decision as to the value of this method has yet to be reached.

In the case of syphilis, several investigators have employed antisyphilitic serum with success,—at least, so they assert. Richet and Hericourt have used the blood-serum taken from a dog inoculated eight days before with blood from a syphilitic patient in a case of old syphilis of twenty years' duration and in one of only eighteen months' duration, with good effects in both, the nervous symptoms of the first and the skin lesions of the latter patient being improved. Gilbert and Fournier, Feulard, Tommasoli, Mazza, Kollman, and Pellizzari have also tried this method with varying results in a considerable number of cases.

In respect to the treatment of malignant growths by the use of a mixture of the toxines of erysipelas and bacillus prodigiosus, by far the best results have been reached by Coley in this country. The early hopes that his methods would relieve a large proportion of cases of inoperable and hopeless sarcoma have failed, for experience has dashed them, but it is an undoubted fact that in some cases the most beneficial results follow the use of this treatment. Much depends upon the strength of the culture, or, to speak more accurately, the activity or virulence of the growth in the culture medium. Probably much of Coley's success depends upon the activity and purity of the bacterial products which he has employed. Coley's studies may be taken as the best series from which to derive conclusions as to this method of treatment. In his most recent paper on this subject he tells us that, grouping the cases so far treated according to their several varieties, we have: Sarcoma, eighty-three: round-celled, fifty-two; spindle-celled, fourteen; melanotic, seven; chondrosarcoma, two; sarcoma, special type of cell not stated, eight. Carcinoma, including epithelioma, sixty-one: of breast, thirty-one; of uterus, three; of cheek, four; of sternum (secondary), one; of tongue, four; of neck, three; of rectum, three; of lip, two; sarcoma or carcinoma, ten cases.

Besides the above cases of malignant tumors, eight other cases have been treated with the toxines. These were as follows: tubercular, two cases; keloid, one; goitre, two; recurrent fibro-angioma, one; mycosis fungoides, one; fibroma, one.

Of the cases of sarcoma, forty-five, or more than one-half, showed more or less improvement. The variety that showed the greatest improvement was the spindle-celled sarcoma; that which showed the least was the melanotic. Next to the spindle-celled in the order of benefit was the mixed-celled, round- and spindle-, then round-celled; while osteosarcoma closely approaches the melanotic in showing but little effect of the toxines. In a series of seven cases of melanotic sarcoma, no improvement was observed in five cases and very slight change in the two others. It should be noted, however, that in most of these cases the tumors were multiple and the disease thoroughly generalized before treatment was begun. Yet in all these cases the antagonistic action of the toxines was so trifling that, were it not for the fact that at least one case of melanotic sarcoma has been cured by accidental erysipelas, there would be little to encourage one to a further trial of the toxines in this class of cases.

With osteosarcoma the results are scarcely more satisfactory. Most of [illegible] cases showed slight improvement; one case, a very large osteochondrosarcoma, was apparently cured, remained well for nearly a year, and then relapsed.

In one case of round-, spindle-, and oval-celled sarcoma (recurrent) the tumor disappeared, and the patient is alive and well more than three years after treatment.

The spindle-celled variety, though embracing a small proportion of the total number of sarcoma cases, forms by far the largest proportion of successful cases.

The results in the treatment of carcinoma were not very encouraging, and this method can hardly be used in this disease with much benefit.

It is worth while to note that in the hands of other surgeons than Coley the results obtained from the joint use of the toxine of erysipelas and the bacillus prodigiosus have been such as to cause many persons to consider their employment futile as to the growth, and harmful to the general system of the patient because of the violent systemic disturbance produced by their use. In two cases in which this treatment was tried by the writer, the approach of death was hurried by their employment and the growth was apparently uninfluenced.

This conclusion is greatly strengthened by the report of a committee of the New York Surgical Society appointed to investigate the value of this method. Their report is as follows:

"Both before and since our appointment as a committee, we have been able to observe, individually and together, a considerable number of cases treated by this means, and in no case have we found any amelioration which held out a prospect of ultimate cure. We have, on the contrary, observed in some cases that the rate of growth of the disease was much more rapid during the treatment. The treatment also imposes a very severe tax upon the strength of the patient, and apparently hastens the cachexia in most cases.

"We believe that in the instances of apparent cure or marked improvement the correctness of the diagnosis is open to doubt.

"We therefore submit:

"1. That the danger to the patient from this treatment is great.

"2. Moreover, that the alleged successes are so few and doubtful in character that the most that can be fairly alleged for the treatment by toxines is that it may offer a very slight chance of amelioration.

"3. That valuable time has often been lost in operable cases by postponing operation for the sake of giving the method of treatment a trial.

"4. Finally, the most important, that if the method is to be resorted to at all, it should be confined to the absolutely inoperable cases."

The treatment should be attempted only in clearly inoperable cases.

Not only have malignant growths been treated by the culture fluids named, but other clinicians have attempted to use serum therapy in these cases. Thus, Emmerich and Scholl have reported six cases of advanced carcinoma treated by serum derived from sheep which had been inoculated with erysipelas. In all these cases the patients greatly improved locally and systemically. The results of these observers have been criticised by Bruns and by Angerer. Richet and Hericourt have reported two cases, one of carcinoma of the stomach and one of fibrosarcoma. Injections of a serum derived from the blood of an ass and two dogs into which the authors had injected the juice of an osteosarcoma produced the most surprising results in both cases, practically a cure in less than a month.

The thyroid gland in its pathological and therapeutic relations to cretinism and myxœdema is of great interest to the physiologist and the physician. Its employment is one of the best illustrations we have of the value of experimental medicine, since it was through the combined results of studies on man and the lower animals that Murray was first led to make practical application of the researches of many Continental and English clinicians and laboratory workers. The observation that myxœdema is usually associated with changes in the thyroid gland, and that extirpation of this gland in man and the lower animals produces myxœdematous tendencies, seemed to point to the conclusion that in such cases the absence of thyroid functions was the underlying cause of the disorder, and the most natural deduction was that patients manifesting such symptoms should be provided artificially with the materials which their own thyroid gland could not provide. At first thyroid therapy consisted in attempting to graft the thyroid of the sheep under the skin of man, but it was soon found that not only was this unsatisfactory, but also that if the gland were given internally it produced equally good results, while it was capable in overdose of producing alarming symptoms. At the present time the dose of the powdered desiccated thyroid gland is from five to ten grains, and of the extract of thyroid, or thyreoidin, two grains. The average thyroid gland of the sheep weighs about twenty-four grains. In this country the preparation usually employed is thyreoidin in capsules or tablets.

Under the influence of this gland myxœdematous patients undergo the most remarkable improvement, their weight decreases, their appearance is greatly modified for the better, and their peculiar mental apathy disappears. In susceptible persons, when full doses are used, there is always some disturbance of the circulation, and it is best during the early part of the treatment to insist upon the patient's remaining in bed. Any undue quickening of the pulse should give warning that the gland is being given too freely. Larger doses are needed in cold weather than in hot weather; and it is to be remembered that while the thyroid treatment of myxœdema is curative in the sense that the patient may be restored to apparently perfect health, it is not curative in the sense of permanent cure unless the administration of the gland is continued in small amounts throughout life.

In cretinism the use of the thyroid gland or its extract is exactly similar to its employment in myxœdema, and the results are extraordinary and far beyond the belief of those who have not actually beheld the improvement.

Thyroid gland has also been used for the treatment of obesity, with varying results. Excessive obesity in childhood is so rare that so far as we know there are no records of its employment in this class of patients. In adults its use has been very successful in some cases, ineffective in others. Thus, in one case of the writer's the patient lost three pounds a week under the thyroid tablets, and in another patient taking tablets from the same bottle no effect whatever ensued.

In the treatment of tetany when dependent upon absence of the thyroid and when due to other causes the thyroid gland has been used with success.

The use of the thyroid gland is contra-indicated by the presence of goitre with exophthalmos, because the overgrowth of the gland is supposed to be associated with an increased internal secretion which causes the peculiar nervous and circulatory disorders of this disease. Under these circumstances it is manifestly unwise to provide the body with a still greater amount of thyroid by its medicinal use. Further, the evidence is that this theory is supported by clinical experience, and that thyroid gland in overdose in healthy persons produces the goitre symptom group. It is, however, only fair to state that Bruns observed a decided improvement in thirty-four cases out of sixty cases of goitre which had thyroid feeding.

On the other hand, we may cite the results of Stabel, who treated ninety-three cases of goitre with thyroid. Out of the ninety-three only four were "cured," and of these two immediately relapsed.

It is also of interest to note that in cases of goitre without exophthalmos the thyroid and thymus glands are of value, probably because in such cases the function of the thyroid gland in the affected individual is impaired.

The use of the thyroid gland in the treatment of psoriasis is to be resorted to only when all other remedies have failed, and it does not always produce good results. In the case of children its use is even more uncertain.

The influence of the thymus gland upon the human being is somewhat allied to that of the thyroid gland, according to certain writers who have obtained good results when using the thymus in mistake for the thyroid gland. Other observers assert that extract of thymus is valuable in true goitre, and that it never produces any of the toxic symptoms when given in overdoses that are caused by overdoses of the thyroid gland. The dose of thymus gland derived from a milk-fed lamb is one gland a day, or from twenty to sixty grains of the thymus extract. It is asserted that the greatest improvement in cases of exophthalmic goitre takes place in connection with the ocular symptoms and the circulatory disorder. It would also seem probable that the thymus gland is useful in cases of goitre without the peculiar systemic manifestations of the form known as Graves's disease. Thus, Mikulicz has treated ten cases of goitre and one of exophthalmic goitre with thymus in the dose of from ten to twenty-five grammes of the minced gland three times a week. The goitre decreases in size very rapidly for fourteen days, but fails to change after six weeks. Out of the ten cases one was rapidly cured, six were much improved, and one was unaffected. In the case of exophthalmic goitre the circulatory phenomena were also greatly improved by thymus. In America, Cunningham has recorded three cases of Graves's disease benefited by thymus gland.

There yet remain to be considered the use of pancreatic essence or the pancreatic gland itself in the treatment of diabetes mellitus, and the employment of bone-marrow, as suggested by Fraser, for the cure or amelioration of anæmia, particularly of the pernicious type.

The use of the pancreas in the cure of diabetes rests primarily upon the observation that disease of this gland which materially interferes with its function often results in the development of glycosuria, and that its ablation in animals is followed by similar effects. Further, in a certain proportion of cases of diabetes the post-mortem has shown the presence of distinctive changes in this gland. The influence of the pancreas in connection with the sugar functions of the digestive apparatus does not stop with the secretion of a ferment which aids in the transformation of starch into sugar, but it is supposed by a process of internal secretion to pour into the blood which passes from its gland cells a substance which aids in the normal assimilation of grape-sugar. There are a few cases on record in which the use of pancreas or its extract has greatly relieved diabetes, but its use is still problematical, so far as its real value is concerned. Naturally benefit will be derived only in those cases in which it is the pancreas that is at fault. Bone-marrow in the treatment of pernicious anæmia is in about the same uncertain position in therapeutics as is the treatment of diabetes just mentioned. There does not seem to be any doubt that its use has been followed by more or less permanent improvement in a few cases, but it is also evident that it is of value only in a few instances which probably call for the use of this source of iron and cell material, as do other cases which call for arsenic and iron. Our use of bone-marrow in any case of anæmia is in one sense purely empirical, unless we know what the cause of the anæmia is. The great difficulty in deciding as to its value is that pernicious anæmia is characterized in some cases by temporary periods of arrest or improvement, which, though they would naturally occur, are attributed to this treatment. In ordinary anæmia bone-marrow may be tried, but there is nothing to show that it possesses as much value as does iron or arsenic. The marrow is obtained from the ribs or the ends of the long bones by soaking the finely minced bone in glycerin for several days and then using the extract so obtained in the dose of one or two drachms a day. Careful attention to the bowels and regulation of the diet are necessary, and in the majority of cases in which the bone-marrow has been used, salol or some other so-called intestinal antiseptic has also been given.

AUTO-INTOXICATION.

By JAMES J. PUTNAM, M.D., and EDWARD W. TAYLOR, M.D.

Definition and Etiology.—To define absolutely what we mean by auto-intoxication is at present impossible, owing to the vagueness of our knowledge. In general we understand by the term those diseases or groups of symptoms brought about by poisons of whatever character created within the body. Such a definition does not clearly mark out the auto-toxic group from certain diseased conditions due to other forms of poison, as, for example, those due to bacterial action. It is, nevertheless, desirable to systematize our knowledge as far as possible by grouping certain toxic agents chiefly from an etiological stand-point. The classification suggested by Van Gieson[1] is a useful one for this purpose.

1. Auto-toxic substances.
2. Bacterial toxines.
3. Combinations of 1 and 2.
4. Extrinsic poisons.
5. Protozoal poisons.

Group 2 does not concern this discussion, since both the action of bacteria and their pathogenic products are particularly treated in other sections of this work. It is no doubt desirable to include products with their causative agents, thereby limiting the field of the auto-toxic group.

Types of extrinsic poisons—Group 4—have been considered in detail under lead and arsenic.

Diseases consequent upon the possible action of protozoa—Group 5—are considered elsewhere. Malaria is as yet the only condition directly attributed to protozoa, but the possibility must be admitted that the exanthemata and mumps may be due to protozoal poisons.

Combinations of the members of the various groups may, and probably often do, occur, but at present our knowledge permits of no specific statements on these points.

The first group—auto-toxic substances—demands, therefore, our special attention. Sharp distinctions between this group and that of the bacterial toxines are particularly hard to draw in the gastro-intestinal tract, where in health as well as in disease auto-toxic substances and bacterial toxines are present. Errors in absorption and general digestive disturbance permit the formation of an active toxic condition, which may, under favorable circumstances, lead to specific disease. Likewise, in the purely chemical digestive process, if toxic products are formed which later are absorbed, an autopoison is introduced into the circulation, which, had it remained in the intestine, would have been innocuous. To go a step further, we may speak of a *secondary auto-intoxication*, when the body is deprived of some chemical substance necessary for the preservation of normal life, as, for example, the withholding of oxygen or the loss of the secretion of the thyroid gland. Various more or less detailed classifications of the auto-toxic group have been attempted. Albu suggests the following:

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[1] The Toxic Basis of Neural Diseases. State Hospitals Bulletin, vol. i. No. 4. To this valuable paper we shall make continual reference in what follows.

1. *Auto-intoxication from the suppression or disturbance of the function of an organ;* for example, myxœdema, cachexia, strumipriva, diabetes, acute yellow atrophy of the liver, Addison's disease, also diseases dependent upon the failure on the part of an organ to destroy poisonous metabolic products.

2. *Auto-intoxications which occur from anomalies in general metabolism without any definite localization.* Gout and oxaluria are examples of this class.

3. *Auto-intoxications which are caused by the retention of the physiological products of metabolism in the different organs.* Herein belong the forms of poisoning due to extensive superficial burns, carbonic acid poisoning in imperfect respiration, uræmia, and eclampsia gravidarum.

4. *Auto-intoxications due to overproduction of the physiological and pathological products of the organism*, such as hydrothionæmia, acetonuria, diabetic coma, and exophthalmic goitre.

Between Groups 3 and 4, and related to both, Albu places the large class of auto-intoxications from the gastro-intestinal tract, with which the production of nervous and presumably mental symptoms is particularly associated. Conditions possibly explainable by this means are tetany, epilepsy, eclampsia infantum, probably certain psychoses, and many other symptoms on the part of the nervous system,—*e.g.*, dizziness, syncope, delirium, coma, sopor, tonic and clonic spasms, epileptoid attacks, maniacal states, and various minor mental disturbances.

Albu makes a further classification from the point of view of the source of the auto-intoxication, as from the skin, lungs, kidneys, suprarenal capsules, gastro-intestinal tract, liver, pancreas, and thyroid gland. Still another possibility is a classification on the basis of physiological chemistry, as indicated by Kraus and Honigman.

Van Gieson points out the dangers of drawing too broad conclusions from the work done up to this time on the toxicity of the urine. The sources of error in isolating toxic substances and the subsequent uncertainty of animal experimentation render many of the results doubtful. For example, it is often most difficult to determine the exact source of an auto-toxic product, whether originally existent in the urine or created by the procedures employed for its isolation. In general, the work on the urine is to be regarded as valuable in bringing evidence towards the general theory of auto-intoxication, provided too much stress be not laid on results hitherto attained.

If we consider the complexity of the chemical structure of the human body and the unstable equilibrium in which many of its component elements must stand, it is easy to realize the possibility of the extensive deleterious effect of altered metabolism as induced by poisons of which we as yet have little knowledge. Either through the development of abnormal and poisonous compounds, or through the failure of the organism to eliminate such poisons as are normally found in its tissues, we have the opportunity offered of resultant profound somatic disorder. In the unstable child organism we may regard these factors as particularly potent, all the more from the fact of a lowered resistance during the period of active growth. Convulsions of childhood and various so-called reflex phenomena are no doubt expressions of irritability, which, according to the toxic view, have as their underlying cause a poison which has gained access to the circulation and thence to the nerve-mechanism, where its final results are manifested. The education of the infant's intestinal tract properly to perform its function is not infrequently attended with errors which lead to temporary outbreaks on the part of the nervous system, due to a transient poisoning. It is possible that future resistance or non-resistance to disease and the diatheses of later life are formed during these early years, dependent upon the kind and intensity of the poison to which the organism is exposed.

Pathology.—The tendency of toxic agents circulating in the system to produce parenchymatous changes in various organs has long been recognized. The special study of the nervous system with relation to its pathological reaction to poisons has been vigorously carried on during the last few years, with results of a positive character. This has been rendered possible chiefly through the exceedingly delicate method of cell-staining first described by Nissl. Recent detailed animal experiments of Berkley by a different method have shown degenerations of nerve-cells as a result of the long administration of alcohol and ricin. Van Gieson has carried on these studies with a large number of toxic substances, including certain bacterial toxines, typhoid fever, diphtheria, and also in experimental and human rabies, uræmia, sunstroke, and experimental thyropravia, with the general result of finding cell changes of a similar sort in all these conditions, whence he concludes that the anatomical expression of a poison acting on the nervous system is that of a parenchymatous degeneration precisely similar in character to such a degeneration occurring in any other organ or system of organs. The degree of the degeneration will, of course, vary in relation to the virulence or the time of action of the given poison, but its character will be invariably the same. It follows, therefore, that the character of the poison is not to be determined from an examination of its results on the nerve-cell. Conversely, however, we may, according to Van Gieson, be dogmatic in our attitude that the existence of a parenchymatous degeneration in the nervous system is evidence of an antecedent poisoning. His own words on the subject are: "Thus, in any acute neural disease, or in the involvement of the nervous system in an acute general illness, even of unknown causation, or in the chronic affection of the nervous system left behind months or years after these two conditions, when *acute parenchymatous degeneration* or its *results* are found, the imprint of this great extensive pathological process on the ganglion cell indicates the action of toxic forces as plainly as the marks on the rocks show the glacial action."[1]

The nerve-cell expresses its beginning disintegration by changes in the deeply staining granules which form a large part of its protoplasm. The tendency of the destructive process is towards a breaking down of the granules into finer ones, until ultimately the characteristic coarsely granular appearance is totally lost. The beginning of such a process Van Gieson terms *cytolysis*, signifying merely cell resolution. It may proceed either to cell destruction, *cytoclasis*, or, under favorable conditions, to restitution, *cytothesis*. In other words, when a certain point in cell degeneration is reached, its restitution is no longer possible, and final necrosis—cytoclasis—is the result. Before that point is reached, however, either a partial or a complete restoration to the normal may take place if the cause which led to the primary change—cytolysis—is removed or weakened in its power.

The degree of cellular alteration is dependent upon the two factors of intensity and duration in the exhibition of the poison. The severity of parenchymatous nerve degeneration stands in direct ratio to the duration of the action of the poison. Recovery from a virulent poison acting for a short time is more likely to occur than in the case of a milder one acting over a long period. Coincident with the various changes in the nerve-cell go the clinical manifestations, and we may see in the tendency to recover or to go on to complete invalidism the counterpart of the cell alterations from cytolysis to cytothesis, or from cytolysis to cytoclasis. In idiopathic epilepsy Van Gieson has not found cytolysis of neurons, which he attributes to the fact that the poison producing, or supposed to produce, the disease is evanescent in character. He, however, believes an overgrowth of neuroglia in the motor areas to be evidence of the action of a poison, inasmuch as a cellular proliferation is an earlier stage of toxic action than a degeneration of the parenchyma.

Symptomatology.—To give an exhaustive account of the symptoms occurring as a result of auto-intoxication is manifestly impossible in the present imperfect state of our knowledge. Bearing in mind, however, the cellular changes already described, certain symptoms and diseases associated with varying degrees of cell alteration may be profitably studied.

As a result of acute poisoning from the gastro-intestinal tract, developing suddenly and acting intermittently over a short period, we may regard the epileptic seizure as the best example. In general, symptoms of an acute and violently spasmodic character, but evanescent, are to be regarded as the clinical manifestation of the sudden development of a virulent poison, whose effect passes off before changes of a permanent character are effected in the cortical neurons. Another and much more definitely established series of symptoms resulting frequently from gastro-intestinal poisoning is that of tetany, less evanescent than epilepsy, but still having a close analogy to it. Should the auto-poison act over a longer period, more definite manifestations of its ravages are found in the cortical neurons, and the symptoms likewise are more persistent. If, again, we suppose an exceedingly mild toxic substance acting but a short time, we should have an explanation for certain attacks of vertigo, eclampsia of children, depression, etc. The vague but important symptoms associated with an acute dyspeptic attack are examples of this class. Again, the symptom of sleep under the condition of fatigue has been thought to be the result of a mildly toxic state, as shown by animal experimentation.

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[1] Loc. cit., p. 420.

It is possible that varied mental symptoms may be induced by the action of toxic agents. The time element and the intensity of the poisonous substance are important in determining the character of the mental manifestation. Thus, an acute delirium,—*e.g.*, typhoid fever,—or a maniacal outburst, or a rapidly developing melancholic state, according to the theory, would be expressive of a cytolysis of cerebral neurons. That symptoms are at one time depressive and again exalted is not susceptible of adequate explanation; but that exhaustion may at times show itself clinically by hyperactivity, as in certain neurasthenic states, is so well known as not to excite particular comment. Delirium may, in a general way, be taken as a type of exaltation of function induced by toxic agents, whereas stupor, confusional states, and coma are symptoms of their depressive action. It is probable that the character of the mental symptoms depends upon the degree of cell alteration, as well as upon the kind of poison in the circulation, and also upon the number of neurons involved in the destructive process. The symptoms of a slowly acting cumulative poison are, of necessity, insidious in their onset and progressive in their course, terminating finally in a permanent condition of dementia. The adolescent form of insanity—hebephrenia—may be included in this category.

Evidence is accumulating to show that symptoms met with in thyropravia, myxœdema, the so-called periodic family paralysis, tetany, and other etiologically still more obscure affections, as, for example, sunstroke, depend for their existence upon a general auto-intoxication of the nervous system.

The possible effect of poisons on the nervous system of the child, if manifested by symptoms which we do not at present thoroughly understand, should become a fruitful object of future study. It is not improbable, in view of the broad general conception of which we have spoken, that the foundation of disease of the most varied sort is laid long before we even suspect its presence, through the subtle action of unknown toxic agents. If this possibility be granted, a careful study of obscure symptoms in the early years of life is more than ever to be practised, combined with a strenuous prophylaxis. Herein, no doubt, lies the chief lesson of the new teaching regarding the poisons.

A demonstration of the truth of the foregoing somewhat theoretical considerations is at present difficult. Cases carefully reported in the necessary judicial spirit are as yet too few upon which to base definite conclusions, and yet occasionally one is extremely suggestive. Thus, for example, Albu reports the case of a boy of ten whose epileptoid attacks were easily and effectively controlled by gastro-intestinal treatment, a return of the convulsions being a regular accompaniment of the suspension of treatment.

The whole problem of the relation of infantile convulsions to intoxication from the intestinal tract, and, secondarily, the relation of infantile convulsions to the graver epilepsy of later life, remains as yet in doubt. This matter will be discussed in other articles. It forms unquestionably one of the most prolific and hopeful fields of investigation relative to the general question under discussion.

Diagnosis and Prognosis.—The conditions resulting from auto-intoxication are too numerous to be considered here in relation either to diagnosis or to prognosis. The individual diseases briefly alluded to are considered in detail in their appropriate sections, so far as they have relation to childhood.

Treatment.—Regarding auto-intoxication from the intestinal tract as a possible source of later disease, certain rules of treatment may be laid down with considerable assurance.

Exercise as much as possible in the open air and frequent baths are no doubt valuable, both as preventive measures and also in improving metabolic activity through increased oxidation and stimulating the skin, and, secondarily, the kidneys, to healthy and vigorous action. A diet composed mainly of milk, with an avoidance of meat during the earlier years of life, is desirable. It is probable that children reared largely on milk are safer against the inroads of disease and develop a greater stability and an increased power of resistance in later life. Particularly is a milk diet to be recommended in those cases where through heredity or any weakening agent, such as an auto-poison, the nervous system is to be regarded as more or less unstable.

In the light of the auto-toxic theory, renewed attention should be directed towards securing free and regular action of the bowels. The cases in which intestinal antisepsis has relieved threatening symptoms on the part of the nervous system should encourage the continued use of this therapeutic measure.

In conclusion, it may be well, in an entirely undogmatic way, to express a certain scepticism regarding the toxic theory in general, and especially as applied to the nervous system. It has occurred to us that possibly in the first enthusiasm of the new theory an occasional confusion of cause and effect has taken place, and that what are taken for results may in some cases be primary and not secondary. Regarding, for example, the nervous system as the regulatory mechanism of the organism as a whole, may it not be that a disturbed state of the nervous system, as manifested in an epileptic seizure, is the cause of the accompanying intestinal disturbance, rather than the converse? This possibility, we believe, remains to be disproved. In any case, careful work from all points of view is necessary.

TOXINS AND ANTITOXINS.

BY VICTOR C. VAUGHAN, M.D.

THE causal relationship of germs to the infectious diseases having been established, the question arose as to the *modus operandi* by which germs cause death. Many theories were advanced to explain the rationale of the process. It is not my purpose to discuss these theories in the present paper. Suffice it to say that it is now a well-established fact that germs cause disease by elaborating poisons which are absorbed and which induce the symptoms of the disease and may lead to death. The study of the bacterial poisons has opened up a wide field for research, which, as yet, has been only partially investigated. It was soon found that certain basic substances are produced by the growth of germs both in artificial culture media and in the animal body. The distinguished Italian toxicologist Selmi proposed that these basic products of bacterial activity be designated as ptomaines, from the Greek word πτῶμα, meaning a cadaver. A ptomaine may be defined as a substance, basic in character, resulting from the growth and multiplication of bacteria. By their being basic in character we mean that they combine with acids forming salts. In this respect the ptomaines resemble the vegetable alkaloids. They have been called animal alkaloids. This appellation was supposed to be suitable because these substances are formed in the animal body. However, it should be borne in mind that they are the products of the growth of bacterial cells, and that these bacteria belong to the vegetable world. The ptomaines are, therefore, vegetable bases. The term "animal alkaloid" certainly is not applicable to these bodies. In the first place, they may be produced by the growth of germs in vegetable media as well as in bouillon and other cultures derived from the animal world. In the second place, the term "animal alkaloid" is more properly restricted to the leucomaines, which are products of the activity of the cells composing the animal body.

Not all ptomaines are poisons. Whether a given substance belongs to this group of bodies or not is determined by its origin and by its basic character. Indeed, most of the ptomaines that have been isolated up to the present time are not poisonous when administered in single doses. However, it is not always easy to distinguish between a poisonous and a non-poisonous substance. The fact that a single dose of a chemical body administered to an animal has no appreciably deleterious effects does not prove that the continued elaboration of this substance in the body of the animal for days, possibly for weeks and months, may go on without detriment to health. No one would assert that thirty grains of quinine administered to a healthy individual in twenty-four hours would cause any marked deviation from health; but we might question the advisability of the administration of this dose of this alkaloid to an individual daily for weeks and months.

All ptomaines contain nitrogen. Some of them contain oxygen, while others do not. The former may be said to correspond to the fixed vegetable alkaloids, while the latter more closely resemble such volatile alkaloids as nicotine and coniine. Since all ptomaines are the products of bacterial growth, it must be evident that the kind of ptomaine formed will depend upon the germ producing it. Thus, the typhoid bacillus elaborates a different basic substance from that found in cultures of the anthrax bacillus. The kind of ptomaine formed is dependent not only upon the nature of the germ producing it, but also upon the medium in which the germ grows. The typhoid bacillus elaborates a characteristic basic substance when grown in beef tea, while in milk cultures of the same germ this substance cannot be found. Furthermore, the production of ptomaines is dependent, to some extent at least, upon the stage of growth reached by the bacterium producing it. All bacterial products are to be regarded as resulting from the growth and development of the germ.

All poisons are toxins; therefore all poisonous ptomaines might very properly be called toxins. However, the term "toxin" has by common usage been restricted to another class of bacterial products, the discussion of which will be entered into directly.

When it was discovered that some of the specific pathogenic germs produce both in artificial cultures and in susceptible animals poisonous basic substances or ptomaines, it was supposed that the symptoms of the diseases induced by these micro-organisms were due in all cases to similar basic bodies, and chemists labored diligently to isolate from cultures of each germ its special basic product. These investigations soon led to a recognition of the fact that this theory had been hastily reached. It was found to be true that the symptoms of each and every infectious disease are due to the chemical products elaborated by the activity of the bacteria, but these chemical products are not in the majority of instances basic in character, and consequently they cannot be called ptomaines. In the study of the etiology of the infectious diseases the ptomaines are of secondary importance among the bacterial poisons. Brieger isolated from pure cultures of the tetanus bacillus as many as four ptomaines, but the poisonous effects of these, singly or combined, are not comparable with those which follow injections of tetanus cultures from which the bacillus has been removed by filtration. The fact that the filtered culture is more poisonous than any or all of its basic constituents necessitates the conclusion that this culture contains some more active body. Results of a similar kind have been obtained with the germs of other diseases. What, then, is the nature of the powerful poisons that are formed in cultures of the bacteria of tetanus, diphtheria, tuberculosis, typhoid fever, anthrax, and other infectious diseases; also in the bodies of animals afflicted with these diseases, and to which the symptoms and death are due? It has been suggested that the specific poisons of the infectious diseases may belong to the class of ferments, and this supposition cannot be regarded as altogether untenable at the present time, neither can it be regarded as an established fact. Brieger and Fraenkel advanced the idea that the diphtheria poison is an albuminous body, comparable with the poisonous proteids found in certain seeds, such as the castor bean and jequirity, or with those contained in the venom of serpents. These investigators obtained from diphtheria cultures an albuminous body which when injected into animals induced the symptoms of the disease. They proposed that the non-basic bacterial poisons should be called toxalbumins. They believed at that time that these toxalbumins have their origin in the splitting up of the proteid bodies in the culture media and in the animal by the action of bacteria. Later researches—a large part of which may be credited to the diligence of these same investigators—have shown that this is not true. Two facts seem at present to be positively proved in regard to the toxins. These are: (1) they are not albumins, and (2) they are not formed by the splitting up of the proteids of culture media or of the proteids in the animal body.

Brieger has obtained the toxins of diphtheria and tetanus in a condition sufficiently near that of chemical purity to demonstrate that they do not belong to the proteid bodies. Uschinsky has proved that bacteria grown in culture media containing no proteids still elaborate their specific toxins. This shows that the toxins cannot be regarded as cleavage products of proteids. It is impossible at present to give any satisfactory definition of a toxin, but we may state that the toxins are specific secretions of the bacterial cells.

It will not be wise within the limits set upon this paper to attempt to discuss all the known bacterial poisons, and we shall confine ourselves to those that are of special interest to the practitioner in dealing with those diseases most frequently met with in children.

THE TOXINS OF TETANUS.—Brieger has obtained four poisonous bases from cultures of the tetanus germ. The first, tetanin, rapidly decomposes in acid solutions, but is stable in alkaline solutions. It produces tetanus in mice when injected in quantities of only a few milligrammes. The second, tetanotoxin, causes first a tremor and then paralysis followed by severe convulsions. To the third no name has been given. This base causes tetanus accompanied by a free flow of the saliva and tears. The fourth, spasmotoxin, induces heavy clonic and tonic convulsions. The same investigator has obtained tetanin from the amputated arm of a man with tetanus, thus demonstrating that this chemical poison is formed in the human body as well as in artificial cultures. It should be understood, however, that the most important poisonous product of the tetanus germ does not belong to the group of ptomaines.

Brieger and Fraenkel secured a toxalbumin from a culture of the tetanus bacillus in bouillon containing sugar. This substance, which consists of the poison mixed with a large amount of the proteid material, is soluble in water, and when injected in small amount subcutaneously in guinea-pigs induces tetanus, which appears in about four days and soon leads to a fatal termination.

Later, Brieger and Cohn obtained the tetanus toxin from cultures of the bacillus in veal broth containing one per cent. of peptone and one-half per cent. of common salt. These cultures were freed from germs by filtration through porcelain. The filtrate was supersaturated with ammonium sulphate. This precipitated the poison, which was found to float on the surface of the fluid. When dried *in vacuo* this crude substance is found to contain 6.5 per cent. of ammonium sulphate. Of the filtered culture 0.00005 cubic centimetre sufficed to kill guinea-pigs with all the symptoms of tetanus. From one litre of such culture one gramme of the above-mentioned substance was obtained, and of this 0.0000001 gramme killed a mouse with the typical symptoms of tetanus. This crude substance contains, besides the poison, albumins, peptone, amido acids, volatile substances, and ammonium sulphate, with other salts. The albumin was removed by precipitation with basic acetate of lead. The peptone, amido acids, and salts were removed by dialysis. Finally, the volatile substances were got rid of by evaporation *in vacuo* at from 20° to 22° C. The toxin obtained in these experiments is a yellow, flaky body, readily soluble in water, without odor, and possessing a taste similar to that of gum arabic. It turns polarized light to the left. It does not give the Millon and xanthoproteic reactions, but does give with caustic potash and copper sulphate a faint violet coloration, not identical with the rose color of the biuret reaction. With the exception of ammonium sulphate, the metallic salts, such as sodium chloride and sulphate, magnesium sulphate, potassium nitrate, mercuric chloride, and potassium ferrocyanide with acetic acid, fail to precipitate the poison as thus purified. Moreover, calcium phosphate, which Roux and Yersin use for carrying down the diphtheria poison, also magnesium carbonate and aluminum hydrate, do not precipitate the tetanus poison. The poison as thus obtained contains no phosphorus and only unweighable traces of sulphur. From the above-mentioned observation Brieger and Cohn conclude that the tetanus poison is not a true proteid.

Of the best preparation obtained by these investigators in the above-mentioned experiments 0.00000005 gramme killed a mouse of fifteen grammes' weight. The authors figure from this that the fatal dose for a man of seventy kilos would be 0.00023 gramme, or 0.23 milligramme, and 0.04 milligramme would induce symptoms of tetanus. The smallest fatal dose of atropine for an adult is one hundred and thirty milligrammes, and of strychnine from thirty to one hundred milligrammes. "From this one can judge of the fearful weapons possessed by the bacteria in their poisons."

More recent researches by Brieger and Boer have shown that when a tetanus culture is treated with mercuric chloride, zinc sulphate, or, better still, with zinc chloride, the toxin is all precipitated. The precipitate thus obtained is wholly insoluble in water. It, therefore, can be thoroughly washed. It is, however, soluble in water containing common salt, or in a feebly alkaline solution. When such a solution is treated with a current of carbonic acid gas the zinc compound is precipitated. Hydrogen sulphide cannot be used for decomposing this compound, because it destroys the toxin. The zinc compound of the toxin as thus obtained still contains a trace of albumin or peptone. They conclude that the toxins of tetanus and diphtheria cannot be considered as ordinary derivatives of albumin. A litre of tetanus or diphtheria culture yields about three grammes of the dried zinc compound, and this contains about three-tenths of a gramme of organic substance, which must represent the total amount of toxin. The zinc compound of these toxins is precipitated by carbonic acid gas, but is not precipitated by mineral acids, by neutral salts, such as ammonium sulphate, nor by phosphomolybdic acid nor phosphotungstic acid. The compound toxins as thus obtained do not give the biuret, xanthoproteic, or Millon test. Neither do they affect polarized light. This shows that these toxins do not consist of peptone, albumose, or albuminate. On being boiled with ferric chloride there appears a distinct red coloration. Whether this is due to the presence of traces of amido acids or not has not as yet been determined. The zinc compound may be decomposed by sodium phosphate, but the toxin which is thus set free adheres closely to the inorganic substances.

Fermi and Pernossi reach the following conclusions from their studies of the tetanus poison:

1. Agar cultures are the most poisonous; next are those on gelatin, and lastly those in bouillon.

2. Chickens, snakes, turtles, and tritons are immune to the tetanus poison.

3. In the above-mentioned animals the tetanus poison may remain and retain its virulence for three days or even longer.

4. Filtrates from agar and gelatin cultures are more resistant to heat than those from bouillon. Like enzymes, the purer the tetanus poison the less stability does it possess.

5. When the tetanus poison is dissolved in water it is rendered inert by a temperature of 55° C., but in the dry state it can be heated to 120° C. without loss of virulence.

6. When the dried poison is mixed with ether or chloroform and heated to 80° C. it is destroyed, but with amylic alcohol or benzole a temperature of 150° C. is necessary to accomplish this result.

7. When dissolved in water this poison is destroyed by direct exposure

to sunlight for from eight to ten hours (with the highest temperature on a blackened thermometer of 56° C.), and after fifteen hours when the temperature does not exceed 37° C.

8. In the dry state the tetanus poison can be exposed to direct sunlight for one hundred hours without loss of virulence.

9. Under the action of an electric current of .5 ampere, continued for two hours, the substance becomes inert.

10. The poison is destroyed by the following substances: lime water, saturated solution, for twenty-four hours; potassium permanganate, fifty per cent., for forty-eight hours; hydrochloric acid, twenty-five per cent., for twenty-four hours; tartaric acid, one per cent., for twenty-four hours.

11. Sulphuric oxide, oxygen, carbonic acid, carbon monoxide, methane, and hydrogen, even after from ten to fifteen hours, do not appreciably impair the poison.

12. Gastric juice destroys the poison through the activity of the hydrochloric acid and not by virtue of the pepsin.

13. Ptyalin, diastase, and emulsin have no action. The effect of trypsin has not been satisfactorily determined.

14. Putrefactive germs do not destroy the tetanus poison.

15. The living, but not the dead, intestines of guinea-pigs and cats destroy the poison.

16. The living intestine of the chick does not destroy and does not absorb the poison.

17. The poison may be eliminated by the kidneys and retain its poisonous properties in the urine.

18. The poison is not a ferment.

The question as to the elimination of the tetanus poison through the urine is one which has been answered differently by different investigators. In several cases experiments upon animals by injecting large amounts of the urine of patients with tetanus have failed to induce the disease. However, this does not show that the poison is not eliminated by the kidney, but only indicates that in these cases the poison was not present in the urine in sufficient quantity to induce the disease in animals. In a fatal case of acute tetanus Vulpius failed to induce tetanus with the urine voided during life, but succeeded with that found in the bladder after death.

Quadu states that the tetanus poison when injected directly into the blood circulates unchanged and unabsorbed for some hours. He also states that a much larger dose is required to induce tetanic symptoms when given intravenously than when given subcutaneously. Moreover, if a sublethal dose be given subcutaneously, the local tetanic symptoms are not intensified by the simultaneous intravenous injection of a second sublethal dose. The observations of this investigator may, however, be explained in another way. It is possible that when the poison is injected intravenously it is so rapidly eliminated from the urine that a larger amount of it is required to induce the disease than when it is given subcutaneously.

Bruschettini has studied the distribution of the tetanus poison through the body in the following manner. Animals were poisoned by injections of the tetanus toxin, and just before death they were killed and bits of various organs were rubbed up with sterilized water and injected into other smaller animals. Emulsions from the liver and suprarenal capsules were invariably without effect, while those from the kidneys were constantly poisonous. This is supposed to indicate that the poison is eliminated by the kidney. The blood taken from the vena cava was found in three out of four experiments to be poisonous. When the injections were made under the skin, the lumbar cord was active in four out of eight cases, and in all when the injections were made directly into the sciatic nerve. On the other hand, when the injections were made under the dura mater, the brain was found to be active, while the lumbar cord remained inactive. From these experiments it is concluded that this poison not only circulates in the blood when injected subcutaneously, but is also deposited in the central nervous system.

Tetanus has been induced in guinea-pigs by injecting into these animals blood-serum obtained by venesection from a fatal case of tetanus in man. With extracts from the liver, spleen, and spinal cord of this case after death like results were obtained in mice.

Ledanter states that the poisonous arrows used by the natives of the New Hebrides are prepared by dipping the points, which are usually made from human bones, first in a vegetable resin and then in slime obtained from swampy places.

Liermann induced tetanus in animals by injecting into them material obtained from the arm of a man who had died from tetanus, and who had been buried for two and one-half years. This would seem to show that the poison retains its virulence for a long time. In this material the tetanus bacillus could not be found. This author explains his results on the supposition that non-pathogenic bacteria may receive toxicogenic properties from the media in which they grow. With the tetanus bacillus widely distributed in the soil, it would not be surprising to find that material taken from the arm of a man dead from any disease after burial for two and one-half years should induce tetanus in animals.

Attempts have been made to find among the chemical products of other germs some antidote for the tetanus toxin. With this in view, Roncali has tested forty different species of bacteria. His results were wholly negative. The tetanus poison was found to be more active in many animals after treatment with the products of other germs, but in no case was there any evidence of antagonism in action.

The Toxin of Diphtheria.—It is evident from the location and limitation of the germ in this disease that the symptoms must be due to a soluble chemical poison. This fact was recognized by Loeffler, the discoverer of the germ of this disease, and in 1887 he attempted to ascertain the nature of the diphtheria poison. He ascertained that evaporation of the filtered culture destroyed its effect upon animals. He also found that ether extracts of cultures were inert. By means of alcohol he precipitated from diphtheria cultures a white substance which when dissolved in water and administered subcutaneously to guinea-pigs caused swelling accompanied by hemorrhage into the muscles and œdema, terminating in necrosis.

Roux and Yersin found that diphtheria cultures filtered through porcelain are markedly poisonous. With such cultures they obtained in animals a serous exudate into the pleural cavity, and marked acute inflammation of the kidney and fatty degeneration of the liver. These symptoms were especially well marked after intravenous injections. After subcutaneous treatments the tissue about the point of inoculation became œdematous and finally necrotic. In some instances paralysis, generally of the posterior extremities, followed injections of diphtheria cultures. They also found that the poisonous substance is precipitated by absolute alcohol, and, furthermore, that it may be carried down mechanically on the addition of calcium chloride to filtered cultures. The great toxicity of this substance is indicated by the statement of Roux and Yersin that four-tenths of a milligramme is sufficient to kill eight guinea-pigs or two rabbits, and that two centigrammes of the calcium chloride precipitate, containing about two-tenths of a milligramme of the pure poison, killed a guinea-pig within four days. Later investigations, however, have shown that the diphtheria poison may be even more potent than is indicated by these figures.

Brieger and Fraenkel obtained from filtered cultures of the diphtheria bacillus a toxalbumin. This substance was found to be insoluble in alcohol, soluble in water, and non-dialyzable. It was precipitated by saturation with ammonium sulphate. It was also obtained by allowing the germ-free filtrate, after being rendered feebly acid with acetic acid, to fall into a large volume of absolute alcohol. It was purified by repeated solution in water and reprecipitated with alcohol. This proteid induces in animals all the symptoms and post-mortem appearances which had been observed to follow the administration of filtered cultures. It is worthy of note that the injection of small quantities of this substance (2.5 milligrammes per kilogramme of the body-weight of the animal) did not produce its effects until after the lapse of weeks. This action is considered by some to indicate that the diphtheria poison is a ferment.

All the above-mentioned poisonous products obtained from diphtheria cultures are to be regarded as very impure preparations of the toxin. The diphtheria poison has been obtained by Brieger in the form of a zinc compound, in a state of purity similar to that in which he found the tetanus poison, already mentioned.

Tangl has shown that the chemical poison is formed in the body as well as in culture-flasks. A large piece of pseudo-membrane was macerated in water in an ice-chest for twenty-four hours and then filtered through porcelain. The filtrate injected into animals produced all the symptoms that had been obtained by the similar employment of artificial cultures. Tangl also observes that in some cases in which the animals were inoculated with a sterilized culture through the mucous membrane a pseudo-membrane formed at the point of injection. The diphtheria toxin has also been found in the tissues, in the blood, and in the urine.

The preparation of diphtheria toxin has become a matter of considerable importance, inasmuch as it is used in rendering animals immune to the disease, and in thus securing the antitoxin. The toxin for this purpose is prepared by growing a virulent culture of the diphtheria bacillus in a thin layer of culture-fluid exposed to a current of air. After a certain degree of virulence has been reached, the culture is treated with a small quantity of carbolic acid, and is used for immunizing animals.

Schierbeck states that the production of toxin by the diphtheria bacillus is favored by the presence of free carbonic acid, and he recommends that the culture be treated with a current of air containing eight per cent. of this gas.

This base was not obtained in quantities sufficient for analysis. The platinum chloride compound crystallizes in rhombic prisms, and the hydrochloride in needles. The red color seems to be inherent to the substance and not due to impurities. The platinum compounds are insoluble in alcohol, soluble in water. The hydrochloride is soluble in both water and alcohol.

ANTITOXINS.

We have very little positive knowledge concerning the nature of antitoxins. It is generally believed that they are proteid bodies, but this belief is not founded upon any sufficient or positive knowledge. The most exhaustive study yet made on the chemistry of antitoxins is that of Brieger and Boer. These investigators have studied the antitoxins of tetanus and diphtheria. For a preparation of the tetanus antitoxin they used blood-serum obtained from a goat, having an immunity value of from eight hundred thousand to three million. For the isolation of their diphtheria antitoxin they used the blood-serum of a horse representing one hundred normal immunity units. They found that those substances which precipitate the proteids in blood-serum carry down with the precipitate the antitoxin. Among agents of this kind they have used ethylic alcohol, methylic alcohol, acetone, acetic acid, ferrocyanide of potassium, and the mineral and organic acids in various dilutions. The precipitates formed by these reagents become insoluble in water after having been dried. However, the dried precipitate can be dissolved in very dilute alkali, and this does not affect the antitoxin. Alcohol and strongly acid reagents destroy antitoxin. The neutral salts, such as ammonium sulphate, magnesium sulphate, etc., also precipitated the albuminous bodies and carried along in the precipitate the antitoxin. They found, however, that by making certain combinations of the neutral salts, and by attention to the temperature at which the processes are carried out, antitoxin may be wholly precipitated from blood-serum. One method of procedure is as follows.

Ten cubic centimetres of blood-serum are diluted with an equal volume of distilled water. To this there are added four grammes of potassium chloride or potassium iodide. The fluid is then shaken with from four to five grammes of finely pulverized sodium chloride, and the mixture placed in an incubator. After standing at a temperature of from 30° to 37° C. for twenty hours, the whole of the antitoxin will be found in the precipitate. In this manner they obtained from ten cubic centimetres of the diphtheria serum four-tenths of a gramme of dried substance which was soluble in its own weight of water, and which was found to possess all the antitoxic value originally possessed by the ten cubic centimetres of blood-serum. It must be understood that this precipitate is not pure antitoxin. It contains, in addition to the antitoxin, albumin and salts.

Another method of preparing antitoxin used by these investigators is as follows. Ten cubic centimetres of the blood-serum diluted with five volumes of water are mixed with twenty cubic centimetres of a one per cent. solution of zinc sulphate or zinc chloride. After standing for a short time the precipitate is collected on a filter. There will often be subsequently observed in the filtrate a cloudiness, but this may be disregarded, as the first precipitate contains all the antitoxin. The precipitated antitoxin may be washed sparingly with water. It is soluble in large excess of water. The precipitate is then dissolved in twenty cubic centimetres of water to which one drop of normal soda solution has been added. The filtrate is treated with a current of carbonic acid gas. If zinc sulphate has been used in the precipitation of the antitoxin the zinc compound is precipitated by the carbonic acid gas, but if zinc chloride has been used the antitoxin remains in the filtrate. The antitoxin compound is now dried in a desiccator. The residue or the dried substance is washed with water, which removes the zinc albuminate but leaves the zinc antitoxin undissolved. The latter may be taken up with very dilute alkali, and be again treated with carbonic acid gas and washed with water; the greater part of the zinc may be removed by ammonium sulphide or hydrogen sulphide. By this method Brieger and Boer obtained from ten cubic centimetres of diphtheria serum one-tenth of a gramme of the powder, readily soluble in water, and possessed of all the antitoxic value previously existing in the ten cubic centimetres of serum.

Up to the present time we can recognize the presence of antitoxins only by their effects. Behring states that the diphtheria antitoxin is not altered by peptic digestion. If this be true, it cannot be either serum-albumin or globulin. The same author believes that the action of antitoxin on toxin is a chemical one, and he compares it to that of an acid on an alkali, or to that of soluble sulphates on phenol in the body. On experimental grounds Buchner combats this idea. He finds that the mixture of tetanus toxin and antitoxin, which has no effect on mice, kills guinea-pigs, and he argues from this that if the antagonism were a chemical one, a mixture of given quantities of toxin and antitoxin found to be harmless to the mouse should also be harmless to other animals. This reasoning seems to be good and conclusive. There is no positive proof that toxins and antitoxins neutralize one the other *in vitro*. Moreover, as pointed out by Roux, a mixture of the diphtheria germ and antitoxin injected into an animal previously treated with bacillus prodigiosus, streptococci, etc., is fatal, although a similar mixture injected into a normal non-treated animal is without effect. That a mixture of toxin and antitoxin which has been kept in a test-tube for so many minutes fails to produce any effect when injected into an animal does not prove that neutralization has taken place *in vitro* or is chemical. It may have taken place *in vita* in the body and be physiological. In fact, the experiments of Buchner, Roux, and others furnish strong support of the view that antagonism between toxin and antitoxin is physiological. It has been suggested that antitoxin is the toxin modified by the fluids and tissues of the body. This undoubtedly is true. But in what does this modification consist?

The possibility of converting toxins into antitoxins by means of electricity was first suggested by Smirnow. He used the diphtheria toxin, and was partially successful. He explained this conversion by supposing it to be due to change in reaction in the fluid. Arsonval and Charrin and Bolton have experimented along this line also with partial success. Bonome and Viola conclude their experiments on converting the streptococcus toxin into antitoxin with the following statements:

1. A high alternating current renders the most virulent cultures of the staphylococcus pyogenes harmless without changing the reaction of the fluid or affecting the form of the germ.

2. The action of this current is confined to the toxin. The germ when transferred to other tubes grows and manifests its full virulence.

3. The toxin is transformed into antitoxin analogous to that of the blood-serum of immunized animals.

4. The antitoxin thus formed is very active and will neutralize *in vitro* ten times the usually fatal dose of a culture of this germ. However, from a recent culture only a small amount of antitoxin can be obtained, because such cultures contain but little toxin.

5. This antitoxin prepared by the electric current possesses powerful protective and curative action against streptococcus infection.

6. While this antitoxin is a true vaccine like that obtained by heating cultures to 55° or 60° C., it is not without injurious effect, and induces in rabbits a progressive marasmus preceded by an elevation of temperature.

7. The antitoxin thus prepared possesses the property of developing in rabbits substances which destroy the germs *in vitro* after long exposure by causing degenerative changes in the bacterial cells.

THE PRODUCTION OF ANTITOXINS.—A very brief *résumé* of the method of producing antitoxins will be given here. In 1880 Toussaint rendered animals immune to anthrax by treating them with the defibrinated sterilized blood of animals dead with this disease. This probably was the first time that dead cultures were used in the production of immunity. In 1886 Salmon and Smith rendered pigeons immune to the bacillus of hog cholera by treating them with sterilized cultures of this germ. In 1887 Sewell rendered pigeons immune to the poison of the rattlesnake by starting with a non-fatal dose and gradually increasing the amount. That this author comprehended at that time the significance of the results that he obtained is demonstrated by the following quotation: "This work was undertaken with the hope that it might form a worthy contribution to the theory of prophylaxis. I have assumed an analogy between the venom of the poisonous serpent and the ptomaine produced under the influence of bacterial organisms." Fraenkel and Simmonds, also Beumer and Peiper, in studying the pathological significance of Eberth's bacillus, observed that when an animal recovered from the effects of either sterilized or unsterilized cultures, and was again inoculated with the germ, it bore the second time a much larger quantity than that ordinarily required to cause death. Roux and Chamberland immunized animals against malignant œdema and symptomatic anthrax with sterilized cultures, and Chantemesse and Widal did the same with the Eberth bacillus. About the same time Foa and Bonome not only rendered animals immune to the proteus valaris, the diplococcus of pneumonia, and the bacillus of chicken cholera, by treating them with sterilized cultures of these germs, but they discovered that the blood taken from the heart or an infusion of the tissues of a rabbit dead from proteus infection injected intravenously into another rabbit made the second one immune to virulent cultures of the germ. Moreover, they observed that the blood and tissue infusions with which they induced immunity formed good culture media, on which the proteus grew abundantly and remained possessed of full virulence. The protected blood had no germicidal properties.

In 1890 Behring began his work on immunity and cure. He rendered animals immune to diphtheria and tetanus by the subcutaneous injection of the specific toxins of these diseases in gradually augmented doses. He ascertained that in this manner the resistance of the animals could be increased a thousand-fold, and that the blood-serum of such an animal could be employed not only to give immunity to men and animals but also to effect cures. He thus describes the effects of large quantities of virulent cultures of the diphtheria bacillus upon animals protected in different degrees with the serum of immunized animals. If one inject into a guinea-pig which has just been infected with ten times the fatal dose of a diphtheria culture an amount of the normal curative serum in the proportion to the body-weight of the animal of 1 to 5000, death does not result, but the animal sickens. At first the sickness is similar to that in a control animal. There is a local œdema that becomes harder and more extensive day after day and is filled with a firm fibrinous exudate. Generally about the eighth day, sometimes only after some weeks, a line of demarcation forms about the tumor, which is often as large as a child's head. The separation continues until the skin peels off, leaving a permanently bare spot, which covers over with tissue. With the same large quantity of virulent culture, but with an increase in the amount of the serum to the proportion of 1 to 2000, there is a slight infiltration, which is soon absorbed, and nothing abnormal can be observed after ten days. With the serum increased to 1 to 5000, the animal remains wholly free from any evidence of the disease.

In 1891 Ehrlich established a high degree of immunity in animals against two of the most powerful vegetable poisons known,—ricin from castor bean and abrin from the jequirity bean. Immunity was easily established by feeding the animals upon small and gradually augmented doses. These poisons are apparently proteid, and in this respect as well as in their great toxicity resemble the venom of snakes and the toxins of the pathogenic bacteria. Immunity established against one of these poisons does not hold good against the other. Later the same investigator showed that a nursing animal taking its nourishment from an immunized mother may receive immunity with the milk.

Fraser and Calmette have established in the lower animals marked immunity against the venom of snakes and have succeeded in obtaining an immunizing serum. Experimental immunity has been established in one or more species of the lower animals by the employment of sterilized cultures of the specific bacteria in anthrax, symptomatic anthrax, cholera, chicken cholera, hog cholera, diphtheria, erysipelas of the hog, influenza, pneumonia, pleuro-pneumonia, swine plague, streptococcus infection, tetanus, typhoid fever, and bubonic plague. Babes states that he has rendered guinea-pigs immune to glanders by the employment of sterilized cultures. However, mallein has not been shown to be of signal value either as a protective or as a curative agent, and its sole use at present is for diagnostic purposes. Many claims have been made for the immunizing of various animals to tuberculosis. The latest of these is that announced by Koch, in which he has used his new form of tuberculin.

The protective value of the serum obtained from an immunized animal might be estimated quantitively according to the following standards: 1. Its immunizing power against infection. 2. Its curative power against infection. 3. Its immunizing power against intoxication. 4. Its curative power against intoxication.

1. In order to determine the immunizing power of a serum against infection a very virulent culture of the diphtheria bacillus (in case of this disease) is prepared, and the smallest fatal quantity of this per kilogramme of body-weight of guinea-pig is determined by experiments on numerous animals. This being ascertained, the work is facilitated and time saved by making ten times the smallest fatal dose the unit in estimating the strength of the serum, because if the smallest dose was used death would result only after many days, and some animals would escape death from the smallest ordinarily fatal dose. By taking ten times the smallest fatal dose death is certain in the control animals, and whether or not the treated ones will suc-

cumb is known after a delay of a few days at most. The difficulties met with in making this determination are the following: (*a*) The diphtheria culture should be of great virulence, and such cultures cannot always be easily and readily obtained. (*b*) Having obtained a culture of the desired virulence, it does not continue of constant strength, but is likely to change from day to day; or on account of a variation of a few degrees in the thermostat in which it is kept it may quickly become worthless. Therefore this determination is not made in the preparation of serum.

2. To determine the curative value of a serum against infection would be a still more complicated task and the results less certain. The virulence of the culture, its age, the temperature at which it had been kept, the lapse of time between the infection and the beginning of the treatment, are factors which it would not be always possible to measure and which would affect the results.

3. A determination of the immunizing power of a serum against intoxication is easily made and gives uniform results. This is the quantitative measure which is now employed in the preparation of protective and curative sera. A culture of marked virulence is prepared and sterilized. This contains a toxin which will retain its properties unchanged indefinitely. It can be kept for months and employed in determining the strength of a large number of samples of serum. The smallest fatal dose of this toxin is determined, and, for the reasons already given, ten times this amount is used as the unit for determining the strength of the serum.

4. Behring and others made frequent tests of serum as a curative agent in animals to which more than a fatal dose of the toxin had been administered and in which the symptoms had already developed. Even when death seemed imminent a dose equivalent to six times the amount necessary to immunize against the same quantity of the toxin saved some of the animals.

From the facts already given it may be concluded that certain poisons, such as the venom of snakes, the blood of snakes, toads, and salamanders, certain vegetable poisons, abrin and ricin, and the bacterial toxins, awaken, and, when repeatedly induced in less than fatal doses, develop in the animal body an antagonistic action which tends to protect the body against the effects of these poisons.

I shall now proceed to give a brief statement of the practical method employed in securing antitoxic serum. In order to prevent repetition I shall confine myself to the method used in the preparation of antitoxin for diphtheria. Various animals may be used, but for practical purposes the horse is the one now universally selected. The animals chosen for this purpose should be young and in perfect health. Before the process of immunizing is begun the animal should be examined by a competent veterinarian and its freedom from disease should be determined. It is customary in this preliminary examination to test the animal for latent glanders by inoculation with mallein. The temperature should be taken twice a day for a week before the injections are begun. When the animal has satisfactorily passed through all these tests the process of immunizing may be begun. Usually about fifteen drops of the diphtheria toxin diluted with several times its volume of normal salt solution constitutes the first injection. The reaction produced by this injection is generally quite marked; the temperature rises from 1° to 5° F. The animal gives distinct evidence of being seriously sick. It refuses food, and shows some difficulty in locomotion. These symptoms gradually abate, and within from three to seven days the animal regains its normal state. The time intervening between the first and the second injection must depend upon the extent to which the animal is affected and the rapidity with which it recovers. However, it is usually from seven to ten days. The second injection usually varies from one to two cubic centimetres. When the latter amount is used a certain proportion of the horses die from its effects. However, those that recover are more likely to prove good antitoxin producers. Some prefer to give the larger amount, and in this way determine the suitability of the horse for the production of antitoxin. About one-third of the horses selected prove unfit for the purpose. They do not yield a large amount of antitoxin, and after a while develop diphtheritic paralysis and die. Indeed, the variation shown by individual horses in their susceptibility to the toxin of diphtheria is quite marked. The quantity of the toxin injected is now rapidly increased, the injections being made usually at intervals of one week until after three or four months from three hundred to five hundred cubic centimetres are injected at one time. After the first three or four injections the horse usually shows but little disturbance; the temperature may be raised one or two degrees, and there may be some disinclination to take food, but these evidences of illness disappear within a short time. Indeed, it sometimes happens that animals gain considerable weight during the process of immunizing. The first injections are made subcutaneously, while the later and larger ones are given intravenously. The temperature of the animal is taken twice a day, and it is given some exercise every day save the one following an injection. After this treatment has been continued for about three months some of the blood is drawn and its antitoxic power is measured. On drawing the blood the skin over the jugular vein is shaved and disinfected, and a sterilized trocar is introduced into the vein. The blood is received into a sterilized vessel. The blood is allowed to clot while standing in an ice-chest. After forty-eight hours or longer the serum is drawn off, and usually some preservative is added. The preservative in most common use is trikresol. This is believed to be less poisonous than carbolic acid, and it is for this reason that it is selected. Camphor has sometimes been used as a preservative, but it has no such action. The value of the blood-serum thus obtained is now to be determined. In order to do this a half-grown guinea-pig is inoculated with a mixture of ten times the minimum fatal dose of the toxin and a small quantity of the serum. It is customary to inoculate four guinea-pigs. One of these receives ten times the fatal dose of toxin and one-five-hundredth of a cubic centimetre of the blood-serum. The second one receives the same amount of toxin and one-one-thousandth of a cubic centimetre of the serum. The third has the toxin and one-fifteen-hundredth of a cubic centimetre of the serum, and the fourth the toxin and one-five-thousandth of a cubic centimetre of the serum. If the fourth guinea-pig dies while the others live, the serum is said to contain in every ten cubic centimetres fifteen hundred units. It will be seen that it contains at least this much, but it may contain much more. Some manufacturers filter the serum through porcelain, while others do not. Filtration through porcelain secures a clearer preparation and lessens the possibility of infection, but it reduces the antitoxic power. When the serum is filtered its antitoxic power should be again determined. In the case supposed above, the filtrate after passing through porcelain is tested in order to see whether or not it contains fifteen hundred units. Should it fall below this amount it is tested in order to see whether it contains one thousand units or not. The figures given on the bottles generally understate the antitoxic value of the serum. This is done in order to be sure that the amount indicated will be possessed by the serum even after weeks and possibly after months. Within the last year a diphtheritic serum containing five thousand units has been obtained, and it is not probable that the end has yet been reached so far as the strength of the serum is concerned. In more powerful preparations the dose necessary to cure or to protect may be reduced.

It is hardly desirable in this paper to discuss unusual methods of immunizing animals, nor is it necessary to give the methods of preparing other kinds of protective and curative serum.

Dungern has shown that the diphtheria bacillus produces more toxin when grown in culture media containing ascitic fluid from man than in ordinary culture media. According to Spronck, the most active toxin is formed when the bacillus is grown upon bouillon prepared from meat several days old in which the sugar has been decomposed. The peptone used in the culture also should contain no glucose.

Kossel has shown that the specific toxin of diphtheria is a secretion of the bacterial cell. Its formation begins with the growth of the bacilli, and it appears in cultures before the cells begin to break down and their contents pass into the solution. When the bacilli die, the production of the poison ceases. In this way we understand that the exaltation of the diphtheria bacillus consists of a development of its specific function.

THE TOXINS OF TUBERCULOSIS.—Koch's tuberculin is an impure preparation of the toxin of the bacillus tuberculosis. The crude tuberculin first prepared by Koch was obtained in the following manner. Meat infusion containing one per cent. of peptone and from four to six per cent. of glycerin was placed in sterilized flasks with broad bottoms. The flasks were only partially filled, in order that the surface of the fluid should be as great as possible. A small mass of a growth of tubercle bacilli was taken from a culture on glycerin, agar, or blood-serum, and floated on the surface of the meat infusion in the flask. The flask was then placed in an incubator at 37° C. In such flasks the bacilli grow abundantly on the surface of the meat infusion, forming a thick yellowish-white layer. After about six weeks growth stops, the bacterial film also begins to break into pieces, and these fall to the bottom of the flask. After having reached this stage, the culture was evaporated to one-tenth of its volume on the water-bath. The concentration increased the percentage of glycerin to from forty to fifty, and this ingredient prevented the growth of extraneous bacteria and rendered the fluid permanent for an indefinite time. It will be seen that this preparation is a complex one. It must contain in addition to the water and glycerin any other unchanged constituents of the original meat infusion, any split products, if there be such arising from the cleavage action of the bacilli on the components of the culture media, and the solid constituents of the bacterial cells. It is evident from the method of preparation that the crude toxin is not destroyed by the temperature of the water-bath. Ultimate analyses of this tuberculin have been made, but it must be evident from the statements just given concerning the complexity of its composition that such analyses are absolutely without value.

The crude toxin obtained as above described has proved of great value as an agent for the recognition of tuberculosis in its early stages. It is now extensively employed and confidently relied upon for the recognition of this disease in cattle. While the tuberculin reaction cannot be regarded as absolutely infallible, it is undoubtedly the surest means in our possession for the early recognition of this disease. Some are now employing it as a diagnostic agent in man. Koch states that under his direction it has been used for this purpose in more than one thousand cases without the slightest injury or harm being done to the individual.

Recently (1897) Koch has made a second most valuable contribution to our knowledge concerning the chemistry of the cell of the bacillus tuberculosis. The following is an abstract of the additional information that he has given us upon this point.

In the first place, he has extracted tubercle bacilli with one-tenth normal soda solution. In doing this, the bacilli were stirred up in the solution and allowed to stand at the temperature of the room, with frequent agitation, for three days. At the expiration of this time the fluid was filtered through paper and neutralized. In this manner there was obtained a faintly yellow fluid, which was found never to be wholly free from bacilli. It contained in the ordinary cover preparation from five to ten bacilli in each field. These bacilli were never found in clusters, but always single. Of course the germs after this treatment are dead. This preparation, because it is an alkaline extract, is designated by Koch as TA. Experiments made with TA show that with very small doses a reaction similar to that caused by the original tuberculin follows. The only difference is that with TA the reaction is more marked and of longer continuance. With relatively large doses abscesses may form about the point of injection. These abscesses are filled with sterile fluid, and may contain the dead bacilli. In order to remove the bacilli TA was filtered through porcelain. However, it was found that not only the bacilli but also a considerable amount of colloid substance remained on the filter. The filtrate was found not to produce abscesses and not to cause the reaction of the original tuberculin.

Koch regards the formation of the abscess by the injection of TA as an important fact, and holds that it teaches us that immunity against this disease cannot be secured by the subcutaneous injection of any fluid containing tubercle bacilli. Of course the amount of this substance might be so small that abscesses would not follow. This is accounted for by the fact that the subcutaneous tissue is able to dispose of a very few dead bacilli without injury to itself.

Former researches had convinced Koch that the tubercle bacilli contained at least two characteristic chemical bodies which belonged to the unsaturated fatty acids. One of these fatty acids is soluble in dilute alcohol, and is easily saponified with sodium hydrate. The other is soluble only in boiling absolute alcohol or ether, and is saponified with difficulty. Both of these take the characteristic stain of the tubercle bacilli; that is, they are colored an intense red with carbolic fuchsin, and retain this color after treatment with dilute nitric acid and with alcohol. Tubercle bacilli from which the first of these fatty acids has been removed by cold alcohol still respond to the stain-test for the germ. After the removal of both of these fatty acids from the bacilli the cell still retains its form, but no longer gives the specific coloration.

Finally, Koch has adopted the following method of preparing extracts of the tubercle bacilli. A well-dried culture is placed in an agate mortar and rubbed with an agate pestle until only a few of the bacilli respond to the specific stain. The powder thus obtained is suspended in distilled water and placed in a centrifugal machine. After this has revolved at the rate of four thousand revolutions per minute for from one-half to three-fourths of an hour, it separates into a supernatant opalescent fluid, which contains no bacilli, and a sediment. The sediment is removed, dried, again rubbed in a mortar, and again placed in the centrifugal machine. There are now obtained a clear supernatant fluid and a solid sediment. These manipulations can be repeated as long as any extract is obtained from the bacilli.

The extracts thus obtained from the tubercle bacilli have been injected into men and animals; they do not produce abscesses. It has been found, however, that the extract first obtained differs from the second and subsequent ones. The extract first obtained in the centrifugal machine is designated by Koch as TO, and the subsequent extracts as TR. The addition of fifty per cent. of glycerin to TO causes no change, while in TR it produces a white flocculent precipitate. This indicates that TR contains substances extracted from the tubercle bacilli which are insoluble in glycerin, while TO contains those substances which are soluble in glycerin. Corresponding to these chemical differences, these preparations act somewhat differently upon men and animals. TO resembles the original tuberculin and the preparation which has been already designated as TA, with the exception that TO does not cause abscesses. Moreover, TO has but little effect in the production of immunity. On the other hand, TR acts differently so far as the production of immunity is concerned. It is true that when given in large doses it causes the tuberculin reaction, but its most important effect is entirely independent of this reaction. While in the use of the original tuberculin and of the preparations here known as TA and TO the so-called tuberculin reaction must be induced in order to get any curative effect, Koch attempts in his employment of the TR preparation to avoid the tuberculin reaction. By beginning with small doses of the TR extract and gradually increasing them, Koch claims that animals can be made immune to the TR preparation, and finally to the tubercle bacillus itself.

Koch states that not every culture of the tubercle bacillus is suitable for the preparation of the active TR extract. He has convinced himself that the tubercle bacillus is more variable in its virulence than has been heretofore supposed. Cultures kept for a long time in the laboratory and grown on artificial media lose their virulence in part. For the preparation of the TR extract the most virulent cultures should be employed, the less virulent ones furnishing a less active or a wholly inert preparation. The cultures must not be too old; in fact, they must be used in as early a stage of their development as is possible. The bacilli must be dried *in vacuo*. Otherwise the substance becomes less soluble and furnishes a less active preparation. The essential constituent of TR is shown to be highly susceptible to chemical and physical influences. The preparation must be carefully excluded from the light.

Koch cautions against attempts to prepare these extracts on a small scale, and claims that they should be manufactured only in laboratories specially fitted for the purpose. Certainly the process of rubbing up the highly virulent tubercle bacillus in a dry powder in a mortar is not to be regarded as altogether free from danger. Koch states that in doing this himself he did not use a respirator, but that he regards the preparation as scarcely less dangerous than working with highly explosive material. The TR extract is preserved by the addition of twenty per cent. of glycerin, which is not enough to cause the precipitation of its active constituents.

The practical application of this preparation is similar to that of the original tuberculin. Injections are made under the skin of the back with sterilized syringes. The fluid contains in each cubic centimetre ten milligrammes of solid substance, and when used it is diluted with physiological salt solution to the extent desired. The beginning dose is one-five-hundredth of a milligramme. Only in exceptional cases is the use of this amount followed by reaction. If the reaction should occur, a smaller dose is employed. The injections are repeated every second day, gradually increasing the dose, avoiding elevation of temperature. When there is an elevation of temperature following an injection, subsequent treatment must be delayed until the normal temperature has been regained. Koch states that, as a rule, he has increased the dose of the TR preparation until he has reached twenty milligrammes. In immunizing animals, from two to three milligrammes are used as a beginning dose in guinea-pigs; correspondingly larger amounts for other animals. In animals already tuberculous, the beginning dose must be smaller.

Koch states that by proceeding in this manner he has succeeded in rendering a large number of guinea-pigs wholly immune against inoculation with the most virulent cultures of the bacillus. In some of these animals there was no reaction at the point of injection, and the inguinal glands remained wholly unchanged for months. In others these glands were slightly enlarged, but were found not to be tuberculous. Some animals at the time of inoculation proved not to be immune. In these the inguinal glands underwent caseation. The internal organs, however, were free from tuberculosis, while control animals showed widely diffused general tuberculosis of the lungs, spleen, and liver. In other animals still less immune, inoculations were followed by tuberculosis of the lungs, while the liver and spleen showed only traces of the disease.

Tuberculous guinea-pigs treated with injections of the TR extract showed without exception more or less marked regressive changes in the tubercular areas. For instance, in the liver, instead of necrotic areas one would observe depressions on the surface, which were so numerous in many instances as to make the surface of the organ very uneven. In the spleen contractions were observed, and in some instances these involved so large a proportion of the organ that the part remaining was very small compared with the normal size. Koch states that he has been able to fully immunize guinea-pigs within from two to three weeks. The treatment must be begun on tuberculous guinea-pigs soon after the inoculation; not later than two weeks. He also holds that this substance will be of value in the treatment of the early stages of tuberculosis in man.

This preparation has been already used by Koch in a large number of cases, including lupus as well as pulmonary tuberculosis. He states that these cases have been markedly improved by the treatment.

Whether or not Koch has succeeded at last in securing a curative agent for tuberculosis, it must be admitted that his work is of the highest scientific value, and that he has furnished us with the most exhaustive research yet made on the chemistry of the tubercle bacillus.

Numerous attempts have been made to separate the curative from the poisonous constituents of Koch's original tuberculin. However, in view of the fact that the work abstracted above, done by Koch himself, is the most complete and most scientific research in this direction, I will omit mention of the work done by others in this line.

THE TOXINS OF PYOGENIC GERMS.—In 1879 Leber concluded from his observations on infective keratitis that the aspergillus must produce certain soluble products which diffuse through the cornea and set up an inflammatory action in the adjacent vascular tissue. Three years later he showed that suppuration could be induced by the introduction of sterilized mercury and copper, and that the pus formed in these experiments is free from germs. In 1884 he induced suppuration by the injection of cultures of the staphylococcus pyogenes aureus which had been sterilized by being boiled for hours. In 1888 the same investigator reported that he had found an alcoholic extract of the dried staphylococcus to be highly pyogenetic. From this extract he has prepared a crystalline body, which he calls phlogosin. This substance is readily soluble in alcohol and ether, sparingly soluble in water, and crystallizes in needles. The crystals can be sublimed, leaving no residue, and the sublimate, which forms in rosettes, still possesses pyogenetic properties.

Christmas filtered bouillon cultures of pyogenic germs and precipitated in the filtrate with alcohol an albuminous substance that affected animals in a similar manner to phlogosin. Hoffa obtained methyl-guanidine from the bodies of rabbits dead of septicæmia.

Buchner has shown that the cells of various bacteria contain a pyogenic substance. Brieger and Fraenkel supersaturated filtered cultures of the staphylococcus with ammonium sulphate, removed the salt by dialysis, and evaporated the solution of the toxin at 40° C. *in vacuo.* The concentrated solution thus obtained killed animals within twenty-four hours. Nannotti evaporated bouillon cultures of pyogenetic germs to one-fourth their volume at 60° C. and then filtered. The filtrate thus obtained in doses of ten cubic centimetres injected intraperitoneally or intravenously did not kill, but when administered daily in doses of from two to three cubic centimetres subcutaneously, death from marasmus occurred from the fourteenth to the fortieth day. When given intravenously or intraperitoneally only fifteen per cent. of the animals experimented upon died. The greater mortality from the subcutaneous injections is supposed to be due to the less rapid elimination of the poison. Rodet and Courmont found that previous treatment of animals with the chemical products of the staphylococcus rendered them more susceptible to subsequent inoculation with the germ. Furthermore, they observed that the kidney-lesions were much more marked when the inoculations were preceded by injections of sterilized cultures. By treating filtered cultures with alcohol they obtained an insoluble body with which they immunized animals to the germ, while substances soluble in alcohol increased the susceptibility of animals to the living germ. Donath induced fever in animals by intravenous injections of from ten to twenty cubic centimetres of cultures of pyogenic germs sterilized at 63° C. Terni believes that the pyogenic properties of the staphylococcus are due to the acids produced by it. The most important acids found in cultures of the pyogenetic germs are propionic, butyric, lactic, and valerianic.

Wolf has made a series of experiments in his studies of the chemical products of the pus-producing germs, which are more interesting in the suggestions to which they give rise than in the results obtained. He placed collodion sacs filled with cultures of the staphylococcus in the abdominal cavities of rabbits, having previously ascertained that the soluble products of the growth of the germ would diffuse through the collodion, while the germ would be retained. The results were somewhat contradictory. In some instances the sacs were found to be broken, and in these cases, of course, death resulted from the action of the living germ. Even when the sacs were not broken, and when their contents were found, as sometimes happened, to be sterile, staphylococci were found in the peritoneal cavity. The question quite naturally arises as to the source of the germs found in this cavity under these conditions. Wolf suggests that they may have come from the intestines. This suggestion is strengthened by the fact that coli germs were found in the peritoneal cavity. It would seem that the chemical products of the staphylococcus having been diffused from the collodion sacs so altered the vitality of the surrounding tissue that the colon germ passed through the intestines into the peritoneal cavity. This is interesting inasmuch as it has a possible bearing upon certain questions which have arisen concerning the bacteriology of peritonitis.

Buchner has found that the quantity of pyogenetic substance in bacterial cells varies with the kind of germ, and that some species (the bacillus prodigiosus, for instance) seem to contain no such body. The bacillus pyocyaneus contains a large quantity of pyogenetic substance. This may be prepared from these germs in the following manner. The germs are taken from potato cultures and rubbed up with water; then they are treated with about fifty volumes of a five-tenths per cent. solution of caustic potash. This forms a mucilaginous mass, which dissolves at the temperature of the water-bath. After being heated for some hours, the fluid is filtered through a number of small filters. The first portions should be refiltered. The filtrate is a greenish fluid, which by the careful addition of acetic or hydrochloric acid forms a voluminous precipitate. This precipitate may be collected on a filter, washed with water, then suspended in water and a few drops of soda solution added, when a dark brown fluid with a tendency to gelatinize in the cold is obtained, containing about ten per cent. of proteid. Animals treated with this pyogenic substance show an increase in the number of white blood-corpuscles, and the proportion of white to red may become as great as 1 to 38. Buchner tested the action of this body upon himself. One cubic centimetre of a very dilute solution containing 3.5 milligrammes of the solid proteid was injected under the skin of the forearm with antiseptic precautions. Two hours later there was marked pain along the lymphatics, especially localized in the elbow and axilla. The temperature showed no marked elevation. On the following day an erysipelatous redness and swelling extended for some inches about the place of injection. The inflamed area felt hot and projected distinctly above the surrounding surface. The lymphatics of the arm appeared like red cords. On the third day the swelling and redness were more marked, and extended from the wrist to the elbow. On the fourth day the symptoms began to recede. Here we have clinically a perfectly typical erysipelas with lymphangitis, and Buchner asserts that all the cardinal symptoms of inflammation—rubor, calor, dolor—could not be produced without involvement of the solid tissues.

Similar but less marked symptoms may be induced by the injection of albumoses from the most diverse sources. For instance, vegetable casein is more or less markedly pyogenetic when injected subcutaneously in animals or men.

THE TOXINS OF THE SUMMER DIARRHŒAS OF INFANCY.—More than one-fourth of the children born in the civilized world die before they reach five years of age, and nearly one-half of these deaths are caused by the summer diarrhœas. Medical men have long sought for the causative factors in the production of these diseases. A history of the various theories proposed to explain the origin and nature of these diarrhœas would form an interesting chapter, but the scope of this paper prevents the writer from going into this subject. Some of these theories have been most absurd, and the causation of the summer diarrhœas of infancy has been attributed to every conceivable fancy, from the influence of the sun-spots on health to the use of baby-carriages. Finally the profession recognized the following facts as influencing the mortality from these diseases: (1) these diarrhœas are almost exclusively confined to children artificially fed, and (2) their prevalence is with equal exclusiveness confined to the warm months of the year. Since cow's milk is the food principally used in the artificial feeding of infants, it became evident that this food must during the summer months undergo changes which render it harmful. A cause for the apparent harmful effects of cow's milk was sought for in the chemical differences between this secretion and the milk of woman. Chemists have suggested various additions and dilutions of cow's milk in order to render its chemical composition more nearly identical with the milk of woman. Prepared baby foods have been very extensively used, but these have not lessened infantile mortality. We now know that the summer diarrhœas of infancy are cases of milk-poisoning, and that the poisons originate in the milk through the agency of bacterial growth. The child taking its nourishment directly from the breast of the healthy mother obtains its milk practically germ-free, while the one taking cow's milk receives along with this food many kinds of bacteria, some of which are very harmful. These diseases are confined to the summer months because the germs which elaborate poisons in milk require a relatively high temperature for their growth. During the hot months of summer these bacteria are widely distributed and easily find their way into milk. They grow rapidly and produce chemical poisons. Furthermore, decomposing matter harbors and supports these bacteria at a time when the out-door temperature is high enough to allow their growth. Attention has been called so positively to the dangers of infected milk within recent years that there has been marked improvement in the handling and care of this article of diet, but that there is room for still greater improvement in this direction must be granted. The pasteurization and sterilization of milk have undoubtedly saved the lives of many children, but it remains a fact that the most intelligent care and attention to details in these processes often fail to secure a food that is altogether safe.

Flügge has made a most valuable contribution to our knowledge of those milk bacteria which may elaborate poisons inducing the symptoms observed in the summer diarrhœas of infancy. He has found in milk four anaërobic bacteria, two of which may produce poisons. If a milk culture of one of these be sterilized by filtration through porcelain and the filtrate be injected subcutaneously in mice in amounts of from three-tenths to six-tenths of a cubic centimetre, these animals die after from three to fifteen hours. Post-mortem examination in these cases shows a distinct hyperæmia of the intestines, and the presence of transudates in the peritoneal and pleural cavities. When the germ-free filtrate is administered to guinea-pigs in doses of five cubic centimetres intra-abdominally, death occurs within from fifteen to twenty hours. It is quite evident that bacilli capable of producing such poisons cannot be considered as harmless constituents of the food of infants. It is worthy of note in this connection that these germs require a temperature of from 30° to 37° C. for their best growth, and that they do not develop below 22° C. Some of these anaërobic bacteria produce a disagreeable odor and an unpleasant taste in milk, and these would probably prevent an adult from drinking it; but it might be taken even in large quantities by the hungry infant. It should be understood that these anaërobic bacteria are frequently present during the hot months in summer in market milk.

The same investigator has reached still more interesting results in his study of the peptonizing bacteria which may be present in milk. He has isolated and studied twelve species of this kind of germ. Milk infected with these peptonizing bacteria develops the bitter, irritating, characteristic taste of peptones, but this would not be observable during the first few days of the growth, and it is at this time that it would be taken by children. A sample of milk may contain millions of these peptonizing bacteria and still be sufficiently pleasant to the taste to be taken without any hesitation. These bacteria grow abundantly at a temperature as high as 44° C., and their spores resist a temperature of 100° C. maintained for two hours. This shows that these bacteria may escape ordinary sterilization. The developed germs are destroyed at the temperature employed in the sterilization of milk, but their spores remain. The development of spores into bacteria may be prevented by keeping the milk after sterilization at a low temperature, because these spores do not develop into germs except when the temperature is high. This emphasizes the fact that keeping the milk at a low temperature after sterilization is an essential part of the process of preparing cow's milk for feeding children.

Of the twelve peptonizing bacteria isolated and studied by Flügge nine failed to develop any poison. Therefore any harmful effects attributable to these must be due to the peptones formed. In view of the fact that peptonized milk has been largely employed in the feeding of infants, it may be interesting to know that there are grave doubts concerning the desirability of feeding children upon peptones. In testing the nutritive value of peptones on the lower animals, and on both healthy and sick men, severe intestinal irritation has been induced. Züntz fed dogs upon peptones, and found that these animals soon suffered from an abundant watery diarrhœa, and eliminated from three to six times as much unused nitrogen as did control animals when fed upon meat. Peiffer caused in himself and in another man intestinal irritation and diarrhœa by the administration of large doses of peptones. Neumeister states, "By long-continued use of these preparations symptoms of marked irritation and injury to the intestines uniformly resulted, and consequently the prescription of albumoses in disease can scarcely be regarded as rational."

With the three other peptonizing bacteria obtained by Flügge from market milk some interesting results were obtained. Cultures of No. 1, two days old, were injected in quantities of two cubic centimetres into the dorsal lymph-sacs of frogs, and caused at first slowness of motion, loss of reflex, and after one hour paralysis of the extremities, and after four hours death. Subcutaneous injections of half a cubic centimetre killed mice after from five to six hours. In these animals, with the exception of the absence of voluntary movements and tardiness of response to stimulation, no symptoms were manifested. Five cubic centimetres given to guinea-pigs intra-abdominally induced marked dyspnœa, and death occurred after from four to seven hours. In these animals the abdomen was distinctly retracted, and handling evidently caused pain. Section showed congestion of the peritoneum, of the serous coat of the intestines, and of the kidneys. Dogs drank large quantities of milk cultures of this bacillus with apparent relish, but after one hour diarrhœa set in, with a discharge every few minutes. Milk cultures of No. 3 were without effect on frogs and mice, but caused sharp diarrhœa, accompanied by severe abdominal pain, when administered by the mouth to puppies. "One of the puppies showed on the second day progressive exhaustion, paralytic weakness of the extremities, and a fall in the temperature. He died on the third day. Section showed hyperæmia of the kidneys and nothing else worthy of note."

"The bacillus No. 8 injected in milk cultures in frogs, mice, and guinea-pigs produced no marked action. When the culture was filtered through a Chamberland bougie and concentrated *in vacuo* to one-fifth its volume, it killed mice and guinea-pigs when injected in doses of six-tenths and five cubic centimetres respectively. Death, which followed in from six to twelve hours, was preceded by dyspnœa and convulsive movements. Post-mortem examination showed nothing characteristic. Even the unconcentrated milk cultures acted powerfully when fed to puppies. After taking this food for from one to two days, profuse diarrhœa set in, but disappeared on the next day, the feeding being discontinued. The diarrhœa was accompanied by great emaciation, weakness of the extremities, and a tottering gait. As soon as the use of the culture was discontinued and ordinary milk substituted, improvement began, and continued until recovery was complete. Two puppies after recovery were again fed with the culture. After a short time the profuse diarrhœa with its accompanying symptoms reappeared."

Flügge believes that the effects above described are not due to peptones, or, if so, this bacteria must elaborate peptones wholly different from those produced by peptic digestion. The symptoms and the post-mortem appearances are not those of peptone-poisoning. Moreover, of the three toxicogenic germs belonging to this group studied, the one forming the smallest amount of peptone yielded the most virulent cultures. Flügge found these peptonizing bacteria frequently present in market milk in pure cultures.

Lübbert has continued the chemical study of the peptonizing bacteria obtained from milk by Flügge. He states that these germs are widely distributed, being found in hay, in the soil, and in the fæces of cows. They act wholly upon the proteids of milk, the amounts of fats and sugar in milk remaining unaltered in cultures twelve days old. The amount of proteid is greatly reduced with the age of the culture. Caseoses are produced by these germs. Amido acids were not found. Milk cultures of these germs twenty-four hours old fed to guinea-pigs caused death within four days. Three young puppies were fed with this milk. They developed severe diarrhœa two hours after taking this food, and died on the fifth, sixth, and seventh days. Another puppy was fed upon milk freshly infected with these germs, and developed a diarrhœa that continued for three days and then terminated in recovery. A fourth dog, several months old, took the milk cultures freely without any effect, thus showing that age gives immunity to this poison in dogs as it does in children. There can be no doubt that susceptibility to galactotoxins is more marked in infancy than it is in subsequent life. Post-mortem examination of the animals killed by the above-mentioned milk cultures showed only slight swelling and congestion of the mucous membrane of the small intestines. The germs were not found outside of the intestinal canal, and both subcutaneous and intra-abdominal inoculations with small quantities of pure cultures were without effect. However, two cubic centimetres or more of the milk culture twelve hours old or older given intra-abdominally caused death. Milk cultures grown in thin layers with large surfaces exposed to the air were so increased in virulence that one cubic centimetre given intra-abdominally to guinea-pigs caused death. An intense enteritis was found in animals killed by intra-abdominal injections of from five to ten cubic centimetres of the milk culture. The serous covering of the intestine was found to be dark red and dotted with pin-head hemorrhagic spots. In some instances a small amount of bloody fluid was found in the peritoneal cavity. Apparently the chemical poison of these bacteria exists within the bacterial cells, and when the bacteria are removed from cultures by filtration through porcelain the filtrate may be quite inert. Furthermore, when bacteria are killed by the action of heat or by exposure to the vapor of chloroform, their poisonous constituent seems to be rendered inert, or at least greatly reduced in virulence. It requires from twenty-three to twenty-four millions of these bacteria injected intra-abdominally to kill a guinea-pig weighing three hundred grammes.

The writer of this paper has obtained these peptonizing bacteria from the milk taken by a child about two hours before the setting in of a very severe form of summer diarrhœa, and can confirm the above-given statements of Lübbert and Flügge. Milk cultures of this germ were kept in an incubator from one to forty days, and at no time after this was the milk, after complete removal of the germs by filtration, found to be poisonous. Indeed, some of these peptonizing bacteria cultures after the fifteenth or sixteenth day contained only dead bacilli, and when this point is reached the unfiltered milk is not poisonous. The peptonizing bacteria with which I have worked are probably not identical with those studied by Flügge and Lübbert, but they belong to the same class.

I would suggest that the susceptibility of infants to these bacteria is due to the great readiness with which the mucous membrane of the intestines of infants absorbs casein, carrying along with it the bacterial cells containing the chemical poison. It is a well-demonstrated fact that the intestinal mucous membrane in the infant may absorb unchanged casein. Later in life the stomach becomes of more importance as a digestive organ and absorption through the intestines is modified.

Some years ago the writer studied the chemical products of three germs obtained by Booker from the fæces of children suffering with summer diarrhœa. Of these bacteria, Booker stated: "'X' was found almost as a pure culture in the fæces of a fatal case of diarrhœa. 'a' was strongly pyogenic when tested last winter. 'A' was isolated last summer, liquefies gelatin, and belongs to the proteus group."

Beef tea cultures of these germs were kept in the incubator at 37° C. for ten days, and then twice filtered through heavy Swedish filter-paper. The second filtrate was allowed to fall into a large volume of absolute alcohol, feebly acidified with acetic acid. Flocculent precipitates formed in the alcohol, and were collected, dried on porous plates, and their action on animals tested. The crude poison thus obtained from cultures of "X" was slightly yellow as seen deposited in alcohol, but became greenish on exposure to the air. It was freely soluble in water, and gave the biuret and xanthoproteic reactions. It could not be classed among the peptones, because it was precipitated from its aqueous solution by saturation with ammonium sulphate. It cannot be called a globulin, because sodium sulphate added to saturation and carbonic acid fail to precipitate it. The toxin obtained from cultures of "a" was flocculent and perfectly white when first obtained, but blackened on exposure to the air. Precipitate from cultures of "A" differed from the others, inasmuch as it was wholly insoluble in water. The following brief notes will illustrate the nature of the symptoms and the post-mortem appearances observed after the administration of these toxins.

A small amount of the poison from "X" was dissolved in water and injected under the skin on the back of a kitten about eight weeks old. Within one-half hour the animal began to vomit and purge, and death resulted within eighteen hours. The mucous membrane of the small intestine was pale, the intestine was contracted in places and contained a frothy mucus. The stomach was distended with gas and contained mucus stained with bile, the liver seemed to be normal, while the spleen and kidneys were congested and the heart was distended.

A second kitten treated with the poison from bacillus "a" vomited and discharged from the bowels green matter. This animal died after fifteen hours, and presented appearances practically identical with those mentioned above.

The third kitten was treated with cultures of the bacillus of "A" suspended in water, and this animal presented substantially the same symptoms and post-mortem conditions as those obtained from the other toxins.

Ten milligrammes of the dried poison from bacillus "a" injected under the skin of a guinea-pig caused death within twelve hours. Fifteen milligrammes of the same substance were employed hypodermically in each of two kittens. One died after forty-eight hours, and the other recovered after two days of vomiting and purging. Two dogs of about five pounds' weight had each forty milligrammes subcutaneously, and after serious illness of two days' duration recovered. During these two days of vomiting and purging the dogs were constantly shivering as with cold, but the rectal temperature stood at from 102.5° to 103.5° F.

The writer once obtained tyrotoxicon from milk which had been taken by a child a few hours before the appearance of the first symptoms of what proved to be a severe form of cholera infantum. Baginsky and Stadthagen have obtained from cultures of a germ found in the fæces of a child suffering from summer diarrhœa a poisonous proteid which causes slight dyspnœa when administered to mice. The coat becomes rough, the animal sits with drooping head, and, when forced, moves sluggishly but without any evidence of paralysis. This marked apathy increases, and death results after from two to three days. Section shows an infiltration about the place of injection, and congestion of the spleen, liver, and peritoneum. The intestine is hyperæmic its entire length, and its upper portion contains a reddish-brown fluid. From cultures of the same bacterium they have also obtained a poisonous ptomaine, which is probably identical with one found by Brieger in putrid horse-flesh, and which has the formula $C_7H_{17}NO_2$.

I have several times found in milk a poison having a very markedly muscarin-like action. It causes diarrhœa, with marked prostration, and especially depresses the action of the heart. Both muscarin and neurin have been found among the bacterial products, but neither has been obtained from milk. Notwithstanding my failure to isolate either neurin or muscarin from poisonous milk, I believe that one or the other, certainly some substance closely allied with these bodies physiologically, is often present in poisonous milk. Theoretically, it is easy to see how neurin may be formed in milk. Cholin, which results from the disintegration of lecithin, is a constant constituent of milk, and by the removal of water from cholin it is converted into neurin. Whether this unknown poison in milk be chemically identical with or closely allied to neurin, its physiological resemblance cannot be doubted.

There are other toxins which have been found in milk and milk products that I must pass over in the present paper. Sufficient has been given to show that, under the influence of toxicogenic bacteria, most deadly chemical substances may be formed in that food which constitutes practically the sole nourishment during the first year of life.

THE TOXINS OF TYPHOID FEVER.—As early as 1885 Brieger obtained from pure cultures of the Eberth bacillus a poisonous ptomaine. This substance causes in guinea-pigs a slight flow of saliva, increased frequency of respiration, dilatation of the pupils, profuse diarrhœa, paralysis, and death within from twenty-four to forty-eight hours. Post-mortem examination shows the heart to be in systole, the lungs hyperæmic, and the intestines contracted and pale. For a while this ptomaine was regarded as the specific poison of typhoid fever, and its discoverer named it typhotoxin. However, there has as yet been no proof that this substance is formed in the human body in typhoid fever, and it certainly is not present in all artificial cultures of the Eberth bacillus.

According to Peiffer, the typhoid fever toxin is contained within the bacterial cells. From three to four milligrammes of this substance for each one hundred grammes of body-weight suffices to kill guinea-pigs. With this poison animals can be rendered immune to the Eberth bacillus, but not to the bacillus coli communis.

In 1889 the writer obtained from mixed cultures of the germs found in typhoid stools a base forming crystalline salt, and capable of inducing in cats and dogs a marked elevation of temperature accompanied by severe purging. The following is a record of one experiment with this substance: "An aqueous solution of the crystals was given to a dog by the mouth at 3 P.M. The rectal temperature before the administration was 101° F. At 3.15 retching and vomiting set in, and continued at intervals for more than two hours. At 3.30 P.M. the temperature was 103° F. At 3.55 the animal began to purge. The first discharge contained much fæcal matter, but subsequently they were watery, and contained mucus plainly stained with blood. At 4 the temperature was 103.5° F., and remained the same until 4.30. The animal was not seen again until 10 A.M. the next day, when its temperature was 100.5° F., and recovery seemed complete."

SCIENTIFIC STUDY OF THE MENTAL AND PHYSICAL CONDITIONS OF CHILDHOOD.

BASED UPON THE EXAMINATION OF ONE HUNDRED THOUSAND CHILDREN.

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ALL who are engaged in practice among children must be well aware that young patients present distinctive points of difference from adults. The marked constitutional variations among children, the heavy infant mortality, the differences in weight and bodily development, as well as the physical, neural, and mental difficulties which often arise during school life, all call for careful scientific investigation. In looking over the medical histories of children and noting the complaints made by parents, it soon becomes apparent that a large number of children present mental as well as physical defects which should be brought within the domain of scientific medicine and placed under the care of the physician. For such reasons it has seemed worth while to note carefully the points seen in children which indicate their bodily development and mental status. The former have often been described by writers on anthropometry, physiognomy, and imbeciles. Brain conditions claim further study and description, a section of clinical investigation which I hope here to advance. I shall best accomplish this purpose by giving an account of the investigation upon which this essay is based.

After several years' work in recording and studying observations[1] upon normal and abnormal children, an account of which has been published, I was able to present a number of physical signs expressing various conditions of bodily development and brain-action, which will be explained.

In 1888 a committee was appointed to conduct an investigation as to the average development and condition of brain-power among the children in primary schools. During the period 1888–92 I had the opportunity of examining, in conjunction with other medical men, one hundred thousand children. A full report has been published.[1] In this inquiry it was determined to look at each child in the school and take notes of all in whom any one or more abnormal points could be observed, as well as any reported by the teachers as dull or backward pupils. A list of points indicating conditions below the normal was drawn up, and others were added according to the experience gained.

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[1] Physical Expression: its Modes and Principles. International Scientific Series, Kegan Paul & Co. The Anatomy of Movement, Growth, and Motion. The Macmillan Company, New York.

The signs observed are of two kinds: A, developmental defects; B, nerve-signs.

A. *Developmental defects* are points in defect of size, form, or proportioning of parts of the body. Thus, looking at the child, we note the cranium and the separate features,—the ears, the nose, the palpebral fissures, and the mouth,—also the growth of the body.

B. *Nerve-signs* are seen in the balance of the head, the spine, the upper extremity, and the digits, as well as in the facial action and eye-movements. These movements and balances of action or postures are observed as signs of the action and condition of the nerve-centres.

The physical health and nutrition of the child were observed. Those who were pale, thin, or delicate were recorded as cases of low nutrition.

The teacher's report as to mental ability was asked in each case presenting signs of defect. The teachers were also asked to present any dull or backward pupils not picked out previously. All these children were recorded as dull.

We thus have four main classes into which the defective conditions of childhood are grouped, each of which is indicated hereafter by the symbol attached:

A. Defects in development of the body.
B. Abnormal nerve-signs.
C. Low nutrition of the body.
D. Mental dulness.

Throughout this inquiry I gave a written description on printed schedules of each of the eighteen thousand one hundred and twenty-seven children noted as presenting one or more points of defect, as seen among the one hundred thousand children in the schools. It has, however, been found more expeditious and convenient to record cases on the card given on the next page.

Such a card is filled in for every case noted; the principal defects are printed; the numbers refer to the published report, in which each defect is described. The less frequent defects are not printed, but may be recorded by adding the number of the particular sign to the card. Thus, if the head be hydrocephalic, the reference number 9 is placed on the card under the description of "cranium," that being the number of the defect in the published report. If defect of speech is to be recorded, the reference number 60 is written below "other nerve-signs." The defects present are indicated by drawing the pen through the name of the defect. A formulated epitome at the right-hand lower corner of the card indicates the main divisions of defect in the case; those present are marked by the pen.

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[1] Report on the Physical and Mental Conditions of Childhood. Published by the Committee on Childhood, 72 Margaret Street, London, W., 1895. The Macmillan Company, New York.

School............................ *Card No*........................
Infant............................ *Reg. No*........................ **BOYS.**
Age .... *Spl. Rep*t ....

A DEVELOPMENT DEFECTS
a 1 CRANIUM
2 Large
3 Small
4 Bossed
5 Forehead
6 Frontal ridge

b 11 EXTERNAL EAR
c 12 EPICANTHIS
d 13 PALATE
14 Narrow
15 V-shaped
16 Arched
17 Cleft
18 Other types
e 19 NASAL BONES
f 20 GROWTH SMALL
g 21 OTHER DEVELMT. DFTS.

B NERVE-SIGNS
43 General balance
44 Expression
45 Frontals overact
46 Corrugation

47 O. oculi lax
48 Eye-movements
49 Head-balance
50 Hand weak
51 Hand nervous
52 Finger-twitches
53 Lordosis
h 54 OTHER NERVE-SIGNS

C NUTRITION
D DULL
E EYE-CASES
64 Squint
65 Glasses plus
66 Glasses minus
67 Myopia, no glasses
68 Cornea disease
69 Eye, lost accident
70 Eye, lost disease

F RICKETS
G EXCEPTIONAL CHILDREN
i 82 CRIPPLES

A B C D E F G

In Class A, *developmental defects*, forty-two signs are described. The most important are those of the cranium. 1. "*Cranium defective*" includes any defect in size, form, proportions, or ossification of the cranium. A case may come under more than one of the classes below. As to a standard of normal size, in a well-developed child of good potentiality the head circumference at the ninth month is seventeen and a half inches; at twelve months, nineteen inches; at seven years, twenty-one inches (this is a rather high standard of size). Head over twenty-two inches circumference in a school child may be considered large; allowance must be made for age. Doubtless many such cases are rachitic. Speaking of a small head, the size of head is referred to as apart from the size of the child for his age. This may be determined by inspection, by the open hand placed on the head observed, and by the measuring-tape. Usually the small heads are from eighteen to nineteen and a half inches in circumference. The cranium may be bossed, with protuberances or outgrowths at the site of the ossific centres of the frontal bones or parietals, at the site of the fontanelle, and elsewhere. The forehead may be narrow, shallow in vertical measurement, or small in all dimensions; it may bulge forward and overhang. The suture between the two halves of the frontal bone may present a vertical ridge, such as is typical of the scaphocephalic cranium and common among microcephalics. The cranium may be asymmetrical (7), one side, usually the left, being smaller than the other; it may be dolichocephalic (8), long in the antero-posterior diameter, or hydrocephalic (9). Other types of cranium (10) are square, conical, or with the anterior half much larger than the posterior.

11. The *external ear* may be defective in its parts, size, or form. Abnormality in size, proportioning, absence of the helix or antihelix, and the texture of the skin are noteworthy; this defect is far more common in boys than in girls.

12. The *epicanthis* is a fold of skin continuous with the lower fold of the upper eyelid placed across the inner angle of the opening of the eyelids, covering the caruncle.

13. The *palate* is second in importance only to the cranium as an indication of defect. It may be narrow without being otherwise altered, the alveolar processes being too close together. The V-shaped palate is pointed more or less sharply in the horizontal plane at the front, the alveolar processes being nearly straight lines, meeting at an acute angle. The vaulted palate is arched in the vertical plane. Other types are the flat and the horseshoe variety.

19. The *nasal bones* may be wide, sunken, or indented. It is well in such cases to examine the naso-pharyngeal passages for obstruction.

21. Twenty-one other defects in development are described, including defect of the features. The face may be asymmetrical, or small in proportion to the calvarium; the features may be coarse, heavy, flat, or the lips thick. The separate features may not be individually malformed, but may be disproportionate to one another or to the size of the face; thus the nose may be small and the face round or flat. The palpebral fissures may be defective in size or form, too small, or the transverse axis sloping outward and downward. Supernumerary ears, remnants of branchial clefts, and other points of defect are sometimes seen.

In Class B, *abnormal nerve-signs*, I have described points for clinical observation, most of which were introduced during the course of this inquiry; they mostly indicate conditions of muscular balance and movement expressing modes of action in the nerve-centres.

43. *General Balance Defective.*—Asymmetrical positions of the body in the spine and shoulders, in holding the arms and planting the feet, slouching, and listlessness.

44. *Expression Defective.*—Want of changefulness, vacancy, fixed expression of face. The visible muscular action and balance in a face may be described, and still there may be an expression that cannot be described anatomically. There may be frowning and corrugation or twitches, and yet the expression may be good. On the other hand, we may have a bad expression, and yet be unable to say what muscles are in action producing it.

45. *Frontals Overacting.*—The frontal muscles almost always act symmetrically, at the same time and in similar degree. Their action produces horizontal creases in the forehead, which may be deep if they act strongly and frequently. Sometimes these muscles may be seen working under the skin in vermicular fashion, with an athetoid movement; in other cases the action is fine, producing minute creases and a dull appearance of the forehead; expression is not necessarily erased. This sign may be seen in infants, but more commonly is found in older children; it may be temporary, and having lasted some years may disappear. It is most common with uneducated children, and is very frequent in imbeciles. Subsidence of overaction of the frontals often indicates attention on the part of the child. It is most common in boys.

46. *Corrugation.*—Knitting the eyebrows, drawing the eyebrows together; vertical creases are thus produced in the middle of the forehead. This sign may coexist with overaction of the frontal muscles; it seems to be more closely associated than any other single sign with some forms of mental stress. Corrugation may be associated with frontals overacting in a similar athetoid movement, producing square creases, vertical and horizontal; when the action is slight it makes the forehead look dull.

47. *Orbicularis Oculi Relaxed.*—The orbicularis muscle gives an appearance of sharp definition to the form of the lower lid, so that its convexity is seen. When the muscle is relaxed there is fulness or bagginess under the eyes, which disappears on laughter. The relaxed condition is expressive of fatigue and exhaustion; it is seen in the nerve-depression accompanying constant headaches, and is symmetrical. It may result from close rooms or from want of sleep.

48. *Eye-Movements Defective.*—When an object is moved at a distance of two feet in front of the face, the child's eyes normally move in following it; in some cases the head always turns towards the object, while the eyes are kept still in their orbits. In other cases there are restless, uncontrolled movements of the eyes. Want of coördinated movements of the eyes by their small muscles leads to inexactness and slowness in seeing and observing, and may lead to inaccurate reading, while incoördinated movements may lead to mental confusion.

When children are told to hold out their hands in front of them, and the action is shown them momentarily, in the normal the arms are held out horizontally, each on a level with the shoulder, the elbow extended and the forearm pronated, while the hand is extended straight with the arm, the metacarpal bones, the digits, and the thumb being all in the same plane: such is the normal. Two abnormal modes of hand-balance may be given, but intermediate types are common also.

50. *Hand-Balance Weak.*—In this type of balance the hand when held out is slightly drooped or flexed at the wrist, the palm or metacarpus slightly contracted laterally, as in a cone, while the digits are moderately flexed. The same weak attitude may be seen in a hemiplegic hand or in a child in deep sleep, when the muscles are not innervated (paretic), if the limb is held free by raising it by the shirt wristband. The type may be varied: with less degree of weakness the hand is as in the normal, but with the metacarpal bone of the thumb drooped; in exhaustion and great feebleness the metacarpus is more contracted or adducted, and the degree of finger-flexion is greater.

51. *Hand-Balance Nervous.*—When the arms are held out at command, the wrist droops, the palm is slightly contracted laterally, the thumb and fingers are extended backward beyond the straight line at their junction with the metacarpus. The elements in this posture may vary in degree; the most essential element is extension backward at the knuckle-joints, and this may be different with the various fingers or be seen with some only. The posture is common in children with slight chorea and those subject to headaches, night-terrors, and tooth-grinding,—*i.e.*, nervous children.

52. *Finger-Twitches.*—In the hand held free for inspection there may be twitching movements of the digits in flexion, extension, or lateral; the latter are the more indicative of the finer grades of cerebral disturbance and are produced by the smaller muscles. It is noteworthy that while finger-twitching is more common in boys, chorea is more frequent in girls.

53. *Lordosis.*—When the hands are held forward, an alteration in the balance of the spine may appear, with an arching forward in the lower part of the back, while the upper part of the spine between the shoulders is thrown back. This arching forward of the lumbar spine is due to weakness of action in the spinal muscles. It is often seen in chorea, accompanied by some degree of temporary lateral curvature.

54. Other nerve-signs less frequently observed, but not less important, are as follows. Grinning, or over-smiling, either spontaneous or on any stimulation to effort. The naso-labial lines thus formed may be fine or coarse, and they may be duplicate. Response in action following a command or in imitation may be inexact or uncertain or slow: some children in school stop to look at the others before responding in their movements; response seems more easily controlled through the eye than through the ear. There may be a number of extra movements preceding the response, such as inclining the head, protruding the tongue, shrugging the shoulders, or repetition of the command.

Speech Defective.—Thick utterance and indistinct speech may depend upon conditions of naso-pharyngeal obstruction. I believe, however, that all forms of palate, except the cleft, are consistent with fair speech if there be no such obstruction. Stammering is a condition of muscular spasm much more common in boys than in girls. It is important to note carefully the area affected by spasm, which may commence by corrugation in the forehead or at one of the angles of the mouth or in an altered facial expression before any sound is produced. This is important in training, as when the spasm is commencing speech should be stopped. Speech may be almost absent, in which case the auditory apparatus should be fully examined. There may be mental defects of speech: repeating the question asked (auditory imitation) without reply is common with imbeciles; the vocabulary may be extremely limited as the result of neglected training.

The main classes of defect seen in childhood having been described, we may pass on to the examination of an individual child.[1] Let the child stand up, and observe him. It is convenient to prevent the child from looking straight at your eyes by telling him to look at a small object held in your hand. The size, form, and proportioning of the head and the separate features may thus be seen; the facial expression and muscular action in the forehead, about the eyes, and in the lower section of the face should be separately noted, as well as movement of the eyes in following the object moved. The symmetry of balance in feet, shoulders, and spine should be recorded. Tell the child to hold out his hands in front, with the palms downward, and momentarily show him what you mean; any deviations from the normal balance as described can now be seen; the movement may be repeated to see the average of his action.

When any neural or mental defect is found, further clinical examination is called for; in particular, as to exact response in action equal on either side. Let the child stand up, and look at him; tell him to look at your hands and do as you do while you make exact movements with your fingers for him to imitate. Note whether each hand imitates equally well in the time and quantity of movement of the fingers; the same fingers may move as in your hand, but more slowly or less in degree. Imitation may thus be better with his right or his left hand when exercised separately, and slight degrees of defect (hemiplegia) on one side of the brain may thus be detected. A boy eight years of age, without other bad words, constantly and without apparent meaning often used the words "beast," "beastly," "dirty ape." He was a very backward boy, but could write with his right hand; when made to imitate movements, these were found to be markedly less exact and slower with the right hand than with the left. It seemed probable that he had some defect of the left hemisphere, with slight aphasia. He was educable and benefited by training. In making the clinical examination some degree of athetosis may be found. The movements of athetosis are characterized by a slow repetitive action, the same parts moving in the same order of succession on all occasions. Often they are combinations of movement such as are not seen in the normal, occurring either on any stimulation or without apparent cause, and effecting no useful result; they are most often seen in hand, fingers, and toes on the same side; occasionally athetosis (like hemiplegia) may be double, affecting both sides of the body. Accompanying athetosis or independent of it there may be muscular rigidity detected in making passive movements of the limbs; this may also be one-sided or may affect both arms and legs. Tremor, chronic chorea, and other defects may be met with in this class of cases.

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[1] See author's "The Children: how to study them." F. Hodgson & Co., 89 Farringdon street, London, E.C.

The value of the physical signs described lies in their significance of physical and neural states corresponding, whose interactions we desire to study. It is in the corelation of the main classes of defect that new information is mostly to be looked for, supplying also evidence as to the real bearing of the individual defects and as to their causation.

Table I. shows that of Class A—*i.e.*, cases with developmental defect, taken as a group—a large proportion present abnormal nerve-signs, indicating weak or disordered brains; while many are delicate, especially among the girls, and more are dull or backward. Again, of Class B—*i.e.*, cases with nerve-signs—a smaller proportion than above are delicate, but a larger proportion are dull pupils.

TABLE I.

Among Fifty Thousand School Children seen 1892–94, showing the Number, with each Main Class of Defect, and the Percentage of each Class respectively that presented Conditions of Defect corelated thereto. Percentages are taken upon the Numbers given.

NUMBER PRESENTING MAIN CLASS OF DEFECT.		WITH DEFECT IN DEVELOPMENT.		WITH NERVE-SIGNS.		WITH LOW NUTRITION.		WITH MENTAL DULNESS.	
Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.
Defect in development, A.									
3616	2235	. . . . . .		54.6	49.0	20.2	32.0	38.3	41.5
Nerve-signs, B.									
3413	2074	57.8	52.8	. . . . . .		18.6	28.8	40.1	42.4
Low nutrition, C.									
1030	973	71.1	74.6	61.0	61.4	. . . . . .		39.0	40.5
Mental dulness, D.									
2216	1463	63.0	63.4	61.8	60.1	18.1	27.0	. . . . . .	

These corelation values, or the frequency of association of the classes of defect, vary much among the individual developmental and nerve-signs, as shown in Tables II., III. I have shown elsewhere[1] that they vary considerably according to the environment and the ages of the children, and it is seen that they have a different value for boys and girls respectively.

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[1] Statistical Society's Journal, London, March, 1896. Methods of Recording Observations among Children, with Special Reference to the Determination of the Causes of Mental Dulness and other Defects.

TABLE II.[1]

Corelation of Defects in Development, with Nerve-Signs, Low Nutrition, and Mental Dulness, among Fifty Thousand Children.

NUMBER OF CASES WITH DEFECTS IN DEVELOPMENT.		WITH NERVE-SIGNS.		WITH LOW NUTRITION.		WITH MENTAL DULNESS.	
Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.
All cranial defects.							
1528	1048	55.6	50.6	25.7	45.8	41.4	45.5
Cranium small.							
327	738	54.1	50.4	46.1	54.1	50.4	47.8
Ears defective.							
1047	268	54.0	47.7	18.7	26.8	32.4	38.4
Epicanthis.							
514	384	44.1	41.6	12.6	19.0	37.3	35.4
Palate defective.							
796	525	55.4	49.9	21.7	29.5	40.7	44.2
Nasal bones ill shapen.							
241	214	54.3	44.3	6.6	8.8	36.1	36.0

TABLE III.[1]

Corelations of Abnormal Nerve-Signs, with Defect in Development, Low Nutrition, and Mental Dulness.

NERVE-SIGNS.	WITH DEVELOPMENTAL DEFECT.		WITH LOW NUTRITION.		WITH MENTAL DULNESS.	
	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.
General balance defective.	68.6	49.7	23.3	32.3	49.2	41.5
Expression defective.	71.0	69.4	27.5	30.8	53.1	53.3
Frontals overacting.	44.1	49.6	16.7	21.7	41.4	46.2
Corrugation.	52.7	55.0	22.6	15.0	45.7	52.5
Orbicularis oculi lax.	69.1	65.3	21.4	32.6	39.8	46.0
Eye-movements defective.	62.6	61.4	16.2	30.9	41.1	45.7
Hand-balance weak.	52.4	38.8	16.0	21.2	40.0	35.3
Hand-balance nervous.	46.0	39.7	20.1	30.6	34.3	32.9
Response in action defective.	61.6	67.8	25.8	30.3	66.9	69.6
Speech-defect.	25.9	62.8	8.6	18.5	23.2	61.4

As I am here dealing with general conditions of childhood and not with conditions of disease defined by a known pathology, we shall best proceed in the study of the mental and physical conditions of children by considering them in groups arranged according to the main classes of defect they present. Of the children seen in the schools, twenty per cent. of the boys and sixteen per cent. of the girls presented one or more classes of the defects described; the larger proportion of the boys that had to be noted is worthy of remark. The children were not tested as to sight, and cases of ophthalmia were purposely passed over; still, two and nine-tenths per cent. of the children seen presented eye-defects as indicated on the card. For a further account the reader is referred to the report.

A child may present a slight developmental defect or a nerve-sign or two without being in any way a defective child or exceptional in mental or educational position. When, however, a child is said to be dull and is seen to be delicate, with defect in development and nerve-signs, he deserves special attention and consideration in detail. "Group 27, A, B, C, D, contains cases with all the four main classes of defect;" the group is included among "children requiring special care, Group 12."[1] "G, exceptional children," includes all whose physical or mental conditions show them to be obviously at a permanent disadvantage therefrom in school and in social life. This group includes idiots and imbeciles, children feebly gifted mentally, children mentally exceptional, epileptics, dumb, all children crippled, deformed, maimed, or paralyzed. All these exceptional children need to be considered individually as to their special requirements. Two classes of children included above need to be specially dwelt upon. "G (78), children feebly gifted mentally." The class includes children distinctly deficient in mental power who cannot be certified as imbecile. No child is included in the group unless it is believed, upon facts observed and the teacher's report combined, to be incapable of school work in the ordinary classes. It is difficult to define what physical conditions seen, as apart from mental tests, indicate the child as unfitted in mental capacity for the usual methods of education, and an arbitrary attempt to do so has not been made. There appears, however, to be a large class of "children feebly gifted mentally," with defect of mental power short of imbecility, but still with some deficiency.

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[1] For further corelations and facts, see report, op cit.

This class of children is now fully recognized. It was in 1889 that the Royal Commission on the Blind and Dumb, etc., after receiving evidence from the early portion of this inquiry, reported "that, with regard to 'feeble-minded' children, they should be separated from ordinary scholars in public elementary schools, in order that they may receive special instruction, and that the attention of school authorities be particularly directed towards this object." The London School Board has now over one thousand of these children in thirty-day classes of special instruction. The recent Committee on Poor Law Schools also fully recognized that special provision is necessary for the children feeble-minded but not imbecile. Several school boards have made provision similar to that provided for London and appointed medical officers. These children are feeble in body and in mental power and need much care as well as training. "9, G, children mentally exceptional," form a small but socially a very important group. These children, while not necessarily mentally dull and without brain-power, appear deficient in certain mental characteristics and in moral sense,—*e.g.*, the habitual liars, thieves, and incendiaries. Others are liable to attacks of total mental confusion, or periods of mental inaptitude or violent passion, or are moral imbeciles. Some are the offspring of insane parents or criminals, and it is possible that some suffer from petit mal; this class is most difficult to deal with. In training any of these groups of children a description of the nerve-signs in each case is of use to the teacher, who in daily training may then endeavor to remove each sign in detail, whether it be facial action and expression, ill balance of body and hands, or defective eye-movements and response in action.

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[1] For cases, see author's "Study of Children and their School Training." The Macmillan Company, New York.

In studying the groups of children and comparing their corelations, we may learn something of the pathology and prognosis of mental and physical defects. Space will not allow me to give here all the evidence available, which has been published in references given, but the general results of research may be explained.

The main classes of defect among children include a larger proportion of boys than of girls. This rule seems to be almost universally true and to be wide-spread in its application. There are more males than females in returns of infant mortality from developmental defects, more idiots, more among most groups of the infirm classes in the census, and more criminals, paupers, and vagrants.

The main classes of defect are frequently associated or corelated, as is shown in Table I. Of Group A, defect in development, twenty per cent. of the boys and thirty-two per cent. of the girls were found to be delicate children; or, looking at the delicate children, seventy-one and one-tenth per cent. of the boys and seventy-four and six-tenths per cent. of the girls presented developmental defects. Further, when the schools visited were arranged under social class, the ill-developed children were still found to be thin and pale, though probably well fed; so also in institutions where the pupils were well boarded.

It is unnecessary to point out these associations or corelations in detail, as they are indicated in the tables; they will be referred to again, as to a great extent they form a foundation for studying conditions of childhood.

The degree of corelation varies in the age-groups and with sex; this is shown in Table IV.

TABLE IV.

Fifty Thousand Children showing Corelations of the Classes of Defect in the Age-Groups, arranged according to Sex.

	AGE LAST BIRTHDAY.	WITH DEFECT IN DEVELOPMENT.		WITH NERVE-SIGNS.		WITH LOW NUTRITION.		WITH MENTAL DULNESS.	
		Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.
Defects in development, A.	7	. . . . .	. . . . .	31.7	28.5	22.7	35.0	36.6	40.8
" "	8–10	. . . . .	. . . . .	19.7	16.0	5.3	8.4	16.6	21.2
" "	11 and over.	. . . . .	. . . . .	21.6	15.3	2.8	4.1	18.3	22.6
Nerve-signs, B.	7	35.1	41.2	. . . . .	. . . . .	19.6	27.4	43.3	47.0
"	8–10	13.9	10.8	. . . . .	. . . . .	3.5	4.9	25.0	23.7
"	11 and over.	15.1	7.4	. . . . .	. . . . .	3.5	4.9	25.8	26.5
Low nutrition, C.	7	52.5	66.1	41.1	36.0	. . . . .	. . . . .	43.6	42.0
" "	8–10	16.9	19.2	16.1	16.5	. . . . .	. . . . .	6.9	6.1
" "	11 and over.	14.5	9.8	26.5	24.2	. . . . .	. . . . .	8.5	10.6
Mental dulness, D.	7	45.9	55.1	49.0	44.1	23.6	30.1	. . . . .	. . . . .
" "	8–10	17.4	19.4	37.8	32.1	2.3	2.4	. . . . .	. . . . .
" "	11 and over.	19.1	15.5	38.6	37.3	1.7	3.0	. . . . .	. . . . .

From this table it appears that in children of seven years and under, among cases with defective development, the association with all other classes of defect is at the highest.

Among cases of low nutrition the coincidence with (caused by) defective development is very high; so also with (consequence) nerve-signs and mental dulness.

Speaking generally, the acquired classes of defect appear to be less frequent as school age advances.

It has been shown that more boys than girls present points of defect in development of body, in nerve-signs, and in mental dulness. As to general delicacy and low nutrition, there is a larger proportion of girls; this fact is in accordance with the experience of medical practice. The methods of analysis rendered possible by this extended research make it possible to demonstrate some important and new aspects of that fact, and show something of the causes of delicacy among girls. Table IV. shows that when girls have a defect in development they are far more liable than the boys to acquire low nutrition and to become dull mentally; this is true of all the age-groups. If, however, we consider only the children without any defect of development, the proportion of boys and girls who are pale, thin, and delicate is equal. The normally made girl does not appear more delicate than the boy. It is the cases of developmental defect that present the largest proportion of pale, thin children; this is specially the case with girls, in whom, therefore, the manifestations of headache, insomnia, hysteria, and exhaustion should receive the greatest attention, while normal girls may lead a fully occupied and active life.

Having explained the principles of observation, description, and classification of children in groups, it becomes possible to deal with some larger questions of social and educational importance as to the care and training of children, and the duties of the medical attendant or medical officer in charge of a school or resident institution, as well as to consider what may be expected from the work of teachers in the care of the children. To effect the best results in the pupils as to mental status, our profession and the teachers should work hand in hand: for this purpose a mutual understanding may be found in observation and description of the child as we see him.

The inquiry made shows that the conditions of children vary in different localities, among different nationalities, and under different circumstances. The corelations of the classes of defect (their interactions) also vary in degree according to the environments of the children.[1]

It is, then, desirable that a small scientific commission of inquiry should be appointed in each county or state for the purpose of determining the conditions of portions of the school population as to their mental and physical power, ascertaining the number of such as are of imperfect development, their distribution, and the possible causes of such defects, as well as the best means of dealing with children who are dull or deficient in mental power.

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[1] See Report, op. cit.

The educational care of the "dull and backward children" and those "mentally feeble, but not imbecile," is as much a duty devolving on the state as is secondary education for the benefit of bright and healthy children.

In view of the harm resulting, not only to the individual, but also to the state, from educational neglect of the child mentally weak (the deficient child growing up dependent and possibly delinquent), it is desirable that provision be made for all such cases, either in day-school classes of special instruction or otherwise. The selection of children requiring such special care should be made upon the report of a medical officer upon a methodical plan, pointing out facts observed by him in the child as to (1) physical health and development, (2) nervous defects observed, (3) mental defects, as well as on the separate report of the teacher. Such children should be individually reported on each year.

A trained teaching staff is needed for the care of children of low mental power, and some knowledge of the scientific observation and description of children should be considered an essential qualification. Special practical and theoretical instruction should be given to teachers in training colleges and elsewhere as to the observation of the physical indication of weakness and mental feebleness in children, and as to the points to which they should direct attention in school classification and teaching, thus enabling them to describe and, if necessary, to report to the school authority on individual children.

Such knowledge in common between our profession and the teaching profession—and the two professions must remain distinct in their functions, as much as the medical staff and the nursing staff in the hospital—will enable mutual help to be given to the case of the dull or weak child. Besides advising that the pupil should not be kept at school work till he is tired, let us point out—giving a card describing the case—the child with defective development and nerve-signs as liable to nerve-disorderliness, and explain what points may be seen as early indications of fatigue,—*e.g.*, lessened expression, fulness under the eyes, ill balance, or less prompt response, with some extra movement (fidgetiness), often accompanied by mental confusion.

SWIMMING, DANCING, BICYCLING.

By JAMES K. YOUNG, M.D., AND JOSEPH M. SPELLISSY, M.D.

SINCE writing the article on "Physical Development" no changes in its domain, of sufficient importance to necessitate revision, have occurred, but it has been recognized that swimming and dancing are worthy of further comment, and that bicycling demands and deserves a special article.

The instructive photographic analyses of animal locomotion by Mr. Eadweard Muybridge, referred to in the original article, have since been utilized by Mr. Thomas A. Edison, his kinetoscope and vitascope being the syntheses of Mr. Muybridge's studies.

The Swedish system referred to in the original article has steadily gained in favor, and is now in many parts of this country the recognized system.

GENERAL CONSIDERATIONS.

Swimming, dancing, and bicycling have special virtue in promoting physical culture. They not only encourage general and symmetrical development, but are also fascinating amusements, sure of performance. For the sake of condensation, that which can be said in common of all three will be said now.

The best place for the enjoyment of these exercises is in the open air, and the most suitable time is during sunlight. They should not be indulged in by the fatigued, the very hungry, or the recently fed, say within an hour and a half after a full meal. The clothing is best of wool, especially the underclothing. Girls should be uncorseted, or, if corseted, there should be no constriction, the stays being worn so loosely as to be easily moved up and down. An admirable substitute for the corset that supports the breasts and leaves the muscles of the back and abdomen unconfined is to be found in the "bust-girdles" offered by several of the hygienic underclothing establishments. These contrivances have shoulder-straps, and upon them, and not upon the hips and abdomen, should come the weight of the lower garments. The limitation in breathing which corseted women suffer, and the respiratory freedom they may all enjoy, and yet have bust support, are graphically shown by the diagrams, photographs, and tables on the accompanying plates. If a skirt is worn, it should be single and not too long. Those not accustomed to exercise must graduate the vigor and the duration of their efforts. Attempts of the uninitiated or ill-conditioned to equal the performance of those in training are frequently punished by resulting physical injury. Girls, as a rule, should intermit these exercises during menstruation.

Many points regarding the developmental character and physiological effects of these recreations in the sound, and also respecting their therapeutic indications and contra-indications in the unsound, though equally applicable to swimming and dancing, will be found only in the chapter on Cycling. Limitation of space and demand for explicit information concerning a comparatively new sport have necessitated this arrangement.

SWIMMING.

Since the original article this art has been commendably fostered by municipalities establishing public swimming-baths for both sexes. Published statistics[1] show that during the civil war there were drowned one hundred and six officers and four thousand eight hundred and thirty-eight men. Great loss of life could be prevented in civil as well as in military life if swimming were a part of the national education.

It has been urged that the *initial*[2] efforts of the beginner should aim at balance in the water (*i.e.*, the ability to support one's self with confidence) rather than at a perfect method of stroke. Swimming, bicycling, and skating demand balance; this requires the cultivation of muscular sense and employs in its maintenance all the muscles of the body. The faculty of balance in any of these exercises is acquired in varying degrees of time by different individuals. But in any one individual its possession is achieved suddenly in the end, almost as if by inspiration. When it is possessed, then attention can be given to form.

In swimming, equilibrium may be preserved or destroyed by slight effort. The beginner's movements are exaggerated: hence he flounders. When he can keep himself up, usually paddling like a dog, then he is in position to learn a stroke. All parts of the body should be kept as much as possible on the same level, and so some of the swiftest swimmers carry their heads almost wholly submerged, turning face up at intervals for breathing. The varieties of stroke were described in the initial article, vol. iv. p. 271.

The perfect distribution of muscular effort in swimming especially commends it as an exercise. Portal and Delpech[3] many years ago suggested swimming for those suffering from lateral spinal curvature. They held that the maintenance of equilibrium in the prone position would require increased energy of the muscles and limbs on the side opposite to that of the bulging ribs or hip. As a developer, many of the virtues of swimming are

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[1] Irving C. Rosse, Journal of the American Medical Association, April 19, 1890.

[2] Richard A. Proctor, Strength, London, 1889.

[3] C. H. Rogers-Harrison, Deformities of the Spine, London, 1872.

PLATE I.

Fig. 1.

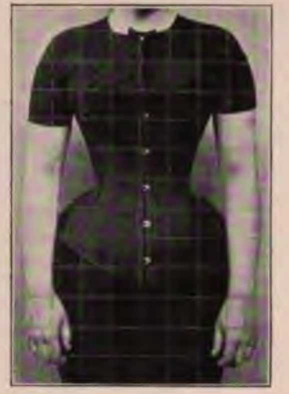


Fig. 2.

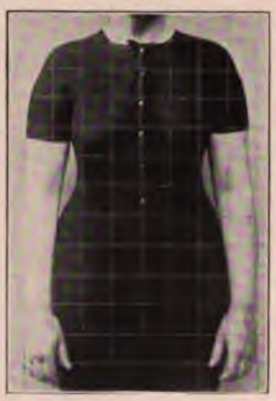


Fig. 3.

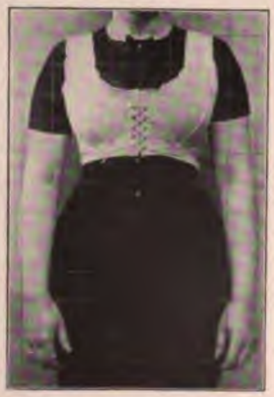


Fig. 4.

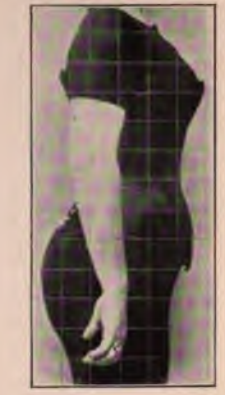


Fig 5.

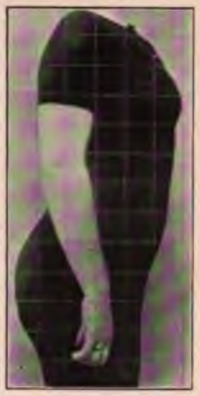
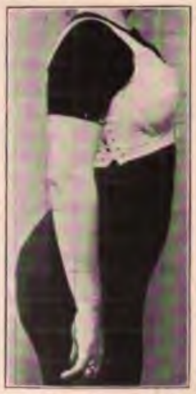


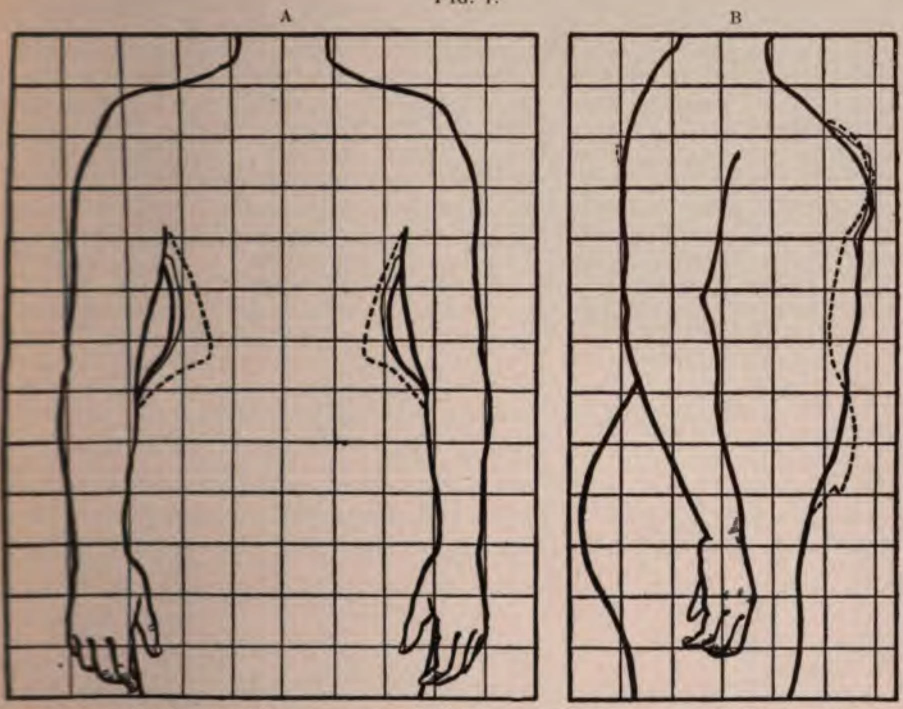
Fig. 6.



Contrasted respiratory capacity: Figs. 1, 4, corseted; Figs. 2, 5, ungirt; Figs. 3, 6, bust-girdled.
(For comparative study see Plate II.)

PLATE II.

FIG. 7.



- - - - - - Outline corseted. ━━━━ Outline uncorseted. ———— Outline bust-girdled.

Diagrams A, B, drawn to scale from outlines of photographs (Plate I., Figs. 1-6), illustrating the thoracic and abdominal compression at the waist and the unsightly abdominal bulging below it produced by the corset, contrasted with the respiratory freedom and more graceful outline of the practically nude figure and with the efficient breast support that may be enjoyed, with no abdominal and a minimum of chest constriction, by the use of a bust-girdle.

TABLE OF MEASUREMENTS, IN INCHES, OF MODEL DIAGRAMMATICALLY REPRESENTED IN PLATE II. AND PHOTOGRAPHED FOR PLATE I.

CIRCUMFERENCES.

	Bust.	Waist.	Umbilical.
Corseted	$35\frac{4}{16}$	23	$36\frac{3}{16}$
Uncorseted	$35\frac{8}{16}$	27	$35\frac{13}{16}$
Bust-girdled	$34\frac{12}{16}$	$27\frac{8}{16}$	$35\frac{8}{16}$

DIAMETERS.

	Transverse.		Antero-posterior.		
	Chest.	Waist.	Chest.	Waist.	Umbilical.
Corseted	$10\frac{10}{16}$	$6\frac{2}{16}$	$7\frac{14}{16}$	$7\frac{14}{16}$	$9\frac{1}{16}$
Uncorseted	$10\frac{4}{16}$	$8\frac{4}{16}$	$7\frac{2}{16}$	$7\frac{9}{16}$	$7\frac{14}{16}$
Bust-girdled	$10\frac{10}{16}$	$8\frac{4}{16}$	$7\frac{10}{16}$	$7\frac{10}{16}$	$7\frac{4}{16}$

found in other sports; particularly its own, however, are the cultivation of endurance, presence of mind, and the ability to preserve and rescue life.

The dangers that attend the swimmer may be summarized as those resulting from indiscretions regarding the time, place, temperature, and duration of the bath, and of exposure after it, also from failure to recognize or respect physical disability.

From five to twenty minutes should be the usual limit of sea-bathing. Chilling, blue lips, fatigue, or other symptoms of distress should signal prompt withdrawal from the water. Sea- is to be preferred to fresh-water bathing. The proper temperature can be estimated only by the reaction of the individual. Physical disability invites the opinion of a physician.

DANCING.

It is not the purpose of this article to describe the many forms of dance, or to do other than make a few general suggestions concerning this exercise in its relation to general development and to health. For the class of people who shun exercise as a duty, but are tempted to it when a pleasure, dancing is especially serviceable. Music and the reunion of the sexes make it one of "the sweetest and most charming of human enjoyments." (Homer.)

Habits formed in childhood are usually maintained throughout life. Station[1] and carriage, not only graceful but productive of health, should be acquired in early years. Suggestion as to posture in standing is usually limited to "keep the shoulders back," totally ignoring the necessity of carrying the chest high and forward, the abdomen in, and the hips back. (See Plate I., Fig. 5.) The habitual standing posture of the majority presents the abdomen as the most anterior bodily prominence. The normal individual, if facing and in contact with a wall while standing at what drill-masters call "attention," should be in contact only at the chest. This position may seem a little forced, but slight exaggeration is serviceable in an ideal. Station and gait are not of least importance in the dancing-master's province.

Many forms of solo dancing, limited, so far as adults are concerned, to the stage, may be taught children with advantage. The Scotch dances—hornpipes and forms of like character—are vigorous and suitable for the normal child. Dances of slower movement—*e.g.*, some forms of square dances, and these rarely require continuous or violent movement—may be indulged in with benefit by those who are handicapped with some organic deficiencies. The contra-indications to cycling apply to violent or continued dancing. The whole muscular system of the lower extremity is particularly involved in dances of the more vigorous character, especially the extensors of the thigh and the flexors of the leg. Where posturing and pantomime are required, not only the muscles of the trunk and the upper

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[1] Edwin Checkley, Natural Method of Physical Training, Brooklyn, 1890.

xpression. The mere
of equilibrium. In
ction of joint and limb
action of a group of
disproportion to the de-
tates them from the exe-
is called muscle-binding.
sailor's fingers, whose grasp
ers have recently recognized
result of undue or faulty action
keeping of the heel near the
leg extensors.
may be viewed through the same
indicated, however, in some cases

most necessary for dancing are usually
ise. Late hours, overcrowded rooms,
clothing during exposure to draught or
old atmosphere, and dancing when already
to gain it much criticism not wholly unde-
dancing need only be beneficial. P. E. Remy,[1]
treatise on this subject in 1824, suggested that
fully be indulged in during sunlight, in the open
drained place. Dancing under such conditions is still
peasantry of Europe, and might be by the children
ring much of the year.

BICYCLING.

of cycling upon the *sound* and the *unsound* has recently
and much exploited.

nsensus of estimable opinion strongly recommends this sport—
sharp limits—not only to those without, but also to some on account
sical defect. On the other hand, misuse of the wheel by the healthy,
lulgence in it at all by the unfit, has already proclaimed it a cause of
onic disease and of sudden death.

Let us consider the physiological influence of the bicycle on the *sound*, and study effects from immoderate as well as from moderate use. The limitations of the latter can thus be defined, therapeutic virtues discovered, and suggestions made and testimony taken as to which classes of the *unsound* may be benefited or injured.

CYCLING FOR THE SOUND.

Wheeling has fascinated many who have been faithless to other modes of physical culture. It entices out of doors and exhilarates with rapid motion easily achieved. It banishes care, and delights with constant change of scene and atmosphere. It is independent, can be enjoyed at any time, and by one. It can, however, afford the boon of companionship, and, like tennis, invites boy and girl on a footing. It is a transport, has made other sports more accessible, and can be enjoyed by many on their way to and from their daily vocations. It has been recommended[1] in rural districts as a preventive of in-breeding by facilitating courtship and marriage between those of distant localities. Finally, wheeling demands the continual exercise of a certain amount of skill and attention, diverts the mind in healthy channels, and calls into play decision, confidence, and pluck.

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[1] P. E. Remy, Dissertation médicale sur l'Exercice de la Danse, Paris, 1824.

The *machine* should be light, strong, and easy running. Its features are too universally known to need description. The arrangement by which part of a revolution of the pedals produces a whole revolution of the driving-wheel is called "gearing." The gear (*e.g.*, fifty-six inches) represents the diameter of a circle the circumference of which represents the distance travelled during one revolution of the pedals. The diameter of such a circle was originally the diameter of the driving-wheel (the front one) of the high machine, in which the application of power was direct.

Adjustment.—Its Relations to Posture, and the Effects thereof.—The higher the gear, the greater the force required to produce—and the greater the distance travelled by—a revolution of the pedals. The lower the gear, the less space covered and the less power required per revolution, and the greater the ability to climb hills.

The handles may be high or low. The high handle permits erect posture, the low necessitates a stoop. Its extreme limit is seen in the "scorcher" and racer, who ride with back horizontal.

Concerning the relative merits of these positions, we quote from Dr. Hammond's tabulated observations[2] of fourteen amateurs and fourteen professionals. The amateurs had from five to thirteen years' experience, and had ridden in that time from five to twenty-seven thousand miles, sitting erect. The professionals—seven of whom had gained national reputations, riding more and faster races than other men—had all ridden with back at right angles to legs, "half," as Dr. Hammond suggests, "shut up, like a jack-knife." The average chest-expansion of the amateurs was four-sevenths of an inch better than that of the average man and one-seventh of an inch better than that of the professionals. "The expansion of thirteen of the amateurs is above normal, and in one it is normal. With the professionals the expansion is above normal in eight, normal in one, and below normal in four." The evil influence of this posture on chest-expansion is apparent. If it has been assumed by eminent racers with comparative impunity, it by no means follows that the average man may do likewise. The racer is such by peculiar adaptability, and he devotes his life to keeping himself in condition. "It is quite probable, if professional racers of less phenomenal abilities were substituted in the second table in place of the national champions, the average chest-expansion would be found to be below normal."

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[1] Rev. E. Werre, Athletics, Industrial Health Exhibition, 1884, vol. iv. 85.

[2] G. M. Hammond, Medical Record, New York, February 2, 1895.

But this is not all. Extreme bending forward in the average saddle tends to bring the weight of the body on the perineum and to press the anterior parts against the peak of the saddle. Hence we have reports of damage resulting from pressure,—contusion and friction. Men are said to complain of frequency of micturition, of discomfort and pain in the parts around the neck of the bladder, of congestion, expected by some to cause chronic disease of prostate and urethra, also of cystitis, and, finally, of bruising and atrophy of the testes. The possibility has been suggested[1] of sterility to males arising from perineal pressure, since such has been observed in a couple of Asiatic tribes who live entirely in the saddle and are effeminate and emasculate. Women are reported[2] victims of vulvar and perineal tumefaction, bruising, and ulceration; of perineal laceration, one case from a fall forward on the peak; of ardor urinæ; of frequent micturition; and of one case of ovaritis, possibly attributable to exposure. With children there is especial fear that friction or heating of the genitalia may institute bad habits.

Finally, the posture is charged with producing spinal deformity, and it certainly does contribute to a stoop in those who bend not from the hips alone but from the back. Æsthetic reasons alone should be sufficient to condemn it. Momentary indulgence while facing a sudden puff of wind or a stiff hill is occasionally unavoidable. All the evils just described may be avoided by the erect posture on a proper support. The handle-bars should be high enough to allow the rider to sit erect and grasp the handles while his wrists are in extension and his arms slightly flexed. The elbows are best kept close to the sides.

The *sitting support* has been divided[3] into saddles and seats. Bicycle saddles resemble equestrian saddles in having a projection in front, the pommel or peak. Bicycle seats are without this prominence.

The Saddle.—The peak is one of a saddle's essentials, and its usual form is flush with the sitting surface; it is a more or less narrow bar, against which, as we have already seen, the cyclist is apt to bear down with injury if he rides "hands low." Riders should, therefore, adjust their saddles to avoid, as much as possible, peak-pressure. To tilt the pommel downward would keep it out of the way did it not at the same time slant the back of the saddle, causing the rider to slip forward. To set the peak much up occasions the same objectionable contact as to ride stooped. A horizontal adjustment or slightly upward tilt tends most to keep the rider back in the saddle. To remain there while pedalling is difficult. The thigh in the

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[1] C. W. Burr, Medical and Surgical Reporter, Philadelphia, 1896, lxxv. 42.

[2] J. H. Prendergast, American Journal of Obstetrics, New York, 1896, xxxiv. 245.

[3] A. C. Roper, Lancet, London, 1896, i. 1341.

DESCRIPTIVE ANALYSIS OF PLATES III. AND IV.

Plate III. proves essential the four requisites of a healthful saddle. (See p. 141.) Compare Plate III. with Plate IV. Note: *First.*—The outer portion of the buttock, capable of support (Plate III., Figs. 8 and 9, *A* and *B*, and Diagram), is unsupported in most saddles (Plate IV., Figs. 10 to 18). Fig. 21, Plate IV., a *seat,* properly fits the contour of the thigh and adequately supports the buttock; *saddles* should be modified to do likewise. *Second.*—The cut-out for the thigh, in saddles, is usually insufficient. Of Figs. 10 to 18, Plate IV., Fig. 10 sins most, and Fig. 15 is best planned in this regard; the application of the Diagram of Plate III. to the sitting-supports of Plate IV., Figs. 10 to 21, graphically shows their individual merits and demerits, and makes plain that the sitting-edge of a seat or saddle should be *concave,* and the degree of concavity should be approximate to the convexity of the rider's thigh. *Third.*—In the female the thighs are so much closer than in the male (Plate III., Figs. 8 and 9, *A* and *B,* and Diagram) that a narrow pommel or a seat with no pommel is indicated, to avoid chafing of the thigh and permit a seat well back in the saddle. *Fourth.*—While in females, seated erect on the tuberosities, the vulva are just above the seat plane (Plate III., Fig. 9, *C*), observe that in males, so seated (Plate III., Fig. 8, *C*), the bulbar urethra is below that plane, and necessitates a healthful saddle to have a depressed pommel, which is also preferable for women riders. Fig. 15, *B, C,* Plate IV., satisfies this requirement better than the other types in our illustration.

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[1] The plaster moulds, Figs. 8, *A*, and 9, *A*, were thus prepared. As, in bicycling, each thigh is nearly vertical at the bottom of its stroke, the models were first posed standing. They stood raised from the floor on blocks. The width of tread was six and one-half inches Each thigh, at an inch below the buttock, was encircled by a plaster bandage. These bandages were inclosed and fixed by a third. When they had set they were removed as one piece, from which the models stepped out. From this preparation the contour of the thighs was traced on a board that spaces might be sawn to admit them. The models stepped into the spaces and the boards were raised till within an inch of the buttocks. The boards were supported at the sides, and covered with plaster to the height of a horizontal plane just beneath the buttock. When this foundation had set, the sides of the board were boxed and plaster was poured in till it reached the height of the greatest profile convexity of the buttock. After the plaster had thickened a little, but while it was still fluid, the blocks were removed from beneath the feet of the models, who slipped down through the thigh spaces *till supported only by their buttocks* and by their hands, which rested as on a handle-bar. An accurate mould was thus obtained in bicycle posture of the models' thighs, perinea, buttocks, and genitals.

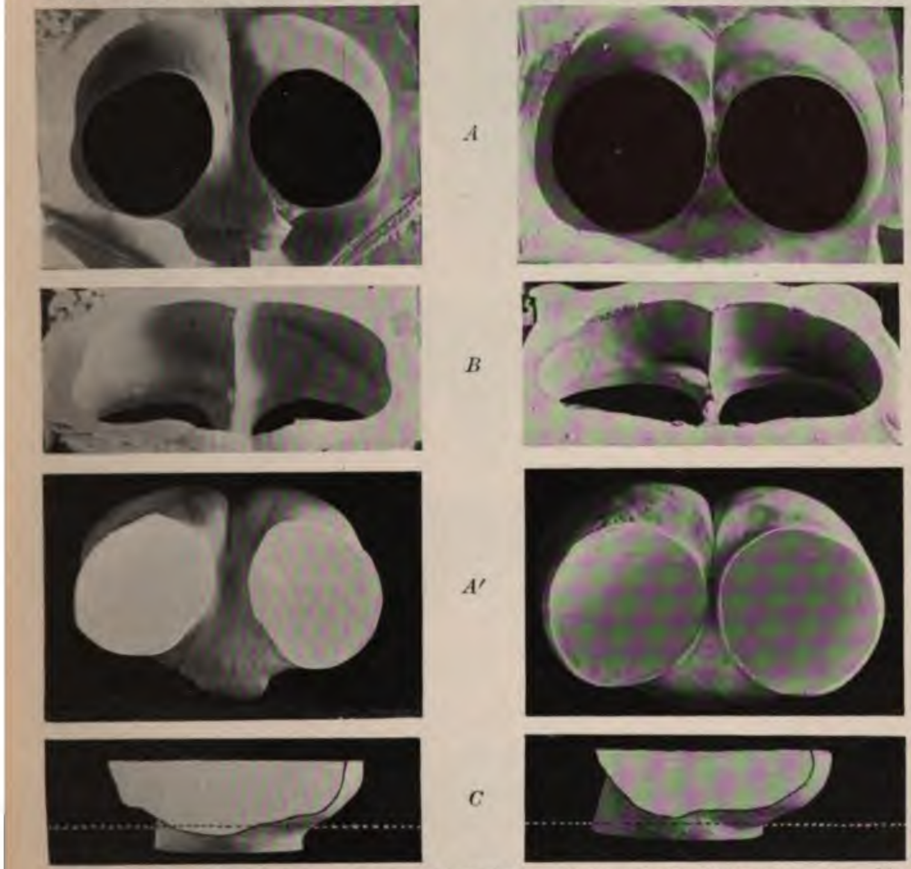
[2] The two outlines of the Diagram of Plate III. were obtained in the following manner: The moulds shown in Figs. 8, *A*, and 9, *A*, were placed on two boards, and on them from the inside of the moulds was traced the exact contour of the thighs. The wood within the thigh circumferences was accurately sawn out, the models stepped into their respective spaces, and the boards were raised till they nearly reached the gluteal folds, when supports were placed under the boards at each side; then blocks were removed from beneath the feet of the models and left them supported by their buttocks, which, being previously greased, left their imprint wherever in contact with the board. From these two outlines thus obtained the composite diagram was traced and plotted.

PLATE III.

MOULDS, CASTS, AND DIAGRAM OF THE SITTING-AREAS OF A LIVING MALE AND FEMALE MODEL IN BICYCLE POSTURE.

FIG. 8.—Plaster moulds and casts—**Male.**[1]

FIG. 9.—Plaster moulds and casts—**Female.**[1]



A, moulds, seen from above. The models' thighs passed through the black spaces; the white isthmuses between the latter were shaped by the perinea; the impressions above were made by the buttocks, and those below by the genitals; *B*, the same moulds seen from in front; *A′*, view, from below, of casts made from the above moulds; *C*, antero-posterior median sections of the above casts.
…………, line of seat-plane beneath the ischiatic tuberosities.

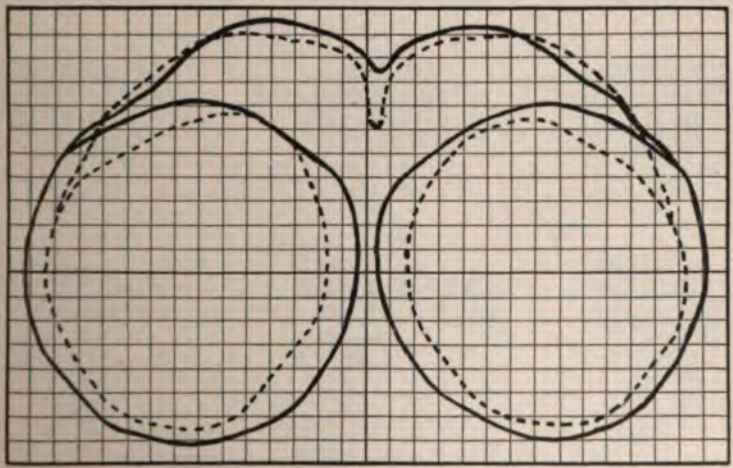


DIAGRAM (scale one-fourth life size), from impressions and tracings, illustrating the variations in the sitting areas of the male and female models posed for the moulds and casts shown in Figs. 8 and 9. ………… male outline. ——— female outline. For method of production see opposite page.[2]

PLATE IV.—SITTING-SUPPORTS. (Scale $\frac{1}{8}$ actual size.)

FIG. 10

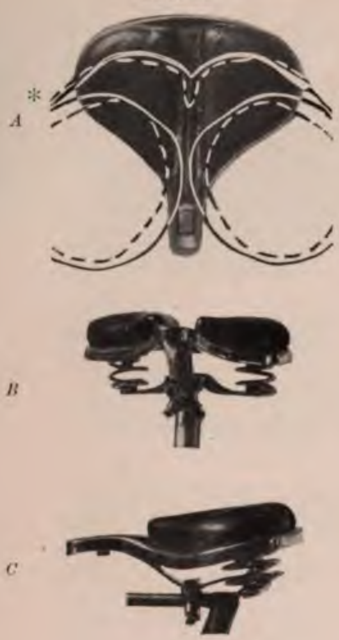


FIG. 11.

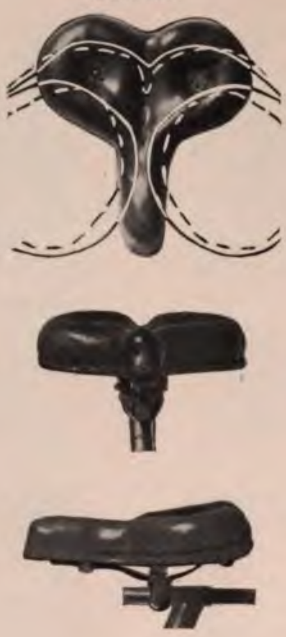


FIG. 12.

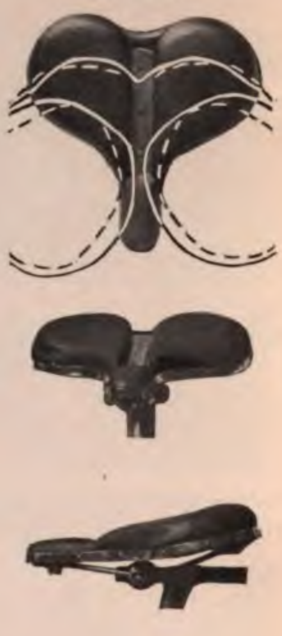


FIG. 13.

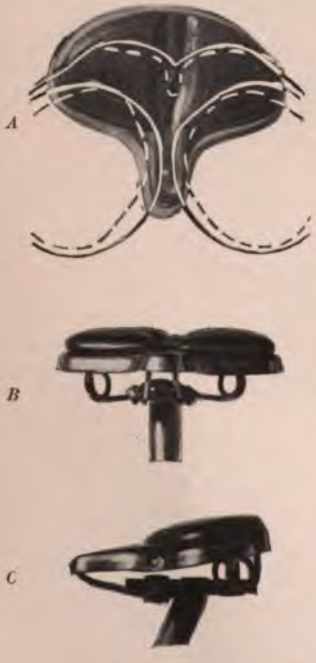


FIG. 14.

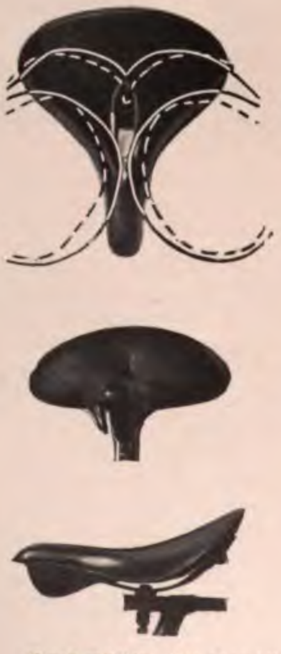
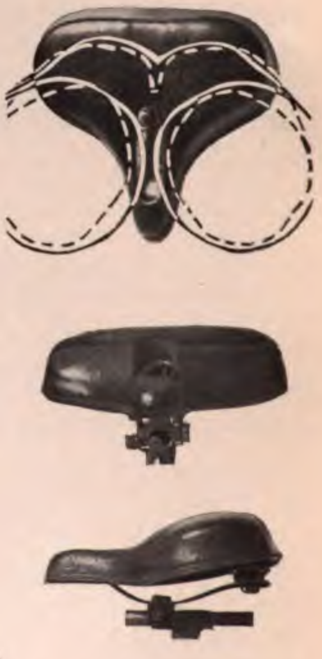


FIG. 15.



Treed saddles. (See page 141.)

* Diagram of Plate III., reduced to one-eighth life size, applied to a group of photographs of popular "sitting-supports," reduced to the same scale.

PLATE IV.—Sitting-Supports. (*Continued.*)

Fig. 16.

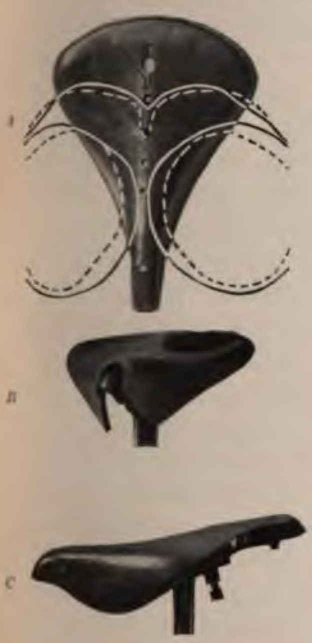


Fig. 17.

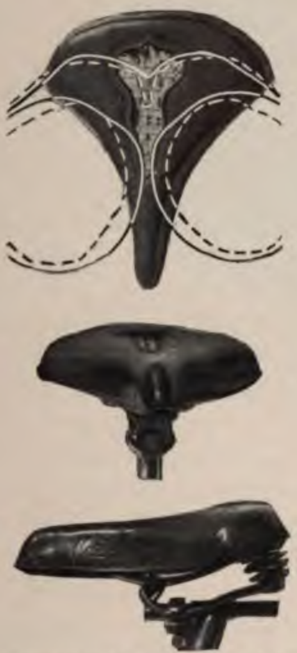
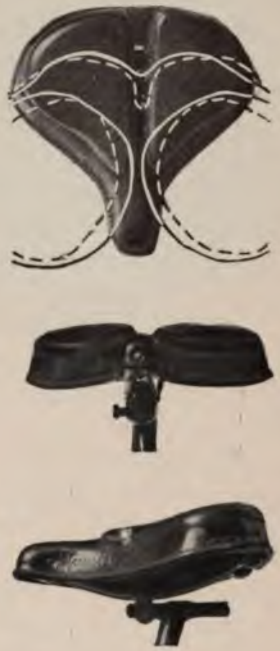


Fig. 18.



Suspension saddles. (See page 141.)

Fig. 19.

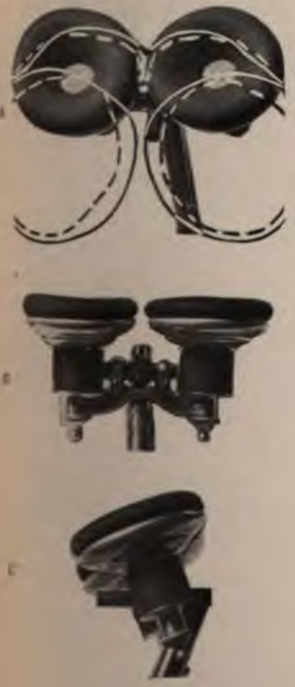


Fig. 20.

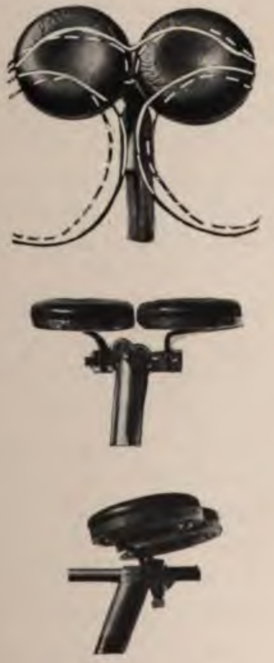
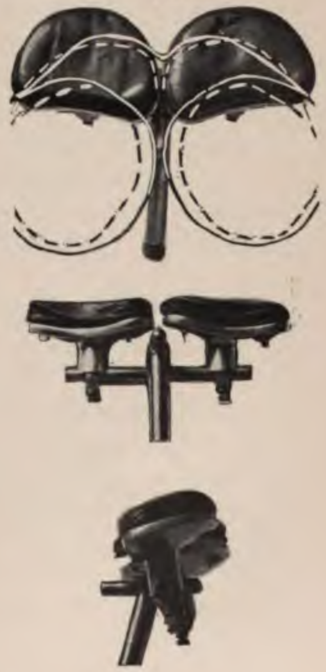


Fig. 21.



Seats. (See page 142.)

downward thrust becomes a lever, the edge of the saddle beneath the femoral crease a fulcrum, the rider's body (from this point of view) a weight that is first lifted off the tuberosities and then urged forward. Thus, by alternate extension of the thighs, there is constant tendency to leverage of the body off the saddle and onto the pommel. A vicious tendency of the pommel flush with the saddle is perineal pressure.

The saddle in the early days of cycling resembled the equestrian article in being built over a wooden frame or tree. The structure was so rigid as to punish the unhardened beginner by supporting him uncompromisingly on his ischiatic tuberosities. The complaints of the latter and fears of transmitted vibration produced the suspension saddle. This has a triangular leather seating surface, not spread over a rigid tree, but suspended by its apex in front from the peak, and at the back by its base from a transverse bar,—the cantle.

This device, at first very comfortable, becomes in time a wedge and then a fore-and-aft-slung strap, passing well between the tuberosities, relieving them, it is true, from soreness, but at the price of median pressure. The resulting disaster has already been described.

It is plain that if perineal pressure by the bicycle saddle is to be avoided: 1. The pommel must be at a lower plane than the sitting surface. 2. The saddle floor must be an unyielding frame or tree. 3. It must be broad enough to seat adequately the tuberosities. 4. The edge presented to the back of the thigh should be cut out to facilitate the latter's descent and minimize its forward levering tendency.

The gain achieved by depressing the peak is material, but at the price of a useful quality; it is undeniable that a slightly rising pommel retards the tendency to slip forward and increases the security of one's seat. It must also be admitted that the return to the tree makes a rigid seat that punishes the unhardened. The four requisites mentioned, however, are now recognized essentials, and the qualities lost are but secondary.

As individuals vary vastly not only in the fleshy but also in the bony conformation of their seat, it is manifestly impossible for one form of saddle to be a universal fit.

There are a number of models now on the market that differ sufficiently in form to satisfy individual variation. A couple of forms aim at universality by dividing the saddle into lateral halves, the distance of central separation being adjustable. Another aspires to solve the difficulty of forward projection—from thigh extension—by having the lateral halves move alternately and independently with each down-going thigh. Some object to this device on the ground of additional work in overcoming a slight spring, others assert that the force thus lost is restored on the return, while still others point out that the division of the peak converts it into two spikes, which may prove dangerous to women mounting from between the wheels.

A firm now advertises the form of its trees to be constructed from casts of clay impressions taken from models—*i.e.*, riders—at the time propelling the wheel.

Dr. Dickinson[1] has called attention to a New York firm that makes individual aluminum trees for its patrons. The method employed is that mentioned above. The tree is covered with felt and leather, has a pommel, and also a central cut-out to do away with perineal or vulvar pressure. Illustrations of this method, in Dr. Dickinson's article, show how great may be individual differences, and therefore requirements.

Bicycle seats have no pommel or peak, and are a radical escape from perineal pressure. Position in them, however, is not as secure to the saddle *habitué*, who misses the pommel. Not only does the latter keep him from slipping forward, but, presenting itself to the inner thigh, it helps him in balancing and steering.

Those who have ridden seats from the beginning, it goes without saying, do not find them insecure. They are eminently adapted for those who cannot brook the slightest perineal pressure.

In most seats the cushions are divided into lateral halves, capable of adjustment in pitch and also in breadth. Mr. Roper very justly criticises[2] a form that has circular seat halves, on account of forward leverage. This fault is avoided in a seat now on the market; the pads are semicircular or crescentic, fit the back of the thigh, and permit extension without forward leverage.

A third seat has a more generous sitting surface and automatic movable pads, like the saddle already described, but lacking its peak and the objections thereto.

The *sellette papillon*, the preferred of *seats* by Mr. Roper, suggests a half-section in leather of a shallow basin supported by a frame at the rim. It looks comfortable, but has not been advertised in this country.

Seats and saddles have an extensive literature, to which this article is much indebted, and to which those desiring fuller information are commended through use of the appended references.

The *position of the sitting support* should be at such a height that, when either pedal is at its lowest point, the rider's foot in extension and its ball on the pedal, the knee will still be slightly bent. This is achieved when the heel of the fully extended leg can be placed on the pedal. To place the support higher than this imperils the rider's security if he is on a seat, and if on a saddle obliges him to saw from side to side over the pommel. In either case, knee and ankle must strain in over-extension. To have the support lower than advised or to have it too far back entails awkward action of the thigh and a loss of power. The support should be forward, nearly over the crank axle. This brings the rider close to his work, enables him to use his weight when necessary, and his power with least waste.

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[1] R. L. Dickinson, American Gynæcological and Obstetrical Journal, June, 1896, 746.

[2] A. C. Roper, Lancet, London, 1896, i. 1841.

PLATE V.

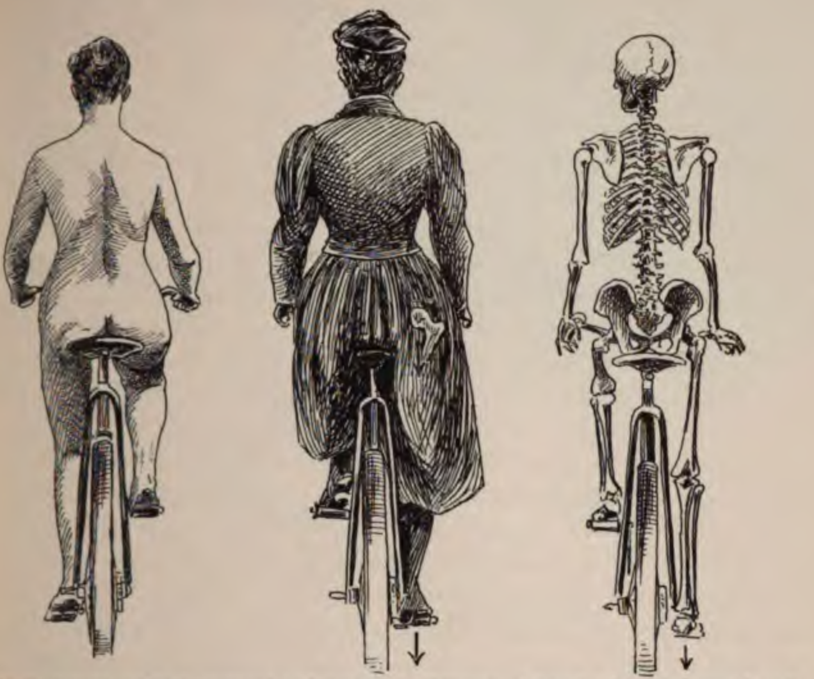

FIG. 22.—Correct saddle adjustment and posture (Dickinson). Exception—handle-bars of nude figure are too wide and cause awkward spreading of the elbows. The tread is of proper width—a perpendicular from the hip-joint drops through the centre of the pedal.

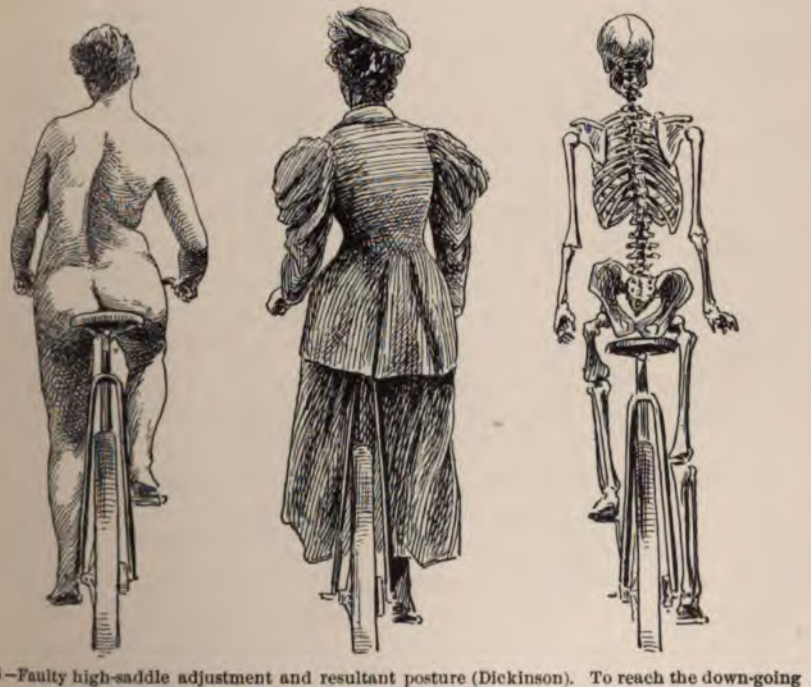

FIG. 23.—Faulty high-saddle adjustment and resultant posture (Dickinson). To reach the down-going pedals the rider must saw from side to side over the pommel.

PLATE VI.

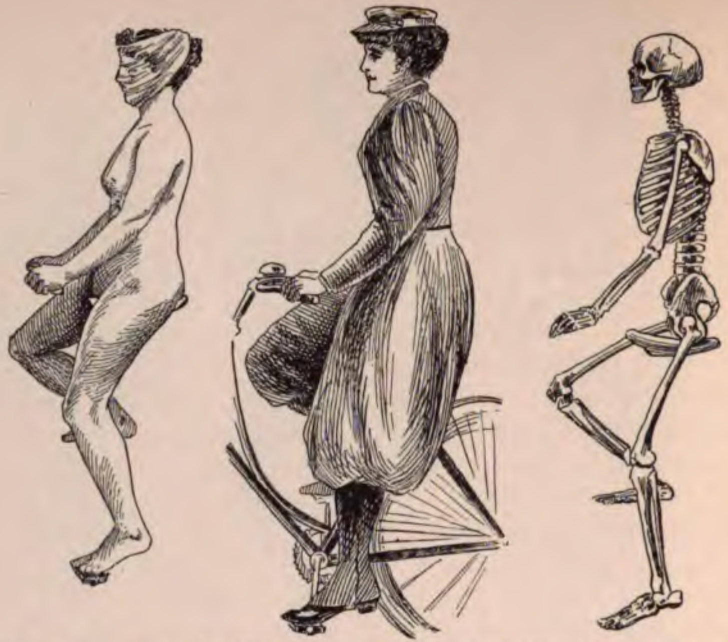

FIG. 24.—Correct posture and saddle adjustment (Dickinson). Saddle forward nearly over crank-axle and at such a height that, with the pedal at its lowest point and the ball of the rider's foot on the pedal, the foot will be in extension and the knee still slightly bent. Contrast this with the overstrained knee and ankle—on left side—in Fig. 23, in which the saddle is too high, and also with the insufficiently extended foot, the farther one, in Fig. 25, in which the saddle is too low. Note in the above figure the steep slant of the far thigh and the strong flexion of its foot while the pedal is at its highest. The heel might even have been dropped a little more, permitting a forward thrust at the dead point, and minimizing the elevation of the knee. Contrast this with the almost horizontal near thigh and the unflexed ankle of Fig. 25. Observe the height of the handle-bar, just permitting a slightly flexed elbow.

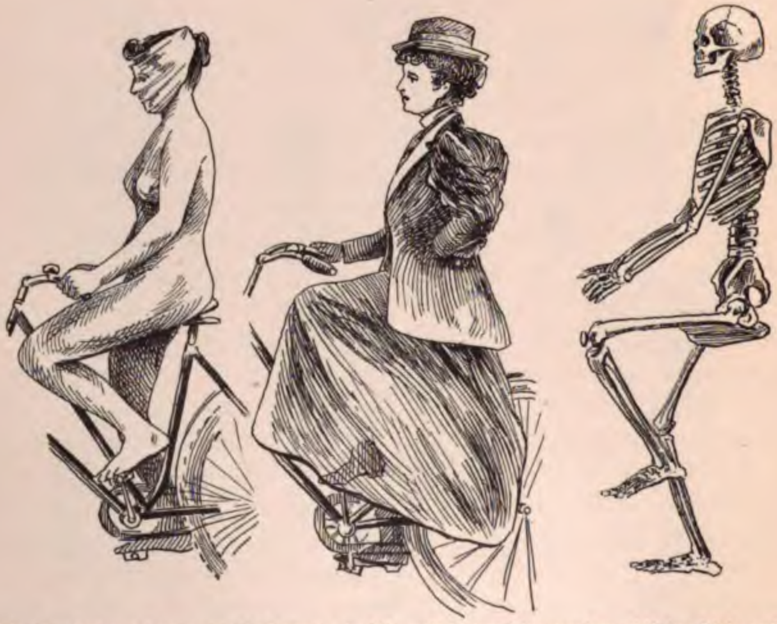

FIG. 25.—Faulty adjustment (Dickinson). Saddle well forward but too low, obliging high, awkward "walking-beam action" of the knee, which lifts and drags the skirt. "Piston-rod action" of the legs also illustrated,—*i.e.*, the mobility of the ankle-joint is almost ignored; in this figure the upper ankle should be well flexed and the heel down, while the lower ankle should be extended. Compare with Fig. 24.

The *frame* is vastly improved by a recent invention, advertised as the "cushion frame." This device, while permitting a rigid seat-post and no variation in distance between seat and pedal, provides for telescopic action of tubes joining the rear fork to the seat-post tube. This action, modified partly by a pneumatic cushion and partly by a spiral spring, minimizes vibration, jolting, and perineal contusion. Perineal contusion, doubtless, has been responsible for many complaints attributed to perineal pressure.

The *brake* is a safeguard for all. For children, women, and those lacking strength and experience it is a necessity.

Muscular development from bicycling is much wider in its distribution and the character of the exercise more violent than the unversed would suppose. Attention is again called to the fact that the maintenance of balance employs practically all the muscles of the trunk. Before the beginner has learned the trick of harmonizing the efforts of his muscles, he keeps himself up by the work of his arms, and at the end of a half-hour's bout dismounts from fatigue, not, as one would suppose, of the legs, but of the arms. Balance later becomes automatic, and the strain is imperceptibly assumed by nearly all the muscles of the body. The mere erect posture, without back support, is at the expense of a broad area of muscular contraction, as the fatigue of unaccustomed riders testifies. However, it is undeniable that those who ride hard and much, especially professionals, suffer abnormal development of the extensors of the thigh. The latter is acquired by some solely through faulty action, which may be due to improper saddle adjustment, already cited, or to what has been termed "piston-rod" action of the legs; this ignores the existence of the ankle-joint. What is called "pedalling" enlists free play of ankle and liberal employment of foot flexion and extension, with resulting calf and leg extensor development. The thigh extensors are thus relieved, and much grace and economy of force gained.

The muscles of the abdomen are employed in mounting, in rising on the pedal to ease one's seat, and in answering the increased respiratory demand. In regard to the latter, the assistance rendered by the muscles of the thorax must not be forgotten. The abdominal muscles of those who ride bent, of course, have additional claim made on them. "Cycling a cause of hernia" has rather been a false prophecy than an observation. The liability to rupture is greatest in mounting and in those who ride stooped. Continuous moderate exercise of the abdominal muscles strengthens them and lessens the liability to hernia.

We cannot here go into the physiology of muscular contraction, and must content ourselves with calling to mind that muscular contraction produces muscle-growth, and that it is accompanied by the elimination of tissue other than muscular, also of carbon dioxide and waste products.

The local effect of acute excess of exercise is local fatigue and stiffness. This is probably due to the retention in the tissues of waste products. The local result of chronic over-exertion of muscle is degeneration. The constitutional effect of acute muscular over-work is fatigue fever.

Respiration.—One of the effects of muscular contraction is increased production of carbon dioxide. This necessitates increased elimination and stimulates respiration. The stimulus is in proportion to the bulk of muscle involved, the violence and frequency of its contraction, and the relative presence of combustible tissue. As cycling employs the largest muscles of the body,—those of the lower limbs,—moves them very rapidly, and, on occasion, heavily taxes their contractility, it is a strong stimulant to respiration.

Those unaccustomed to exercise are loaded with a greater proportion of combustible tissue than those "in condition," and, consequently, flood their circulation with carbon dioxide and other products of combustion on the slightest effort. The wheelman who has ridden himself breathless has been generating carbon dioxide more rapidly than he could eliminate it. If he push this disproportion to extremes, he may become unconscious from asphyxia and die of syncope.

Emphysema and pleurisy—of the latter of which two cases have come under our care—have punished injudicious wheelmen. The dyspnœa from cycling tempts to mouth-breathing. This permits the inhalation of air that has not been filtered, moistened, or moderated in temperature by passage through the nose. Dust and germs are of course admitted, and pharyngitis, laryngitis, catarrhal throat and bronchi, as well as phthisis, are reported[1] as resulting.

The admirable influence of erect cycling on chest-expansion has already been noted.

Circulation.—Bicycle exercise, like other exercise, stimulates the circulation. The quickened blood-flow extends, of course, to the capillaries as well as to the larger vessels, and all the elements of the body enjoy increased opportunities for assimilation and nutrition. Dr. Hammond's cases—already quoted—nearly all exhibited cardiac hypertrophy without dilatation. This increase in heart-muscle bulk is the natural consequence of increase in rate and force of heart-contraction. The term hypertrophy is relative, and Dr. Hammond suggests that cycling is becoming so universal that it may improve the cardiac standard. Dr. Herschell,[2] on the contrary, has been impressed with the evil results of cycling excess upon the heart, and fears increased prevalence of cardiac disease. He points out that when cycling has produced extreme breathlessness, the latter may so stimulate the rate of ventricular contraction, while damming the pulmonary circulation, that the right heart ceases to be able to empty itself before again contracting; that the residual blood increases in bulk till it causes acute dilatation of the right heart and, possibly, death from *asystole.* Such cases have usually been observed in abnormally weak hearts. Those not pushed too far have recovered, some without, some with, permanent valve-disease from over-

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[1] A. C. Roe, Buffalo Medical Journal, 1896-7, xxxvi. 339.

[2] George Herschell, Cycling as a Cause of Heart-Disease, London, 1896.

PLATE VII.

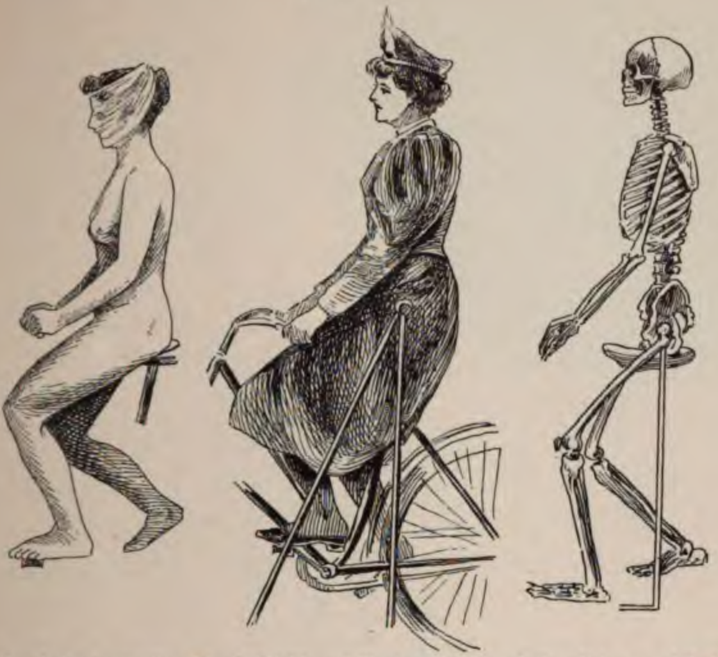

FIG. 26 and the contrasting Fig. 27 (Dickinson) illustrate by the relative distances of their pedals from a line dropped perpendicularly through the hip-joint that the power is applied most advantageously with the seat well forward. In the middle cut the handle-bar is too low, and hence the arm is too straight.

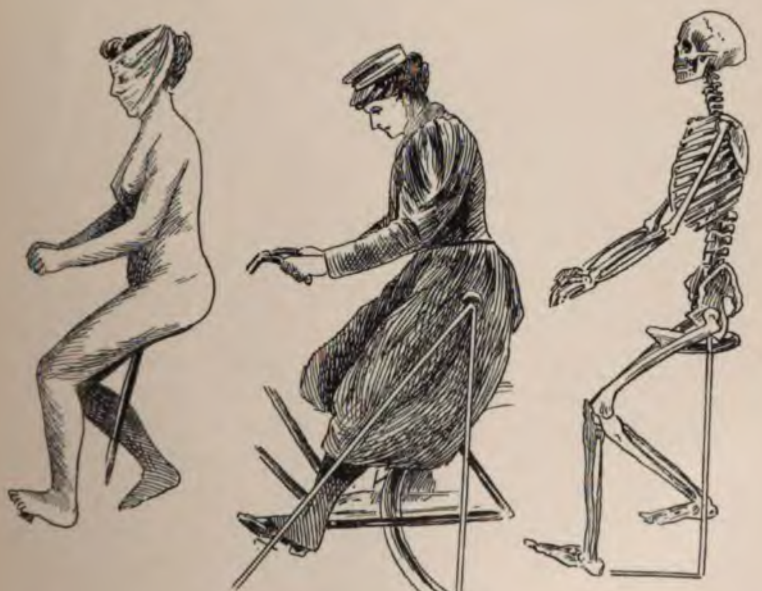

FIG. 27.—In contrast with Fig. 26 (Dickinson). Saddle too low and too far back. This adjustment has been commended to beginners. The low seat within reach of the ground makes mounting easy and inspires confidence.

PLATE VIII.

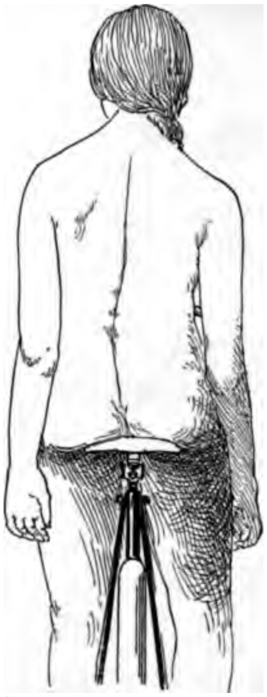

FIG. 28 Case of scoliosis "seated on an ordinary bicycle." (Kiliani.)

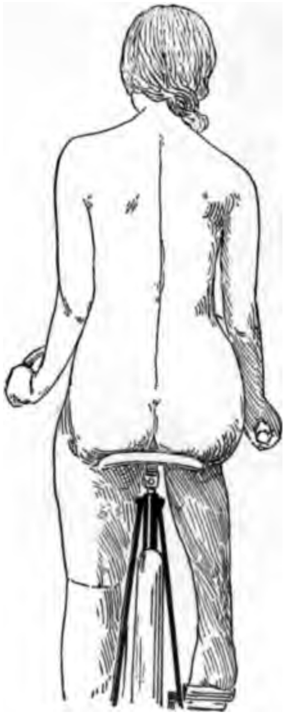

FIG. 29.—Same patient on "a modified bicycle; right pedal high, spine practically straight." (Kiliani.)

strain. On the other hand, "Rupture of the heart while cycling" has been seen as a coroner's item in the newspapers.

Derangements in function are also attributed to wheeling. Acceleration in rate is very common, and, even when temporary, is prolonged, lasting with some a couple of hours after the dismount. Either chronic or occasional palpitation is contributory to hypertrophy. Intermission of beat has been mentioned as a cycling consequence, and also angina.

Dr. Herschell seems to fear more for the heart of the trained wheelman than for that of the occasional rider. The evil consequence of acute excess is a prompt and dramatic warning that may be heeded and followed by recovery. But the trained rider may go on for years in apparent health, while structural change is slowly advancing in vessel- or heart-wall, to be discovered only when the harm is done. Hypertrophied muscle is liable to degeneration, and so the hypertrophied heart. Dilatation follows. Consequences of simple hypertrophy are increased blood-pressure and rapidity of heart-action. These factors contribute to sacculation of the aorta, and later to incompetence of its valves, with sclerosis. This is "sometimes called the *athlete's heart*." (Osler.)

The observations of Mr. Treves[1] and Mr. Turner[2] acquit cycling of the production of varices.

Nerves.—Increased blood-current rate from bicycling improves the nutrition of nerve as well as other body elements. The happy mental effect of wheeling has already been dwelt upon. The cultivation in cord and in cerebral cortex of coördinating centres and of motor areas develops with the efficiency of the parts they control.

At one time there was much comment upon the evil results to the central nervous system of the bicyclist from transmitted vibration. Mr. Turner has in the past year published some painstaking experiments,[3] from which he concludes that the symptoms attributed to vibration were really those of fatigue fever, and he seems to deny any vibratory distress.

While fatigue fever from exhaustion, independent of vibration, is of greater moment than distress resulting from vibration alone, still the latter is not a quantity so small as to be ignored, and where coexistent with exhausting work, as driving a wheel, may be largely contributory to the sum of resulting distress.

Terminal nerves also are affected, numbness of the hands being due to contusion of the ulnar received from the handles, while saddle-cramp arises from pressure and buffeting of the sciatic.

Effects upon other Tissues and upon Organs.—If bicycling increases bulk by growth of muscle, it diminishes girth by removal of fat. Vigor of circulation is increased in special organs, and freer elimination of waste products, such as carbon dioxide, urea, uric acid, etc., results, the action of the skin is increased, appetite, digestion, and absorption are improved, intestinal peristalsis and circulation are accelerated, and rectal evacuation is facilitated.

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[1] Frederick Treves, Physical Education, Philadelphia, 1892, 84.

[2] E. B. Turner, British Medical Journal, London, 1896, i. 1510.

[3] Ibid., i. 1212.

Conclusions.—The foregoing review of the effects of cycling on the *sound* gives us a fair knowledge of its good tendencies in moderation and of its evil effects in excess. Danger from accident or overdoing unsuits it for children under seven years, and makes one chary of advising it to be learned by adults over sixty. Each case must be judged by itself. There is mention[1] of Major Knox Holmes at eighty-four doing a "century" on a tricycle. For the sound female, except during menstruation and pregnancy, cycling is almost as universally recommended as for the sound male. Mr. Treves's position, that "it is doubtful if cycling can be declared to be good or suitable exercise for young women and young girls," is almost isolated.

In Recapitulation.—1. The machine should *fit* the rider in all details. These are weight, gear, length of stroke, the saddle and its adjustment, and the height and form of the handle-bar. In purchasing, arrangement should be made by which different saddles may be tried before one is selected. Women and girls need a broad seat, and, like children, should ride with low gear. Children also require shorter stroke.

2. The rules of hygiene already formulated as to clothing, particularly for girls, the care of the overheated body, the proper time for exercise, with relation to eating, etc., are again referred to for emphasis.

3. Riders must sit erect and keep well within their powers, notably children and the inexperienced. These should all walk hills of steep incline, and must not ride too fast nor too far. It has been generally recommended that one hill be walked for every two ridden. Those who eat, sleep, and feel well are within their ability; those who fail in any one of these are outside their powers. Such should reduce their exercise one-third; if necessary, intermit, and then gradually renew. Limitations must be respected.

CYCLING FOR THE UNSOUND.

Therapeutic use and contra-indications alike will be briefly considered. Conditions grossly unsuited to the exercise will, of course, be ignored. It must also be recognized that each case must be judged on its own merits, and when cycling seems advisable it should be recommended with the same explicitness as a drug. How far, how fast, whether permitted only on the level or up how steep an incline, whether in town or only on unfrequented roads, with or without an instructor or a companion, are all questions of importance. The effects should be watched and the exercise forbidden where it appears ill advised.

Muscular deficiencies in the ill developed and in some of the malformed and diseased can be improved by cycling. With special regulation of seat, handle-bar, and stroke, the wheel has been advocated in the treatment of lateral spinal[1] curvature (Fig 7). It has also been employed in the improvement or cure of paretic muscle, of hysterical paralyses and contractures, of paralyses due to anterior poliomyelitis and to neuritis, both, of course, in the chronic stage.

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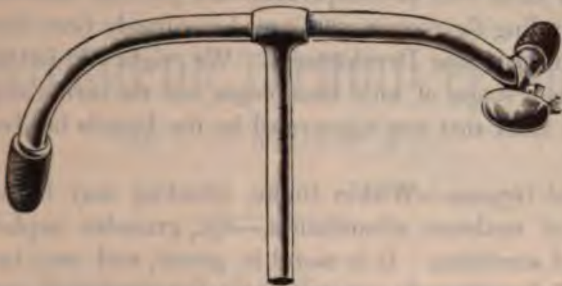
[1] E. B. Turner, British Medical Journal, London, 1896, i. 1337.

FIG. 7.



Modified handle-bar.

Joints stiffened by rheumatism or trauma have been limbered by wheeling. Active inflammatory or tubercular disease is, of course, a contra-indication.

Respiratory Diseases.—Since this exercise increases chest-expansion, even in those beginning it after thirty, and also favors general nutrition, it is most suitable for those who are still sound but are expected to develop phthisis. Likewise it has been advised for the moderately phthisical, who have been through the earlier stages and are considered cured, their affected lung area being quiescent, no blood-spitting, etc., and dulness and lack of expansion the only signs observable.

Wheeling has proved very useful for the after-effects of pleurisy, the breaking up of its adhesions, and the renewal of expansion.

Vascular System.—Functionally weak heart-muscle, tendency to fatty deposition, and, in its early stages, mitral insufficiency, may in some cases be improved by cycling, which betters the general health and retards degeneration and dilatation.

Speed, hills, breathlessness, and riding soon after meals are, of course, to be particularly guarded against in heart cases.

Contra-indications for wheeling are tendency to dilatation, fatty degeneration, aortic and tricuspid disease, aneurism, atheroma, and angina.

With regard to varices, the eminent authorities who acquitted cycling of vein dilatation differ as to the advisability of the sport for those already afflicted. Mr. Treves thinks these cases should abstain from wheeling, while Mr. Turner reports them to be benefited, and suggests the aid of a supporter in varicocele and of an elastic stocking for leg cases.

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[1] G. T. Otto Kiliani, Medical Record, New York, October 31, 1896, 615.

Nervous disorders[1] of a functional character, such as neurasthenia, hypochondria, hysterical paralyses and contractures, sexual perversion, and abnormal sexual appetite, are much benefited by cycling, which improves the general health, stimulates and diverts the mind, and takes the place of hard work for those who have superabundant physical reserves. The suitability of bicycling for certain paralyses has already been discussed under the heading of Muscular Development. We might add to the list cerebral gumma, the later stages of mild hemiplegia, and the earlier stages of ataxia. A case of the latter that was aggravated by the bicycle has been under our observation.

Abdominal Organs.—Within limits, wheeling may be advised in selected cases of moderate albuminuria,—*e.g.*, granular nephritis and that from gout and scarlatina. It is useful in gravel, and may be indulged in by those suffering from hernia and from hypertrophied prostate, if the former is happy in an efficient truss and the latter in a properly fitting saddle. Hemorrhoids, and particularly constipation, are favorably influenced by this exercise.

By women, suffering in diverse ways as such, bicycling has been used with great advantage. For example, there may be mentioned selected cases of pelvic trouble after the inflammatory period has passed, of leucorrhœa and menorrhagia, of dysmenorrhœa and of malpositions of the uterus. All the elements of such cases must be considered, and when the bicycle is advised for them it should be conditional upon the patient keeping the effects of the exercise under her physician's observation.

Contra-indications are diseases of liver, spleen, or kidney, which involve organic change, and certain types of nephritis, also vesical calculus, cystitis, and urethritis.

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[1] G. M. Hammond, Journal of Nervous and Mental Diseases, January, 1892.

ENTERIC OR TYPHOID FEVER.

By J. C. WILSON, M.D.

Definition.—An acute infectious disease, characterized clinically by fever, gastro-intestinal catarrh, a scanty eruption of isolated slightly elevated rose-colored spots which disappear upon pressure and are developed in successive crops; anatomically by lesions of the lymph-follicles of the intestines, enlargement of the mesenteric glands and of the spleen, and parenchymatous changes in the viscera. The bacillus of Eberth is present in the lesions.

Synonymes.—Infantile remittent fever; infantile hectic fever; slow nervous fever; gastric fever; typhus abdominalis; ileo-typhus.

"Enteric fever," proposed by the late Professor George B. Wood, seems the most appropriate term for the affection. It is now extensively employed among English-speaking physicians, and especially in army and navy medical circles and in government reports.

History.—Those who are interested in the growth of knowledge concerning enteric fever as a substantive affection should consult Murchison's "Continued Fevers of Great Britain," 1873, or my work upon "The Continued Fevers," 1881. The names of Bretonneau and Louis in France, Jenner in England, and Gerhard in this country are conspicuously identified with the investigations by which this disease was separated from other forms of fever and its pathology made clear. Enteric fever in infancy and childhood is of shorter duration, milder course, and lower mortality than the same disease in adult life, while its temperature conforms, usually, to the remittent rather than to the continued type. To these differences is due the fact that the early periods of life were long thought to enjoy an immunity from this disease. Rilliet and Taupin, who published nearly at the same time, in 1839 and 1840, independent descriptions of enteric fever as it appears in childhood, showed this view to be erroneous, and demonstrated the fact that the fever prior to that time generally known as infantile remittent was identical with the typhoid fever of adults.

Etiology.—Age is of great importance. Enteric fever is very rare in the first and second years of life, but from this time the liability progressively increases until adolescence. Murchison has recorded a case in an infant six months old who was attacked at the same time with her mother, and Ogle a case at the age of four and a half months. The anatomical diagnosis was made in each of these cases.

In a series of fifty cases treated in the Mary J. Drexel Home, Philadelphia, analyzed by my assistant, Dr. Henry F. Page, three occurred under five years, twenty-one between the fifth and tenth years, and twenty-six between the tenth and fifteenth years.

In a series of one hundred cases treated in the Pennsylvania Hospital, studied by my assistant, Dr. Walter Roberts, four occurred under five years, twenty-two between the fifth and tenth years, and seventy-four between the tenth and fifteenth years.

It has been stated that cases in the first two years of life have almost invariably been observed in general epidemics. To this rule there are undoubted exceptions. I have seen two well-characterized cases in private practice, no epidemic existing, one of them at the age of eighteen months, with Dr. Ashton, last year.

The Exciting Cause.—The micro-organism described by Eberth, Koch, Gaffky, and others, and known as the bacillus typhosus or bacillus typhi abdominalis, is constantly present in the specific lesions of enteric fever. This bacterium is the cause of the disease.

The bacilli are motile. They are about one-third the diameter of a red blood-corpuscle in length, and about three times as long as broad. The ends are blunt and rounded, and show areas of dense protoplasm. They grow readily in pure culture upon nutritive media of various kinds, and can be differentiated from other bacteria with which they were formerly confounded, especially the bacterium coli commune. They have been found in the blood, especially in that drawn from the spleen and the rose-colored spots. They develop in clumps in the lymph-tissues of the intestine, in the mesenteric glands, in the spleen, in the bone-marrow, and in the liver. They have been found in the bile, in the urine, and in the sweat. Their presence has been demonstrated in endocardial vegetations and in serous and purulent exudates in different parts of the body. They cannot be discovered in the intestinal discharges of adults until the seventh, sometimes not until as late as the sixteenth, day.

This organism retains its vitality outside of the body for a period of time the extreme duration of which is not known. In stagnant and in running water it is measured by weeks. Its power of growth and its pathogenic properties persist after freezing and after alternate freezing and thawing. It grows luxuriantly in milk without altering the physical properties of that fluid.

It has been shown that the bacilli retain their vitality in dried and moist fecal matter; that they also grow and multiply upon the surface of the soil, into which, if it be moist, they penetrate to a considerable depth.

Typhoid bacilli reach the interior of the body by way of the alimentary canal. If, as has been assumed in some instances to be the case, they are inhaled with particles of dust floating in the air, it is probable that they become entangled in the secretions of the mouth and pharynx and are swallowed.

Enteric fever is in the vast majority of instances contracted by drinking water which has been defiled by sewage. The prevalence of the disease in its endemic form in cities and many local epidemics are directly traceable to this cause. Water that has been recently boiled may become contaminated from an unclean filter or from ice that contains the bacilli. The origin of local epidemics has in many instances been traced to the pollution of the water-supply by a single patient, as in the case of the great epidemic in Plymouth, Pennsylvania, in 1885. In a population of eight thousand, twelve hundred cases occurred. Limited local epidemics and house epidemics may frequently be traced to a single patient.

Less common is infection by milk which has been mixed with polluted water either intentionally or in cleaning the cans.

Articles of food may be contaminated by the soiled fingers of those who prepare it or of the attendants upon the sick, and the bacilli may be transported by house-flies from the fecal discharges of patients to articles of food, especially to milk. Raw vegetables and other foods that have been washed with water containing typhoid bacilli may become the source of infection; and it has recently been shown that oysters "freshened" for the market by exposure for a short time in the fresh water of streams defiled with the sewage of towns or cities may, if eaten uncooked, give rise to enteric fever.

Limited epidemics of enteric fever, as of other transmissible diseases, have occurred under conditions in which it has been impossible to trace the source of the infection, and sporadic cases not infrequently occur under circumstances that cannot be explained. Our inability to trace the infection does not warrant the assumption that the infecting principle of enteric fever is a germ widely diffused in nature. On the other hand, there is abundant reason to believe that it is invariably derived, either directly or indirectly, from a previous case of the disease.

Pathology.—Eberth's bacilli, having gained access to the organism by the gastro-intestinal tract, penetrate the lymph-follicles of the intestines, in which they develop. They are transported by the lymph-current and the blood from the site of their primary growth to the mesenteric glands and the spleen and widely throughout the body, growing in clumps in various organs and in particular in the lymphatic tissues, for which they show an especial predilection. During this process toxic principles are elaborated, to which the constitutional phenomena are due. The bacilli are not found in the fecal discharges in the early course of the disease, but first make their appearance about the time that necrosis of the lymph elements takes place. This fact shows that they do not develop as do the bacilli of cholera,—free within the lumen of the bowel.

The greater number of the bacilli produced in the body are probably destroyed during the course of the attack. The remainder are discharged with the stools and to a slight extent with the urine; some few in the vomited or expectorated matters and in the perspiration. They are not eliminated by the expired air nor in the exhalations from the surface of the body. Enteric fever is not transmissible in the same manner as typhus fever, measles, or small-pox, and cannot be regarded as contagious in the ordinary meaning of the term. It is communicated from the sick to the well by ingesta containing the typhoid bacilli, and in the vast majority of cases by drinking water defiled with sewage.

Pathological Anatomy.—Enteric fever in young infants is rare and a fatal result is infrequent. Our knowledge of the lesions is, therefore, comparatively limited. From what is known, however, it may be assumed that the local intestinal process is much less intense than at later periods of life. There is hyperplasia of Peyer's patches progressively more marked from the upper portion of the ileum to the neighborhood of the ileo-cæcal valve, and often limited to the latter region. The solitary follicles of the small intestine and the colon are also implicated. In a considerable proportion of the cases terminating fatally the lesions of the agminate and solitary glands do not go on to necrosis, and when ulceration does take place it is seldom extensive or deep, and perforation is rare. It may be assumed that necrosis and ulceration are infrequent in cases terminating in recovery. Autopsies not rarely show in cases in which the clinical diagnosis has been fully established only slight injection and infiltration of Peyer's patches, the solitary follicles, and the mesenteric glands, conditions not uncommon in the intestinal diseases of childhood, and in scarlet fever, measles, and diphtheria. Under these circumstances the anatomical diagnosis must rest upon the results of cultures of the intestinal contents, the lymph structures, and other organs. The appendix may be the seat of ulceration.

It is in accordance with the above facts that the enteric fever of childhood in a large proportion of the cases differs markedly from that of adult life, and in particular that it is very commonly attended by insignificant intestinal symptoms. The infantile type of the disease is occasionally observed in adults. Cases have been reported in which the symptoms have been beyond question those of enteric fever, but upon section the intestinal lesions have not been found, though enlargement and softening of the mesenteric glands and of the spleen and parenchymatous changes in the other viscera have been present.

Vascular injection of the mucosa surrounding the affected glands and other evidences of mild catarrhal enteritis are present. The mesenteric glands are swollen, and the spleen is enlarged and softened.

Parenchymatous changes in the viscera are much less frequent and, as a rule, less advanced than in adults.

The mesenteric glands show changes similar to those which occur in the intestinal lymph-glands. They are early in the course of the disease enlarged and hyperæmic; later they are pale and present minute areas of necrosis. In the majority of instances these lesions appear to undergo complete resolution; occasionally, however, softening and cheesy changes take place, and the glands may ultimately become calcareous. The lymphatic glands in the fissure of the liver, the entero-peritoneal glands, and the bronchial glands show similar changes.

The spleen is enlarged in almost all cases; its substance is softened. Upon section, the pulp is brownish red or chocolate-colored, and not infrequently shows hemorrhagic infarcts.

The liver is early in the course of the attack hyperæmic and slightly enlarged; later it becomes pale. The liver-cells are granular, fatty; the nuclei indistinct or absent. Minute disseminated areas of necrosis are sometimes seen. It has not yet been definitely determined whether these areas are caused by the direct action of the bacillus of Eberth or by the action of toxalbumins. The subject has been studied by Walter Reed, who regards the latter explanation as more probable. Pylephlebitis has occurred in rare instances. Typhoid bacilli have been found in the pus of liver abscess and in large numbers in the contents of the gall-bladder.

The Kidneys.—The changes in these organs consist of cloudy swelling, with granular degeneration of the cells of the convoluted tubules. Acute nephritis is very rare in the enteric fever of childhood. Disseminated minute areas of round-celled infiltration, sometimes going on to softening and necrosis, have been observed. The appearance may be that presented by miliary abscesses. Typhoid bacilli have been found in these collections of pus. The presence of the bacilli has been demonstrated in non-albuminous urine; with greater frequency when albumin is present, and in cases of pyuria.

The Heart and Blood-Vessels.—Inflammation of the endocardium is very rare, and usually slight. Pericarditis is also rare. The myocardium is relaxed and flabby. Granular and fatty degeneration occur. The striation of the fibres is indistinct or lost. The changes in the vascular system that frequently occur in the enteric fever of adult life—namely, proliferative and obliterating endarteritis and phlebitis with thrombosis—are extremely rare in childhood.

The Respiratory Organs.—The lesions of ulcerative laryngitis may be present. Evidences of bronchitis, broncho-pneumonia, hypostasis of the dependent portion of the lungs, and splenization are common. Pulmonary infarction may occur. Croupous pneumonia is not common. Plastic pleurisy and pleural effusion are rare; the effusion is in a majority of the cases purulent.

The Nervous System.—Gross anatomical changes are rare. Increased vascularity of the pia, with punctiform hemorrhages into the brain-substance, occurs. Cerebral embolism has been noted. Abscess of the brain may be observed as a result of otitis. After death late in the course of the attack, œdema and moderate distention of the ventricles may be present, and are to be attributed to the wasting of the cerebral substance.

Among the less common complications of enteric fever in childhood may be enumerated parotid bubo; gangrenous inflammation of the mouth or genitalia; peritonitis following perforation, or as the result of infection through the wall of the gut; suppurative arthritis; multiple abscesses, and furunculosis. The lesions of tuberculosis are not infrequently observed in the bodies of children who succumb to enteric fever.

Symptomatology.—The modifications of the general clinical course of enteric fever peculiar to early life are common in children under ten years of age. Between the tenth and the fifteenth year many of the cases present the symptoms seen in adults. On the whole, the reaction to the infection in early life is less intense than later. Symptoms referable to the intestinal lesions are less conspicuous than those due to derangement of the nervous system. The onset is often abrupt; the temperature usually conforms to the remittent type throughout the attack; the duration is shorter, and serious complications and sequels are far less frequent.

Onset.—A period of prodromes, characterized by lassitude, headache, coated tongue, and loss of appetite, and the insidious onset, with gradual rise of temperature, are less common than in adult life. A considerable proportion of the cases begin abruptly with symptoms of acute indigestion and a sudden rise of temperature. In the combined series of one hundred and fifty cases above referred to, the onset of the attack was abrupt in forty-one. Vomiting is not uncommon; it occurred in twenty-two cases out of one hundred and fifty. Convulsions or chills are rare. Early bleeding at the nose is less frequent in children than in adults. In other instances the symptoms of the onset are those of an ordinary influenza,—repeated shivering, headache, and rapid elevation of temperature. Or, again, the patient may complain of sore throat, and upon inspection there is found an erythematous angina, with enlargement of the tonsils. In rare instances the attack begins with the symptoms of croupous pneumonia, and in the course of twenty-four hours the physical signs of consolidation of the lung may be made out. Cases presenting these phenomena may be arranged in two groups: one characterized by an early localization of the typhoid process in the lung, the other by the association of two coincident pathological conditions,—pneumonia and enteric fever.

The Digestive System.—Loss of appetite is marked throughout the attack. Upon the occurrence of defervescence, however, hunger is often urgent. There is moderate thirst. The tongue early in the course of the disease is moist, slightly swollen, and often covered with a whitish fur, the edges and tip being of a bright red color. The dry, fissured tongue of the graver cases in adult life is not often seen. Sordes is likewise comparatively rare. Cracking of the lips and fissures about the angle of the mouth are common in the severer cases. The mucous membrane of the palate and pharynx is not infrequently the seat of a mild catarrhal inflammation. Membranous pharyngitis is not common. Suppurative otitis media occurs with great frequency in some epidemics.

Nausea and vomiting are much more common than in adult life. Persistent vomiting is usually due to some complication, such as nephritis, meningitis, or peritonitis.

Intestinal Symptoms.—Liquid stools occur at some time during the course of the disease in more than half the cases. Diarrhœa is rarely profuse, the number of discharges commonly ranging between two and five in the course of twenty-four hours. They are usually yellowish or brownish-yellow in color, thin, with now and then scybalous masses, sometimes mucus, occasionally undigested milk-curds. Diarrhœa was noted in seventy-seven out of one hundred and fifty cases under fifteen years of age. In a small proportion of the cases there occurs a regular daily movement of semi-solid consistence, not differing in appearance from that of health. In a larger proportion there is constipation. This condition was observed in forty-two out of one hundred and fifty cases. The state of the bowels is much influenced by the amount and character of the aliment. Tenderness upon pressure in the ileo-cæcal region or around the navel, and exceptionally over the whole abdomen, occurs. It is, however, less common and less marked than in adults. Tympanites is relatively infrequent in childhood, and when present is usually of moderate degree. It was present to some degree in seventy-four out of one hundred and fifty cases. Some degree of tympanites is frequently associated with constipation, and in rare cases enormous abdominal distention occurs in children. Very great tympanites is associated with deep ulceration.

Intestinal hemorrhage is less common than in adults. It occurs most frequently at about the end of the second week. In a series of one hundred cases of enteric fever in children observed in the Pennsylvania Hospital intestinal hemorrhage occurred in two instances. The first was a child six years old, who had large hemorrhages upon the fourteenth and seventeenth days of the attack; the second, a child aged thirteen, who had small hemorrhages on the twelfth and thirteenth days of the attack. Both cases recovered. No case of intestinal hemorrhage occurred in fifty cases treated in the Mary J. Drexel Home. Hemorrhage is less frequent under the age of ten than it is between the tenth and fifteenth years. The amount varies from a slight trace to a copious discharge of bright red blood. Concealed hemorrhage may occur. When the blood loss is considerable, the symptoms of internal bleeding are present, and after a time blood appears in the stools.

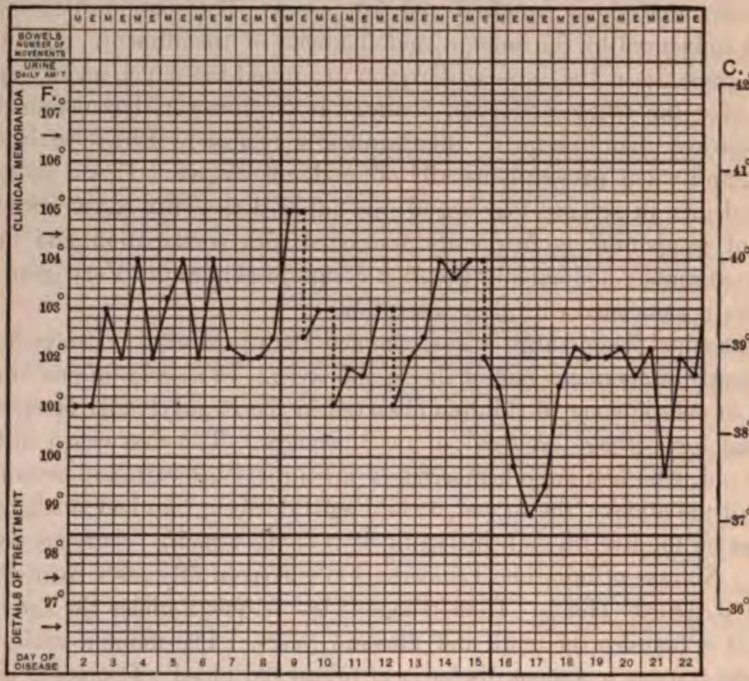
Henoch has reported a case in which hemorrhage occurred in a girl ten years old in the course of a relapse which began in the third week. There was at first an insignificant bleeding, which was followed on the next day by a very copious hemorrhage, with fatal collapse. Fatal intestinal hemorrhage occurred in a child twenty-two months old, in whom, upon post-mortem examination, the lesions of enteric fever were found.

Hemorrhage is attended by an abrupt fall of the temperature, as shown in the accompanying chart.

Perforation of the intestinal wall is extremely rare in childhood. In one hundred and fifty cases under fifteen years of age perforation occurred in but a single instance. The patient was thirteen years of age. The symptoms developed suddenly on the twenty-eighth day of the attack. Death followed in a few hours.

Henoch has recorded a case in which perforation occurred in a boy aged eleven in the fifth week after convalescence had apparently been established. Statistics present a wide range of variation in regard to the frequency in children. Among two hundred and thirty-two cases, according to Barthez and Rilliet, it occurred in three only. Among seventy-three persons in whom this accident is noted, Murchison found fourteen to be under fifteen years of age.

CHART I.



Severe enteric fever; hemorrhage on the ninth day; girl nine years old. Seen in consultation with Dr. S. Solis Cohen.

The occurrence of this accident is often preceded by hemorrhage. The perforation is usually situated in the lower part of the ileum, but it may be in the caput coli or in the appendix. Acute septic peritonitis immediately results. This is almost always diffuse, though it may in rare instances be circumscribed by the immediate formation of adhesions to an adjacent loop of gut or other viscus. The occurrence of perforation is manifested by abdominal pain, which rapidly extends over the entire abdomen, symptoms of collapse, vomiting, and the rapid development of tympanites. The abdominal walls are rigid, there is extreme tenderness, and the patient lies with thighs flexed upon the belly. Peritonitis may arise without actual perforation, in consequence of infection at the seat of ulceration through the serous coat of the intestine. To this category must be referred the majority of the cases in which, during the course of enteric fever, the clinical phenomena indicate a local inflammatory process, starting in the ileo-cæcal region and terminating in recovery.

I have reported an instance of this kind.[1] The patient was a puny girl twelve years of age; her defervescence began in the third week. On the seventeenth day the morning temperature was normal, and on the twenty-first defervescence was complete. On the twenty-third day after the beginning of the sickness the patient suddenly complained of severe pain in the abdomen and had a chill, which was followed by vomiting, intense nausea, and thirst; the axillary temperature was 104.5° F.; the belly tense and tender; the pulse small, hard, and 140. Decubitus was dorsal, with the knees drawn up. The whole abdomen was exquisitely tender upon palpation, the focus of tenderness being in the right iliac fossa. The urine contained a trace of albumin and a few hyaline casts. Recovery took place in three weeks. On two occasions in the course of the following month, however, a larger meal than usual was followed by fever, lasting two or three days, and by tenderness in the right iliac fossa. A similar case, also terminating in recovery, has recently occurred in a boy fifteen years old in my service in the German Hospital.

Peritonitis may also occur as the result of the rupture of a pseudo-abscess, caused by the softening of a mesenteric gland or in consequence of the rupture of an abscess in the gall-bladder, rupture of the spleen, or, very rarely, in consequence of the rupture of an abscess in the abdominal wall. The comparative infrequency of perforation and hemorrhage in childhood is due to the mildness of the intestinal lesions.

The Spleen.—Enlargement of this organ occurs in over ninety per cent. of all cases. The increase in the volume of the organ may be usually recognized by percussion and palpation towards the end of the first week, sometimes as early as the third or fourth day. The enlargement of this organ may usually be made out at about the time the rash appears. The more rapid the rise in fever the earlier does the spleen enlarge. The lower border of the area of splenic dulness varies from two to five centimetres below the border of the ribs. Great restlessness on the part of the child or the presence of tympanites may render it difficult to determine positively the presence or, when present, the extent of splenic enlargement. Enlargement of the spleen was definitely made out in ninety-two of one hundred and fifty cases. If the tumor of the spleen persists after defervescence, the danger of relapse is to be apprehended. Tenderness upon palpation is occasionally observed. Spontaneous pain in the splenic region is not common; it may result from perisplenitis. Softening infarcts may prove the starting-point of peritonitis.

The Circulatory System.—The pulse-rate during the active febrile movement in one hundred and forty-three cases ranged from 70 to 134 per minute. The pulse-frequency in enteric fever is relatively low as compared with the elevation of temperature, and this want of accord is more marked in older children. Dicrotism is uncommon in young children.

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[1] Philadelphia Medical Times, December 11, 1886.

Endocardial murmurs were noted in eighteen out of one hundred and fifty cases. The point of maximum intensity when recorded was usually above the apex or at the base. In these cases, with a single exception, the murmur disappeared during convalescence. No murmur was detected in the seven cases under five years of age.

The Nervous System.—Nervous symptoms are almost constantly present. They are, however, much less marked in infancy and childhood than in adult life. Cases in which they are absent through the whole course of the attack are rare.

Headache, common enough in older children, is apparently rare in those under ten years of age. When present, it is usually referred to the forehead or temples, but may affect the whole head. It subsides early in the course of the attack. It was noted in one hundred and three out of one hundred and fifty cases. Under five years, seven cases; headache observed in five: between five and ten years, forty-three cases; headache in twenty-six: between ten and fifteen years, one hundred cases; headache in seventy-two.

The mental condition is characterized by apathy and indifference, even in cases of moderate severity. The child lies with its eyes half closed, lightly somnolent; when spoken to, it turns away or replies in monosyllables, frequently with evidences of irritation, often, even in advanced childhood, refusing to show its tongue, or, if showing it, forgetting to draw it in again unless told to do so. There is some degree of impairment of hearing, and dilatation of the pupils is common. Sleep is light and easily broken, and insomnia is sometimes troublesome. Vertigo may occur.

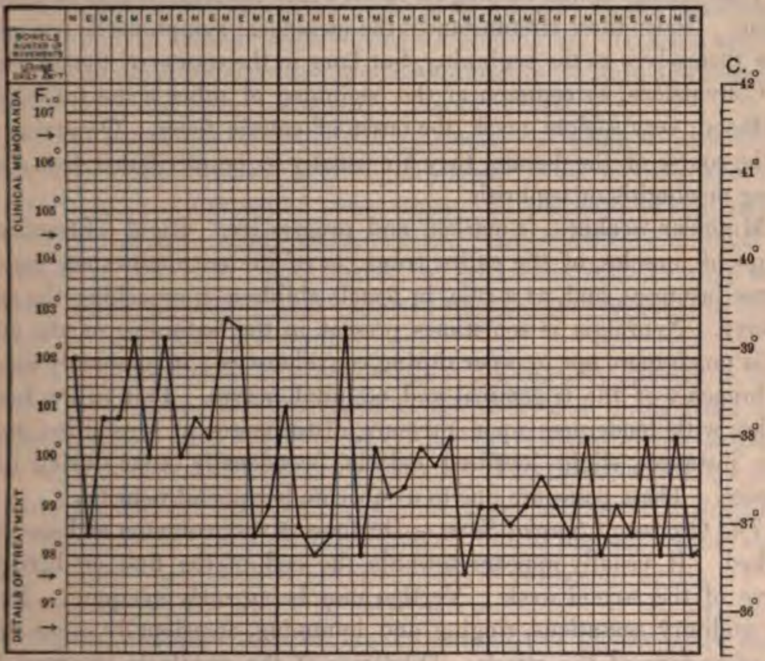
The well-known correspondence between delirium in sickness of every kind and the intellectual development and mental habit of the individual is in accordance with the fact that in enteric fever in childhood apathy, somnolence, and stupor are more common than delirium. The last symptom was present in twenty-four out of one hundred and fifty cases. In infants and young children the familiar wandering delirium of enteric fever in adults may be accompanied or replaced by sudden sharp and prolonged outcries with excitement.

In a girl, aged twelve, of refined parents and carefully brought up, the delirium took the form of coprolalia, and for a period of ten days profane and obscene words and phrases were repeated with a richness of vocabulary and a volubility not less astonishing than distressing.

The marked nervous symptoms seen in grave cases in adults—subsultus tendinum, carphologia, coma vigil, and tremor—are rarely seen in young children. In the worst cases, especially in older children, all these symptoms may occur. Grinding of the jaws, tremor, and deep coma are symptoms of ominous import. Cutaneous hyperæsthesia is frequently well marked and extensive. It is in the majority of instances restricted to the abdomen and lower extremities, and may be associated with rachialgia and points of spinal tenderness.

Occasionally the onset of the attack is abrupt, with intense headache, vomiting, painful rigidity of the back of the neck, pupillary phenomena, squint, and general hyperæsthesia,—symptoms which suggest meningitis. Such cases have been described as the cerebro-spinal form of enteric fever. In the early days of the disease the differential diagnosis cannot be made, but the appearance of rose-spots, the occurrence of a splenic tumor, the range of the temperature, and the reaction to the Widal test usually render the diagnosis a simple matter towards the end of the first week. At this period the nervous symptoms commonly subside, and the subsequent course of the case is that of an ordinary enteric fever of varying severity.

CHART II.



Cerebro-spinal form of enteric fever; boy aged four years; Pennsylvania Hospital.

A Russian boy, aged four years, was admitted to the Pennsylvania Hospital on the fourth day of an illness attended with pain in the limbs and swelling and tenderness of the belly. Upon admission there was slight painful rigidity of the muscles of the back of the neck, together with spasmodic contractions of the muscles of the arms and hands. Except dilatation, there were no pupillary symptoms, no strabismus, no trismus. There was no disease of the ears. A few hours after admission the patient's temperature suddenly fell from 102° to 95° F. It rapidly rose again. The child was fretful, and preferred to lie upon his side with his limbs strongly flexed. He cried out with pain upon being moved. Ankle-clonus was present, and the knee-jerks were increased. A small amount of albumin, with hyaline and granular casts, appeared in the urine. There were one or two herpetic vesicles upon the lips and a few pustules upon the occipital region and upon the trunk; the tongue was moist and thickly coated with a white, pasty fur; it was red at the tip and edges. Upon the third day after admission a group of typical rose-spots appeared upon the abdomen and chest and the spleen was made out to be enlarged. About this time there was rapid amelioration of the nervous symptoms; the reflex phenomena subsided; pain on movement disappeared, and the case presented the symptoms of a typical enteric fever of moderate severity in childhood. Treatment, systematic cold bathing. Convalescence retarded by furunculosis.

True cerebro-spinal meningitis in very rare instances occurs as a complication. Under these circumstances the meningeal symptoms do not usually show themselves at the beginning, but later in the course of the attack.

Convulsions, so common at the beginning of other acute infections in childhood, very seldom mark the onset of enteric fever. Occurring later in the course of the disease, they are usually to be attributed to a complicating meningitis or nephritis.

Muscular weakness is marked and progressive. Rigid contractions of groups of muscles, of the entire trunk, or of the extremities are met with in rare instances, and, as a rule, in female children approaching the age of puberty. Neuralgia is sometimes present in the beginning of the attack, but is much more apt to occur during convalescence; it commonly involves the branches of the trigeminal and occipital nerves. Pain in the feet and ankles, with tenderness upon pressure, disturbances of sensibility, and, in some instances, slight localized œdema, occasionally occur during convalescence. These pains are due to a subacute peripheral neuritis.

The Organs of Special Sense.—Deafness is very common and sometimes marked. It usually appears towards the end of the first or during the course of the second week. Vertigo may be an early symptom. Subjective auditory sensations, ringing and humming, occasionally occur during the early days of the attack. Dilatation of the pupils is observed in at least three-fourths of the cases.

Aphasia without hemiplegia may occur. Among the rare nervous accidents of enteric fever in children are paraplegia, hemiplegia, and paralysis of the ocular or laryngeal muscles.

The knee-jerks and the cutaneous reflexes are slightly increased. In deep stupor they may be enfeebled or absent altogether.

Insanity may develop after the defervescence,—post-febrile insanity. The nervous symptoms peculiar to the attack are commonly most severe after the disease has become fully established and subside as defervescence takes place. Those that develop during convalescence usually run a course of some weeks or months, with gradual improvement, and in a majority of the instances ultimately terminate in complete recovery.

The Urinary System.—The changes are those observed in the other acute infectious diseases. There is early in the course of the attack a diminution in the quantity of urine, which may not reach one-half the normal amount. With convalescence the secretion of urine becomes copious, while its specific gravity falls. The reaction is usually acid. The specific gravity early in the course of the attack may range from 1.020 to 1.030. With defervescence it falls below the normal. The quantity of urea present in the urine has been found by some observers to be increased; by others, diminished. Uric acid is increased. Copious deposits of urates may occur at any time in the course of the attack. The chlorides are diminished sometimes to a mere trace.

Albuminuria is not uncommon. This condition was noted in thirty-one out of one hundred and fifty cases. In a great majority of the instances the albumin is present in mere trace,—febrile albuminuria. If acute nephritis occur as a complication, the amount of albumin may be large and associated with renal casts and blood,—the renal form of enteric fever.

Pyuria is not common in children. Retention of urine is unusual.

The toxicity of the urine is much increased. This increase occurs independently of the elevation of temperature. It continues throughout the febrile movement and during convalescence. Roque and Weill found the elimination of toxins in cases treated by systematic cold bathing to be uniformly increased, and in some instances the increase amounted to five or six times the amount of the normal.

The Diazo-Reaction.—This reaction, first described by Ehrlich in 1882, consists of a peculiar color developed in the urine and in the foam when the urine is shaken in a test-tube, by the action of diazo-benzine-sulphuric acid in the presence of an excess of ammonia. Two test-solutions are used: first, a five per cent. solution of sulphanilic acid in a solution of hydrochloric acid in distilled water (fifty cubic centimetres to one thousand cubic centimetres), and, second, a one-half per cent. solution of sodium nitrite. Forty cubic centimetres of the first and one cubic centimetre of the second are mixed together. Equal parts of this mixture and of the urine are placed in a test-tube. A strong solution of ammonia is then allowed to trickle down the side of the tube. If the reaction occurs, a dark garnet or cherry-red disk appears at the junction of the ammonia and the fluid, and upon brisk shaking there develops a uniform red color throughout the fluid, with a delicate pink tinge upon the supernatant foam. After standing for some hours an olive-green precipitate is deposited. With normal urine the red color is not developed, and the foam is brownish yellow. This test acquires its value from the fact that the reaction occurs in the greater number of cases of enteric fever at some time during the course of the attack. It occurs, however, in several other diseases, especially in acute miliary tuberculosis, chronic tuberculosis, measles, scarlatina, malarial fever, and pneumonia.

Menstruation occurring during the attack in girls at puberty is sometimes profuse and prolonged.

The Skin.—The eruption appears in children somewhat earlier than in adults. It is not invariably present. This phenomenon was noted in one hundred and thirteen out of one hundred and fifty cases. In seven cases under five years the rash was present in all; in forty-three cases between five and ten years it was present in thirty-seven; in one hundred cases between ten and fifteen years it was recorded as present in sixty-nine only. It may be met with as early as the third or fourth day. There is no relation between the abundance of the rash and the severity of the other symptoms. In some mild cases there is copious rash. The individual rose-spots differ in no respect from the eruption as seen in adults. They are not found during convalescence, but reappear, together with the other characteristic symptoms of the disease, upon the occurrence of relapse. They are not seen upon the dead body. They are most frequently found upon the abdomen and the lower part of the chest anteriorly. They are frequently found upon the back, especially between the shoulder-blades, and are occasionally present upon the upper part of the thigh. In doubtful cases they should be sought for in these situations. In rare instances the typhoid rash is encountered upon the arms and legs, and more rarely still a few spots are seen upon the face.

Erythema.—A diffuse, faint, erythematous rash is sometimes observed in children during the first week. It is commonly most distinct over the abdomen and over the flexor surfaces of the limbs. Its usual duration does not exceed three or four days, though Murchison states that it occasionally persists throughout the attack. It is not peculiar to enteric fever, but is met with in other febrile affections, especially in individuals of fair complexion and delicate skin. If the erythema be prominent, and particularly if it be associated with a slight sore throat, the disease may be mistaken for scarlatina.

Herpes is less common in enteric fever than in other febrile affections. It occurs, however, in proportionately greater frequency in children than in adults.

Urticaria also is sometimes encountered as an accidental rash. Abundant sudamina occur late in the course of the disease in cases attended by free sweating.

The *tache cérébrale* may be produced in all cases.

In the early course of the attack the skin is usually dry; the palms of the hands are dry and yellowish in color. Sweating is not infrequent in the latter part of the attack. Slight desquamation may be observed during convalescence. The hair falls out, and transverse, lustreless bands, indicating the disturbance of nutrition that has attended the course of the disease, appear upon the nails.

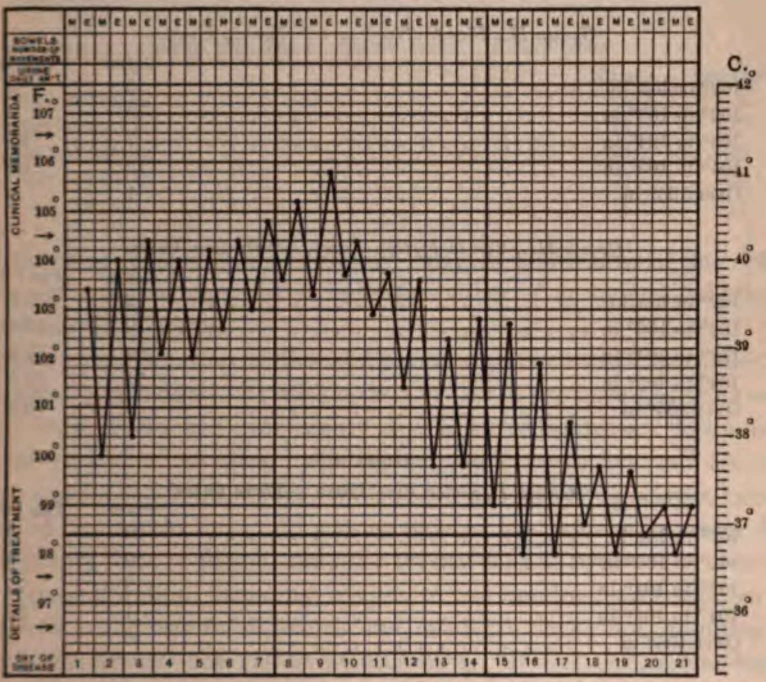
Emaciation is marked. Boils and abscesses of the skin occur during convalescence. These lesions are usually distributed upon the back, buttocks, and outer aspect of the arms and thighs. Furunculosis appears to be more common in cases treated by systematic cold bathing than in others.

Bed-sores are not common in childhood. In rare instances in grave cases superficial sloughs may occur over the sacrum or the trochanters, or at the elbow, the heel, or the occiput.

I have never observed *taches bleuâtres* in childhood.

The facial expression of the older children ill of enteric fever has the same peculiarities as that so often described in adults. We see here the dull, weary expression, the pallid countenance, with dusky flushing over one or both cheek-bones, a facies the significance of which is accentuated by dilatation of the pupils. In younger children, however, in mild cases, and in those treated by systematic cold bathing, these peculiarities of the physiognomy are not usually present.

CHART III.



Severe uncomplicated case of enteric fever; boy aged eight years; treatment expectant symptomatic. Seen in consultation with Dr. Fisher, of Bound Brook, New Jersey.

Fever.—The temperature is invariably elevated, ranging from 101° to 106° F. I have no knowledge of afebrile enteric fever in childhood. In the milder cases the rise of temperature is gradual and the ordinary manifestations of fever are frequently not pronounced, the actual condition being revealed only by the use of the thermometer. The gradual access of fever does not, however, invariably occur. In a considerable proportion of the children the onset of the attack is abrupt and the rise of temperature takes place more rapidly than in adults. It is in this group of cases that defervescence by rapid lysis, or in some instances by crisis, occasionally happens. In some uncomplicated cases the range of temperature closely conforms to [illegible] in the majority of instances the peculiarities of the temperature [illegible] the term "infantile remittent" are ob[illegible] [illegible] analysis of the temperature range in one [illegible].

[illegible] TEMPERATURE.

SERIES A.

[illegible] under Five Years. Four Cases.

	Cases.	Per Cent.
[illegible]	0	0
[illegible]	0	0
[illegible]	0	0
[illegible]	3	75
[illegible]	1	25

Group II., Five to Ten Years. Twenty Cases.

	Cases.	Per Cent.
102° or less in	0	0
102° to 103° in	0	0
103° to 104° in	7	35
104° to 105° in	12	60
Over 105° in	1	5

Group III., Ten to Fifteen Years. Sixty-four Cases.

	Cases.	Per Cent.
102° or less in	2	3.1
102° to 103° in	10	15.6
103° to 104° in	24	37.5
104° to 105° in	24	37.5
Over 105° in	4	6.3

SERIES B.

Group I., under Five Years. Three Cases.

	Cases.	Per Cent.
102° or less in	0	0
102° to 103° in	0	0
103° to 104° in	2	75
104° to 105° in	1	25
Over 105° in	0	0

Group II., Five to Ten Years. Twenty-one Cases.

	Cases.	Per Cent.
102° or less in	1	4.7
102° to 103° in	3	14.3
103° to 104° in	3	14.3
104° to 105° in	11	52.0
Over 105° in	3	14.3

Group III., Ten to Fifteen Years. Twenty-six Cases.

	Cases.	Per Cent.
102° or less in	1	3.6
102° to 103° in	2	7.6
103° to 104° in	4	15.3
104° to 105° in	12	46.0
Over 105° in	7	26.0

COMBINED TOTALS.

Group I., under Five Years. Seven Cases.

	CASES.	PER CENT.
102° or less in	0	0
102° to 103° in	0	0
103° to 104° in	2	28.6
104° to 105° in	4	57.2
Over 105° in	1	14.3

Group II., Five to Ten Years. Forty-one Cases.

	CASES.	PER CENT.
102° or less in	1	2.3
102° to 103° in	3	6.9
103° to 104° in	10	23.2
104° to 105° in	23	53.5
Over 105° in	4	9.3

Group III., Ten to Fifteen Years. Ninety Cases.

	CASES.	PER CENT.
102° or less in	3	3
102° to 103° in	12	12
103° to 104° in	28	28
104° to 105° in	36	36
Over 105° in	11	11

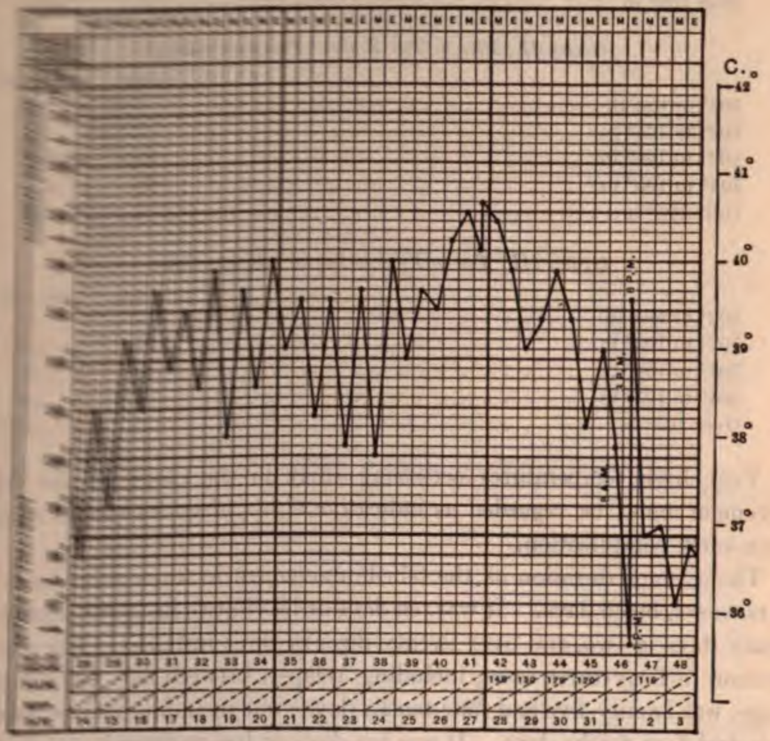
Very high temperatures occurring early in the course of the febrile movement must be regarded as indicative either of an intense infection or of an early complication.

The average duration of the febrile movement in seven cases under five years was eighteen days. It was ten days or less in two instances, from ten to twenty days in two, and over twenty days in three instances. The average duration in forty-three cases, including relapse, between five and ten years of age, was twenty-five and four-tenths days; not including relapse, twenty-three and four-tenths days. It was ten days or less in three instances, from ten to twenty days in fifteen, and over twenty days in twenty-five instances. The average duration, including relapse, in one hundred cases between ten and fifteen years, was twenty-three and one-tenth days; not including relapse, it was twenty-one and five-tenths days. It was ten days or less in six instances, from ten to twenty days in thirty-five instances, and over twenty days in fifty instances.

During the early convalescence the temperature frequently fluctuates in subnormal ranges, and is liable to be elevated by trifling causes, such as errors in diet, constipation, and emotional disturbances. These temporary accessions of fever, to which the term "recrudescence" has been applied, commonly last some hours, rarely more than a day or two, and are to be distinguished from true relapses.

The attack of enteric fever in a great majority of instances confers complete subsequent immunity. There are, however, many exceptions to this rule, so that a second or even a third attack has developed as a result of remote independent infection in the same individual. The escape in house-epidemics, or local outbreaks, of adults subjected to the same influences and using the same food and drinking-water as those who develop the disease, must be explained by an immunity acquired at some previous period of life, and in most instances during infancy or childhood.

CHART IV.



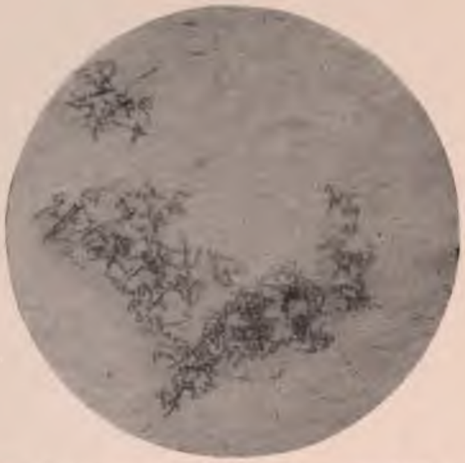
Critical defervescence upon the forty-sixth day of the attack and the nineteenth day of a single relapse; girl aged eight years. Seen in consultation with Dr. Lloyd.

The relapse is manifestly the result of a reinfection from within the body of the patient. It is, in truth, a repetition, with some modifications, of the primary attack. Why the primary attack confers lasting immunity in the greater number of individuals—an immunity that runs out in the course of some years in a few, and leaves the patient with the liability to prompt reinfection in the course of a few days or weeks in from three to fifteen per cent.—is a problem that awaits solution.

Relapses are probably not so common in childhood as in adult life. I have seen no second attack after an interval of months or years in a child.

In one hundred and fifty cases relapse occurred in no case under the fifth year; in three cases, between the fifth and tenth years; and in thirteen cases, between the tenth and fifteenth years. The total number of relapses was sixteen,—ten and six-tenths per cent. The average duration of the relapse in children between five and ten years of age was ten and five-tenths

FIG. 1.



The serum reaction. Large masses of agglutinated, motionless bacilli, separated by open spaces. (From a specimen prepared by Dr. J. C. Da Costa, Jr.)

days; in children between ten and fifteen years it was nine and five-tenths days. The average duration of the period of apyrexia between the defervescence of the primary attack and the beginning of the relapse was, in children between five and ten years of age, seven days; in the cases between ten and fifteen years, five and six-tenths days. No case in which relapse occurred terminated fatally.

Multiple relapses were not noted.

The blood in enteric fever has been studied in this country especially by Thayer and Cabot, whose results substantially agree.

The number of red corpuscles at the beginning of the fever is usually normal. During the first two weeks there is a slight progressive diminution, rapidly accelerated during the third week, and frequently continuing into convalescence, the lowest point being usually reached about the first week of convalescence.

Thayer finds that the degree of anæmia has, as a rule, a direct relation to the severity of the case, but grave anæmia may follow cases that appear to be mild. Severe post-typhoid anæmia constitutes a grave sequel of the attack.

The hæmoglobin diminishes with the red corpuscles and to a greater proportionate extent. Its return to the normal is less rapid. The leucocytes early in the attack are about normal, but their number gradually falls, reaching the minimum about the end of defervescence. As a rule, the diminution is slight and the return to normal is gradual. The absence of leucocytosis in a suspected case is of diagnostic value.

Cabot calls attention to the fact that in the beginning of the attack the number of leucocytes is often increased (11,000), and that there is a corresponding increase of the red cells, the ratio of red and white remaining normal. This he attributes to concentration of the blood as a consequence of restricted diet, diarrhœa, or sweating. Numerous observers have directed attention to the fact that there is an apparent increase in the corpuscular elements of the blood immediately after the cold bath. Cabot finds that in rare instances leucocytosis occurs when no complication can be discovered during life. He is inclined, however, to believe that "in all cases in which leucocytosis exists constantly, some complication is present, though unrecognized. The possibility of a secondary septic infection, an osteomyelitis or phlebitis of internal veins, cannot be excluded without further evidence." The effect of inflammatory complications, such as local or general peritonitis, phlebitis, otitis media, abscesses, pleurisy, and pneumonia, is almost invariably to produce a leucocytosis amounting to from 12,000 to 24,000. In patients greatly exhausted by the disease severe complications may occur without being accompanied by an increase in the number of leucocytes. Pyæmic and septicæmic processes are likewise attended with leucocytosis.

Widal in 1896 described a peculiar and characteristic effect of the blood-serum of enteric fever upon the typhoid bacilli in cultures. Upon the addition of one part of blood-serum to ten parts of culture-fluid containing active bacilli, the bacilli rapidly lose their motility and become agglutinated in clumps. Dr. Wyatt Johnston, of Montreal, discovered that this power of producing agglutination of the bacilli is possessed by dried blood as well as fresh. This power is inherent not only in the blood-serum of enteric fever patients, but also in the serum obtained by blistering or the fluid drawn from a serous cavity. The exposure of these fluids to a high temperature destroys the power of agglutination. Cultures not over twenty-four hours old should be employed,—preferably about fifteen hours old. The culture-medium may be agar or bouillon, and its reaction should be slightly alkaline. In the majority of cases this property of the blood-serum is developed in the first week, but in some cases, confirmed by post-mortem examination, the serum has apparently not developed the property until the second or third week, and in some few instances the reaction has been absent through the whole course of the attack. It would thus appear that even where all sources of error are eliminated a negative reaction is not absolute evidence of the absence of enteric fever. The blood-serum of individuals who have suffered from an attack of enteric fever retains the power of agglutination for a considerable time, a period measured in some of the recorded cases by many months.

The application of the serum-test in the case of the fœtus and the new-born has yielded varying results. Widal and Sicard obtained a positive reaction with the blood of the young born of a rabbit inoculated with typhoid bacilli six days previously. Griffith has reported a case in which a woman suffering from enteric fever gave birth at term to a well-developed child whose blood, obtained when it was seven weeks old, produced the characteristic reaction. This writer also cites a case of Chambrelent and Saint-Philippe in which the reaction was produced by the blood of the mother suffering from enteric fever and that of her child born in the eighth month of pregnancy. On the other hand, Etienne failed to find the reaction in the blood of a fœtus although that of the mother gave a positive reaction, and Charrier and Apert also failed to find the reaction in the blood of a three-months fœtus although the blood from the placenta gave it. These cases are all referred to in Griffith's article.[1]

A woman twenty-three years old was admitted to my ward in the German Hospital on the 23d of April, 1897, in the fifth month of her second pregnancy and on the eighth day of an attack of enteric fever. She aborted on the twelfth day. Her blood gave positive reaction with the serum-test, while no reaction followed the test made with that of the fœtus. At the end of April, 1897, I saw, in consultation with Dr. McLernon, a woman suffering from severe, well-characterized enteric fever, with excessively high temperature, who died at the end of the third week of the attack, two days after giving birth to a puny child, which, however, survived. The blood of this child when two weeks old produced the characteristic Widal reaction.

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[1] Medical News, May 15, 1897, p. 626.

I desire, in passing, to call attention to the fact that in several instances recently under my observation of pregnant women suffering from enteric fever in whom abortion has taken place, this accident has been attended by a rapid and permanent fall of the temperature to normal. In the case above referred to in the German Hospital the temperature fell upon the day of the abortion—the twelfth of the attack—in twenty-four hours from 103° to 98° F., and after fluctuations of less than two degrees remained normal, the patient making a good convalescence. In another case in the German Hospital, under the care of a colleague, a primipara, aged twenty-four, aborted in the fourth month of her pregnancy and on the twenty-fourth day of the attack. The temperature, which had ranged between 103° and 104° F., fell rapidly to 98° and did not again rise. This patient also made a good convalescence.

Intercurrent and Concurrent Diseases.—Enteric fever during its course fails to confer upon the patient an immunity from other acute infections. The ordinary infectious diseases of childhood may precede, coexist with, or follow it. Patients suffering from scarlet fever have developed enteric fever; on the other hand, scarlatina has occurred as an intercurrent affection during the course of enteric fever; and again, instances have been reported in which the eruptions of the two diseases have coexisted. Measles, pertussis, and varicella may also develop during enteric fever, either as intercurrent maladies or during convalescence. Diphtheria and pseudodiphtheria occur frequently as complications in the course of enteric fever in childhood. The exudate may develop upon the pharynx, the larynx, and the mucous surfaces of the genitalia. Noma or stomatitis appears to be more frequent in Europe than in this country.

The complications noted in one hundred and fifty cases were: abscess in four cases, bed-sore in one case, furunculosis in five cases, otitis media in four cases, parotitis in one case, pyæmia in one case, tonsillitis in two cases.

Diagnosis.—CLINICAL.—Well-characterized cases of enteric fever in children may after the fifth day of the attack usually be diagnosticated without difficulty. Prior to that time it is, however, in most instances impossible to make a positive diagnosis, but a provisional diagnosis of enteric fever may be based upon the concurrence of the following morbid phenomena,—namely, febrile movement with evening exacerbation, headache, epistaxis, diarrhœa, either spontaneous or following the administration of mild laxatives, and progressive asthenia. A direct diagnosis may be made if the febrile movement continues and is associated with diarrhœa, abdominal tenderness, enlarged spleen, slight tympany, and the development of the characteristic lenticular rose-spots. In obscure cases the coexistence of a house or local epidemic is of diagnostic importance.

The differential diagnosis from certain other acute febrile disorders may be attended with considerable difficulty. The diseases with which enteric fever may be confounded constitute two groups: first, those which it resembles in the first week of its course, and, second, those which it resembles in its more advanced stages.

The first group includes febricula, influenza, and certain of the exanthemata in the stage of onset.

Febricula and *simple continued fever* develop abruptly with a rapid rise of temperature, and usually terminate in the course of a very few days. Enlargement of the spleen and abdominal symptoms are commonly absent; rose-spots do not occur.

Influenza may resemble enteric fever in the first week of its course. The onset is apt to be more abrupt, the headache, rachialgia, and rheumatoid pains more severe, the early asthenia more marked, and, in the majority of instances, the process more rapid, than that of enteric fever, so that the patient suffering from influenza is usually convalescent before the period at which the eruption and splenic tumor show themselves in enteric fever. The epidemic or pandemic prevalence of influenza is of diagnostic importance. Resemblance is especially marked in the gastro-intestinal form of influenza. Influenza and enteric fever occasionally occur at about the same time in the same individual. I have observed cases in which patients have suffered from an attack of epidemic influenza which has run its course during the period of incubation of enteric fever, the symptoms of the latter affection supervening during the period of convalescence from influenza.

Measles.—The prominence of coryza and bronchial catarrh, the rash, and the epidemic prevalence of measles render the diagnosis comparatively easy.

Scarlet Fever.—The abrupt onset, the erythematous angina, vomiting, and the occurrence of the rash in the course of the second twenty-four hours, taken together, form a morbid complexus not seen in enteric fever.

The Variolous Diseases.—The mild chill, high temperature, intense headache, lumbar pains, and the appearance of the characteristic rash on the third day render the differential diagnosis between this group of acute infections and enteric fever in most cases an easy matter.

Typhus.—The onset of the fever is abrupt, stupor develops early, the defervescence is usually critical. The rash appears about the fourth day and persists until the end of the fever. It consists of red or dirty pink isolated spots, becoming petechial, and then not disappearing on pressure. They are numerous and widely distributed over the body and extremities. Between the spots is to be seen a peculiar subcuticular mottling.

The second group includes pneumonia, acute miliary tuberculosis, peritonitis, appendicitis, entero-colitis, pyæmia and septicæmia, and certain forms of malarial fever.

Septic and Central Pneumonias.—In the pulmonary form of enteric fever—pneumo-typhus—the differential diagnosis may not be possible until towards the end of the first or the beginning of the second week. There are forms of pneumonia, especially those in which the pulmonary lesion is at first central and gradually extends to the periphery of the lung, and the septic and so-called cerebral pneumonias,—not uncommon in childhood,—which present symptoms closely resembling those of enteric fever. In these varieties of pneumonia the onset is not attended by rigor, the rise of temperature is gradual, pleuritic pain is absent, and the physical signs of pneumonia may not be detected until the lesion has progressed to the periphery of the lung, a period often measured by several days.

Acute miliary tuberculosis presents in some instances a close resemblance to enteric fever. The history of previous chronic cough or pleurisy may point to the true nature of the attack. The temperature does not conform to type, dicrotism is absent, the abdomen is retracted, the *tache rouge* does not occur, and the bacteriological examination of the stools may show the presence of tubercle bacilli. The facies is different. In acute miliary tuberculosis the pulse is usually rapid and irregular, its frequency and rhythm showing constant and rapid fluctuations. The headache of enteric fever is usually dull, while that of tuberculous meningitis is acute. Vomiting is much less common in enteric fever than in tuberculous meningitis. We must note also the facts that convulsions are rare in enteric fever, and that the headache usually disappears during the course of the second week, upon the occurrence of delirium, while in tuberculous meningitis headache and delirium may alternate through the whole course of the sickness. In the cerebro-spinal form of enteric fever the onset of the attack is, however, abrupt, with intense headache, photophobia, painful retraction of the muscles of the back of the neck, twitching, delirium, and vomiting. The differential diagnosis between enteric fever and meningitis cannot, under these circumstances, be made clinically until about the end of the first week, when the appearance of abdominal symptoms, rose-spots, and splenic enlargement coincidently with the subsidence of the cerebro-spinal symptoms reveal the true nature of the illness.

Tubercular peritonitis may for a time closely resemble enteric fever. The abdominal pain and tenderness are more marked than in enteric fever, the fluctuations of temperature are irregular, the pulse is small, tense, frequent, irregular, and not dicrotic, the belly is doughy, and the presence of ascites may frequently be demonstrated.

Appendicitis may in rare instances simulate enteric fever. The course of the temperature, the absence of splenic enlargement, the localization of the abdominal phenomena in the right iliac region, the tenderness upon pressure at a point about midway between the anterior superior spinous process and the navel, slight rigidity of the abdominal muscles on the right side, and, in chronic or recurrent cases, the fact that the thickened appendix may be recognized upon palpation, are factors of importance in the differential diagnosis. The presence or absence of leucocytosis is of diagnostic value.

Entero-colitis can scarcely be confounded with enteric fever. The fever and constitutional disturbances are symptomatic of a local process. The spleen is not commonly enlarged, abdominal pain is conspicuous and severe, and rose-spots are absent.

Pyæmia and *septicæmia* from any cause may present a superficial resemblance to enteric fever. The history of the case, the absence of rose-spots, and the character of the temperature range should put the cautious physician upon his guard against a diagnostic error.

Trichinosis is attended with fever, vomiting, and diarrhœa. To these symptoms are also added the evidences of a diffuse myositis, together with chronic and general œdema. Epistaxis and splenic enlargement are rare; rose-spots do not occur.

Certain forms of malarial fever closely resemble enteric fever, especially when abdominal symptoms are present. Vomiting, diarrhœa, splenic enlargement, cerebral symptoms, and the condition known as the "typhoid state" may occur in both affections. The differential diagnosis will rest upon the presence of the eruption on the one hand and the discovery of malarial bodies upon the examination of the blood on the other. The absence of the characteristic rash of enteric fever is without positive diagnostic value in obscure cases, since in about twelve per cent. the eruption does not show itself during the whole course of the attack. The fact must not be overlooked that malarial and enteric fever may coexist in the same patient; but this association is rare, and cannot be regarded as establishing the existence of a definite nosological entity such as is indicated by the misleading term typho-malarial fever. In all doubtful cases an examination of the blood should be made. The discovery of the hæmatozoa of Laveran is conclusive evidence of the malarial nature of the disease.

The Diazo-Reaction.—The development of the characteristic color-changes in the urine and foam is of diagnostic value. In a series of twenty-nine cases studied in my ward in the Pennsylvania Hospital, the characteristic reaction was observed in eleven out of fourteen between the tenth and twenty-second days. In fifteen cases, in which the test was made later than the twenty-second day of the attack, the response did not occur. The value of this test is impaired by the fact that it occurs in other diseases, especially acute affections, as miliary tuberculosis, measles, scarlet fever, malarial fever, and pneumonia, which at their onset are liable to be confounded with enteric fever.

The examination of the blood is of great diagnostic value. As a rule, to which there are very rare and doubtful exceptions, leucocytosis does not occur in uncomplicated enteric fever. On the other hand, local inflammatory processes, as deep-seated abscesses, hepatic abscess, pyophlebitis, and appendicitis, are attended with leucocytosis. Leucocytosis also occurs in pyæmic and septicæmic processes.

The blood-count has a special diagnostic value early in the course of the attack in doubtful cases. The actual increase in the number of leucocytes due to blood-concentration may be deceptive, but in these cases the relative proportion between the white and the red corpuscles is more or less closely maintained. Inflammatory and septic complications render this test valueless.

The Serum-Test.—The discovery of Wyatt Johnston, of Montreal, that the fluid obtained by moistening the dried blood with water gives the reaction in a prompt and satisfactory manner, makes this method available for municipal laboratory diagnosis. In many of the larger cities arrangements have now been completed by which a specimen of the dried blood of a doubtful case may be examined in the laboratory of the board of health. This method has been practised with very satisfactory results in the Laboratory of Hygiene of the Bureau of Health of Philadelphia, under the administration of Dr. Abbott.

Directions for Taking the Blood.—Thoroughly cleanse the skin of the patient's finger-tip or lobe of the ear. After carefully drying, prick it with a needle previously sterilized by heating over a lamp or gas-flame and allowed to cool. One or more large drops of blood are taken up upon a piece of paper supplied for the purpose, allowed to dry, then properly folded and returned in an envelope provided for the purpose to the laboratory. A slip is to be filled out with the date, hour, name of the physician, his residence, the name of the patient, his age and residence, and statements indicating the following facts,—namely, whether or not it is the first specimen taken in the case; the day of the attack; whether or not the patient has previously suffered from typhoid fever, and, if so, at what date; a statement as to whether or not the following symptoms have been observed: diarrhœa, fever, enlarged spleen, tenderness in the ileo-cæcal region, rose-spots, delirium. If there be any suspicion as to the source of infection, it is to be stated, and there is a space upon the blank provided for general clinical diagnosis and remarks.

The following circumstances influencing the interpretation of the results obtained are set forth in connection with the blanks: "The test is not likely to yield positive results before the fourth or fifth day of an attack of typhoid fever. The condition of the blood causing the reaction persists for some time after convalescence, hence care must be taken to exclude the coincidence of a positive result due to a previous attack of typhoid fever. A negative result obtained with the serum of a case of suspected typhoid affords presumptive evidence against the diagnosis of typhoid fever, but this is only a probability, especially if the examination has been made in the first few days of the disease. The examination should then be repeated during the next few days. The presumption that a case in which a negative result has been obtained is not typhoid becomes correspondingly stronger as the stage of the disease becomes more advanced. (Widal.) In doubtful cases giving negative reaction, send further samples at intervals of two or three days so long as there is a possibility of typhoid fever. Please inform the laboratory of any discrepancy between result of test and the subsequent course of case."

The general statistics of the subject indicate that the reaction occurs in about ninety-five per cent. of cases of true enteric fever.

Elsner's Method of Diagnosis in Enteric Fever by Cultures.—This method may be employed for the detection of typhoid bacilli in water, articles of food, or the soil, but it is especially useful in their detection in the stools of patients, and therefore constitutes an important diagnostic procedure. The culture-medium employed is composed of Holt's acid potato gelatin with one per cent. of potassium iodide. A limited number of bacterial forms only will grow upon this medium. Among these are the bacterium coli commune and the typhoid bacillus. Of these the former grow much more rapidly than the latter. The colonies of typhoid bacilli after forty-eight hours present the appearance of small, glistening masses, which have been compared to minute drops of water. The subject has been carefully studied by Elsner, Lazarus, and Chantemesse, and by Richardson in this country. By this means the diagnosis has been made as early as the seventh day.

The Elsner culture method is an addition to the resources of the diagnostician, and may prove of great importance in certain doubtful cases. It has, however, a limited field of application. Richardson points out the fact that, as the examination of cases other than enteric fever fails to show the presence of the bacilli, this method constitutes a refutation of the hypothesis occasionally advanced that the bacillus typhosus is an organism wide-spread in nature and generally present in fecal discharges.

Prognosis and Mortality.—The prognosis of enteric fever in individual cases must be guarded, since in cases running an apparently favorable course death may occur towards the end of the third or during the fourth week from some unforeseen accident or complication. In individual cases the prognosis is unfavorable when an intense infection is manifested by the rapid development of grave symptoms,—intense pyrexia, cardiac asthenia, ataxic phenomena, and the occurrence of a number of cases in the same house or in the immediate locality. The prognosis becomes unfavorable also upon the development of intestinal symptoms of high grade, such as copious diarrhœa, abdominal pain, or great tympany. Uncontrollable vomiting is an unfavorable sign. Intestinal hemorrhage, perforation, and serious complications, such as ulcerative endocarditis, meningitis, pneumonia, pleural effusion, diphtheria, the development of the exanthemata as intercurrent or consecutive affections, render the prognosis grave.

The mortality in childhood is decidedly lower than in adult life. Enteric fever shows an extremely variable intensity in children, the severer cases, however, being the exception rather than the rule. Death rarely occurs in consequence of the intensity of the typhoid infection itself, but rather is the result of some accident or complication. The largest proportionate number of deaths occurs in the course of the third week. The mortality varies in different years and in different epidemics. The death-rate in twenty-six hundred and twenty-three cases collected from the reports of twelve different observers, almost all in hospital practice, by Holt, was five and four-tenths per cent. The more serious cases only are, as a rule, admitted to hospitals. In the one hundred and fifty cases above referred to, the total number of deaths was six, or four per cent. Holt regards the mortality, including all cases, as probably not exceeding three or four per cent. Earle some years ago observed twenty consecutive cases in two years without a death. Forchheimer in an epidemic in 1888 treated seventy cases without a single death. The death-rate among children in the first year is high, but from the end of the first year until the tenth year it is, I believe, much lower than hospital statistics indicate,—not, in all probability, exceeding one per cent. This opinion is based upon the frequency with which I have encountered very mild cases in private practice,—so mild that, except for the splenic enlargement, the appearance of the rose-spots, and a continued fever of ten or twelve days' duration, the nature of the malady could not have been suspected,—and the great infrequency of fatal cases in private practice at this period of life. On the other hand, in two hundred and eighty-four cases between the second and fifteenth years observed in the Boston City Hospital and at the West End Nursery and Infants' Hospital, Morse records a mortality of seventeen, or six per cent.

Treatment.—PROPHYLAXIS.—Enteric fever is in theory a preventable disease. The objects of prophylaxis are: first, to prevent any case from becoming a source of further infection, and, second, to rectify faulty sanitary arrangements which lead to the pollution of water by fecal discharges from any source. The fecal discharges and urine of every case should be at once thoroughly disinfected. For this purpose, solutions of chlorinated lime containing at least twenty-five per cent. of available chlorine of the strength of six ounces (one hundred and eighty grammes) to the gallon (four litres) of water or of carbolic acid solution five per cent. may be employed. The latter is less efficacious and requires a longer time. An excellent disinfectant for the stools is milk of lime prepared by slaking freshly burnt quicklime and stirring up the powder with twice its volume of water. This should be freshly prepared and added to the stool in equal bulk. When the bed-pan is to be used, it should contain about half a pint of the disinfectant solution, and immediately afterwards the same volume or more of the disinfectant should be poured into the pan, the whole being thoroughly mixed by agitating the vessel. The vessel should then stand for two or three hours before it is emptied into the privy or water-closet. Thoroughly disinfected stools may be emptied into the ordinary privy-vaults without danger, or, mixed with fresh earth, they may be buried in a trench at a distance from any source of water-supply. The important point is that they be thoroughly disinfected. In country places it is not uncommon to mix them with sawdust and burn them.

The mattress should be protected by a rubber cover. Articles of bedding and garments should be immediately changed when soiled. A tub should be in the sick-room as a receptacle for soiled garments, bed-linen, and towels. These articles should be immediately moistened with a five per cent. solution of carbolic acid and removed promptly to the laundry, where, without handling, they should be placed in a kettle and boiled for half an hour. They should then be washed with soft soap and thoroughly rinsed. If practicable, before being used again they should be exposed to the sun for some hours.

During epidemics, drinking-water and milk should be subjected to boiling, and uncooked vegetables should be avoided. The possibility of the conveyance of typhoid bacilli from the excreta of the patient to articles of food, particularly milk, by means of the house-fly is not to be overlooked. With a view to preventing this accident, receptacles containing the food must be carefully covered.

In the prophylaxis against enteric fever an abundant and pure water-supply is of first importance. Stress must be laid upon the natural history of the water. The ideal supply is that derived from mountain lakes or rivers in regions without population, or from artesian wells. It is impossible to protect streams that flow through populous valleys. Where it is necessary to procure the water-supply from such sources, the endemic and occasional epidemic prevalence of enteric fever can be prevented only by filtration upon a large scale and in accordance with scientific methods before distribution.

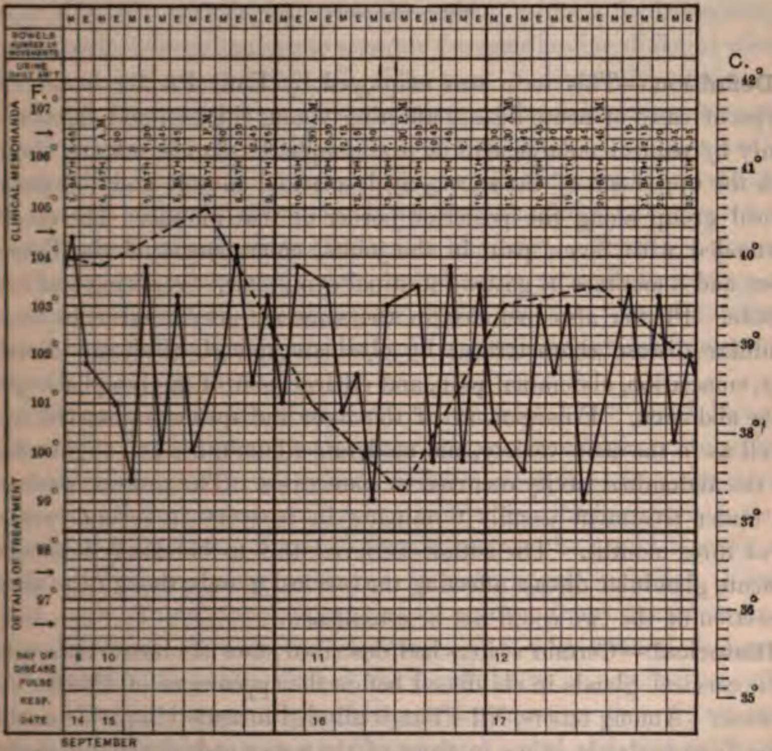
The Management of Individual Cases.—The result of the treatment in enteric fever is largely influenced by the attention given to the details of general management and the nursing of the patient. Strict rules in regard to rest in bed and the use of the bed-pan and urinal are not necessary in the case of children, nor can they always be enforced.

The diet must be liquid throughout, and should consist of milk, broths, raw eggs, etc. The free administration of fluid is important. The details of the diet in the case of young children must be determined in accordance with the age of the individual patient and the previous plan of feeding. In most instances no departure from the usual aliment is necessary beyond the strict avoidance of solid food.

In a large majority of children ill with enteric fever the treatment should be expectant. In some cases the symptoms demand special therapeutic measures, and the plan of treatment becomes expectant symptomatic. The general details of treatment have been set forth in my previous article in the Cyclopædia, and do not require repetition here.

Treatment by Systematic Cold Bathing.—I have employed with satisfaction the method of Brand in treating enteric fever in children in the wards of the Pennsylvania Hospital. This method with some modifications is exclusively practised in the children's wards of the Mary J. Drexel Home in connection with the German Hospital. The children are bathed whenever the temperature attains 102° F. The bath, at a temperature of 90° F. at the time of immersion, is gradually reduced by the addition of cold water to 75° F. The duration of the bath, if well borne, is ten minutes. If cyanosis, shivering, or distress occurs, it is shortened to five minutes. As a rule, the children are somewhat frightened, struggling and crying at first, but after the administration of a few baths they do not usually complain. The Sisters who do the nursing are very gentle and careful in the administration of the baths. In very young children the temperature of the bath at the time of immersion is made even higher than 90° F., and the child is placed upon a blanket and slowly lowered into the tub, so as to avoid the shock of quick immersion. These baths are repeated, as in the case of adults, every third hour, if the temperature reach 102° F. Whiskey is administered in iced water immediately before and after the bath, the quantity varying from twenty or thirty minims to a fluidrachm, according to the age of the patient and other conditions.

CHART V.



Effect of single baths upon the temperature. The dotted line shows the range of temperature as determined by morning and evening observations; the solid line the effect of the individual baths. Uncomplicated case; recovery; defervescence complete upon the eighteenth day of the attack; girl aged fourteen; Pennsylvania Hospital.

The temperature falls from two to three or four degrees and slowly rises, as in adults. After the patient is taken out of the bath and put to bed, reaction occurs promptly; nourishment is then administered, and the child usually falls into a gentle sleep. The number of baths administered during the course of the attack ranges from twenty to forty. In exceptional cases of long duration sixty or even eighty baths have been administered.

GLANDULAR FEVER.

By ALFRED STENGEL, M.D.

Definition.—This term was employed by Emil Pfeiffer in describing a series of cases of acute disease affecting young children and characterized mainly by painful enlargement of the lymphatic glands, notably those beneath the upper end of the sterno-cleido-mastoid muscle and the posterior cervical group along the posterior border of that muscle. The condition is attended with fever, pain in the joints, enlargement of the liver and spleen, and sometimes by gastro-intestinal symptoms,—vomiting and loss of appetite. Pfeiffer also included in the same category a subacute form of glandular disease characterized by obstinate though moderate diarrhœa, fever, emaciation, abdominal pain, and enlargement of the lymphatic glands of the abdomen. Enlargement of the liver and spleen is observed in this as well as in the acute variety, and moderate albuminuria and slight effusion into the abdominal cavity occurred in some cases. The process is subacute, and under treatment usually terminates in recovery in several weeks or two or three months. The author believed this to be closely related with the acute glandular disease affecting the cervical glands, though the absolute connection of the two could not be established.

Historical.—Certain writers had described cases of obscure enlargement of the cervical glands in childhood before the appearance of Pfeiffer's contribution. Among others, Nil Filatow alluded to acute idiopathic swellings of the lymph-glands lying in front of the upper end of the sterno-cleido-mastoid muscle and behind the angle of the lower jaw, attacking children from two to four years of age, though sometimes older children, and accompanied by high fever. These cases ran a course of from two to three weeks, the glands reaching their maximum size in three or four days and then gradually subsiding, and the fever continuing for five or ten days from the beginning of the disease. Subsequently the same author reviewed the disease described by Pfeiffer, and inclined to believe that this was the same condition described in his previous contributions. Other authors have described similar glandular enlargements more or less remotely related with infections (scarlatina, rubeola, and rubella) or unassociated with other conditions. After Pfeiffer's study was published, additional contributions were made by Heubner (in the same number of the journal in which

Pfeiffer's original article was published and as a commentary on the same), von Starck, Rauchfuss, Protassow, and others. (See Bibliography.) In America a remarkable series of ninety-five cases has been described by J. Park West, and smaller numbers of cases by Whitney, Hamill, and Roussell. The disease has been described in some of the recent text-books (Ashby and Wright, third edition), but has been ignored by other writers (Holt). A most excellent and comprehensive review, to which I am much indebted, is that of Hamill.

Pathology.—In his original paper, Pfeiffer stated that "without doubt this clinical complex, like so many other clinical pictures, will be found to embrace different pathological processes, and it remains for bacteriological and pathological investigations to separate the cases having different etiological or anatomical characteristics." This view undoubtedly expresses the fundamental facts regarding this condition. Glandular fever cannot be regarded in any other light than as a symptom-group of more or less definite character. It has not been proved, nor is the likelihood very great, that a specific general infection with cervical glandular enlargement constitutes the essential pathology of the cases described. There is a tendency, as in the case of other newly described diseases, to include various conditions, not certainly of similar nature, under the title "glandular fever," and there is, therefore, already difficulty in separating spurious from genuine instances.

Pfeiffer considered the probability of the cases he observed being instances of abortive infections, such as scarlatina or diphtheria. He excluded this possibility from want of definite evidence, as well as from the peculiar enlargement of the post-cervical group of glands, which ordinarily do not enlarge in such diseases. Some of the milder cases he would identify with the febricula of the older authors. Other writers, including Filatow, inclined more strongly to believe that glandular fever represents an ill-developed form of other known infections. It is quite natural that certain authors consider the probability of the disease being a peculiar type of influenza. The occurrence of glandular enlargement in this disease, sometimes of considerable extent, has been long known, and was particularly emphasized by Guitéras. In addition, it may be remarked that influenza is one of the diseases in which general systemic infection may occur without marked local lesions of the mucous membranes. These facts furnish some justification for the assumption that glandular fever may represent a type of this disease, altogether aside from the fact that glandular fever has sprung into notice and has been so abundantly observed during the recent years of prevalence of influenza. House-epidemicity is another feature common to the two conditions. Czajkowski claimed to have found influenza bacilli in the blood of some of his cases, but it must be stated that the recognition of these organisms in the blood is at least of questionable certainty, and other investigators have not confirmed the observation. It is impossible to decide from the existing facts whether the condition is an independent infection or not. It is likely, however, that it represents a form of cryptogenetic infection, the portal of entrance being more or less doubtful, though probably some part of the mucous surfaces lining the naso-pharyngeal or adjacent passages. The peculiar localization of the glandular disease (posterior to the sterno-cleido-mastoid muscle) is difficult to explain. Similar involvement does not occur in other infections attended with lesions in the throat or nose. This fact leads to the suspicion that, after all, glandular fever may be some well-known infection in which the primary lesion of the mucous membrane has a hidden seat, and therefore escapes detection. Certain authors believe it to be a form of intoxication dependent upon gastro-intestinal disorders, principally constipation, but this view is not sustained by sufficient evidence. The striking and early involvement of the cervical glands and the comparative insignificance of the involvement of the abdominal or other glands force us to the conclusion that there is some form of infection which gains entrance to the organism in the naso-pharyngeal or adjacent regions. The pathological anatomy is practically unknown. The general external appearance of the glands is that of an active lymphadenitis. In a few cases periadenitis, causing boggy infiltration of the surrounding tissues, has been observed (Comby); and some writers have described cases terminating in suppuration, though Pfeiffer claimed that this never occurs. There is great difficulty in reaching a decision on this point. We have not discovered any reports of cases in which the post-cervical glands were mainly involved as Pfeiffer described, and which terminated in suppuration.

Associated pathological conditions of the fauces and tonsils and of the kidneys, spleen, and liver will be alluded to in the discussion of the symptoms.

Etiology.—Glandular fever is essentially a condition of early childhood. Pfeiffer places the limits of age at from five to eight years. Others have held that it is almost entirely confined to the first two years of life, but undoubted cases have been observed up to the age of sixteen years. Hoerschelmann, Rembe, and Czajkowski each report one occurring in adult life. Sex does not enter decidedly into the etiology, though Neumann saw nineteen boys and but eight girls affected. In West's cases there were fifty-three boys and forty-three girls, while in fifty-nine other cases, collected by Hamill, there were thirty-seven boys and twenty-two girls. The disease is more common in the cold months of the year, and exposure to cold seemed directly responsible for a few of the reported cases.

Pre-existence of infectious diseases is occasionally noted: thus, Heubner and Moussous found it after measles; Rauchfuss and Korsakoff, after scarlet fever; Comby, after whooping-cough; and Hoerschelmann, after influenza. Coexistence of known infectious fevers is not admitted, as such cases are generally excluded from the category of glandular fever.

There is little doubt of the infectious nature of the disease, the occurrence of house-epidemics having been very frequently observed. This was particularly striking in the series of West, whose ninety-six cases were distributed among forty-three families. Little work has been done in a bacteriological way to determine the nature of the disease. Streptococci have been found in suppurative cases and in associated anginas. In addition, it may be noted that the occurrence of hemorrhagic nephritis in some measure renders the streptococcal nature of the disease likely. Reference has already been made to Czajkowski's discovery of influenza bacilli in the blood.

Symptoms.—The onset, according to Pfeiffer, is abrupt, and rarely, if ever, preceded by prodromes. Others, however, have described premonitory symptoms of various kinds, such as headache, lassitude, or gastro-intestinal disturbances. After these, or without warning, the child is seized with rigors or actual chills, and the temperature rapidly ascends to 102° F. or 103° F., or in severe cases to 105° F. At the same time great restlessness is noted, and the child complains of headache and pains in the limbs. Sometimes the initial symptom is vomiting, and very soon after the beginning of the fever the tongue is found to be furred. By the second day glandular enlargement is discovered, as a rule, upon one side, and in the course of the next two or three days this progresses and the glands on the opposite side become involved. At the same time slight coryza or redness of the fauces may occur, and in some instances there has been severe angina. The child now holds the head in a fixed position of torticollis and complains of pain on manipulation; the fever continues irregularly, and gastro-intestinal symptoms—constipation or occasionally diarrhœa—with enlargement of the spleen and liver are noted. After a duration of from four to seven days the active symptoms with the fever rapidly subside, and the glandular enlargement gradually disappears in from ten days to three or four weeks. The convalescence is protracted in many cases on account of the considerable anæmia.

The individual symptoms may now be considered in detail.

The temperature rapidly rises after the onset and reaches a height of 102° F., 103° F., or even 105° F. In the milder cases, which Pfeiffer noted as having a resemblance to the febricula of older literature, the temperature reaches the normal in a day or two, while in severe cases it continues for several days, and sometimes for a week or two, being exceedingly irregular and at times almost typically intermittent. The final defervescence is often quite abrupt, and critical discharges have been noted, especially by West. Sudden accessions of temperature may mark the involvement of new glands, and after the decline of the fever recrudescences may occur under the same circumstances.

In addition to the fever, the general restlessness, the rapid respirations and pulse, the headaches and pains in the limbs, together with enlargement of the spleen and liver, which occur in the great majority of cases, point to a general state of infection. Marked nervous symptoms are rarely observed.

The most characteristic symptoms are those connected with the glands. In Pfeiffer's description it was pointed out that swelling of the glands beneath the upper end of the sterno-cleido-mastoid muscle and along its posterior border at the same part is distinctive; but in some of the more recent contributions enlargement of the glands in the anterior part of the neck has been specially prominent. This was noticeable particularly in West's series. The enlargement begins upon one side and a day or two later involves the corresponding glands of the other side. The swelling increases rapidly until the individual glands reach the size of a nut, and the neck as a whole is considerably enlarged. Sometimes but two or three glands are involved, at other times a number, and any of the cervical groups—submaxillary, carotid, or post-cervical—may be involved. In a few cases implication of the salivary glands—submaxillary, sublingual, or parotid—has been noted, but this is unusual and doubtless accidental.

The swellings are tender on pressure. Occasionally there is periglandular infiltration, but the overlying skin is unaltered. Suppuration has been observed in a number of cases, though the diagnosis was not entirely clear in these. The usual termination is gradual resolution, occupying a period of one or two to several weeks. In a few instances the swelling did not subside for two, three, or even four months.

During the acute stages the child, as a rule, holds the head on one side and cries when attempts at rotation or movement towards the other side are made. Difficulty in swallowing, hoarseness, and dilatation of the pupil have also been noted, and are doubtless, in part, dependent on the glandular swelling.

General glandular enlargement may be noted. Most frequently the mesenteric groups are involved, and may be palpated through the abdominal walls. In these cases, too, abdominal pain is sometimes a decided symptom. Less frequently the axillary or inguinal glands are implicated, while occasionally vague discomfort in the chest, paroxysmal cough, and vomiting, together with dulness on percussion posteriorly between the scapulæ and near the roots of the lungs, suggest tumefaction of the tracheobronchial nodes.

In many, though not all, cases examination of the throat reveals inflammatory conditions, and coryza is frequently associated. The mucous membranes of the fauces and the tonsils are generally a little reddened, but severe cases have been observed in which decided angina and even pseudomembranous deposit have appeared. Catarrhal and suppurative otitis media have been observed in a number of cases.

The gastro-intestinal symptoms are rarely severe. In most instances there is constipation, and sometimes this is obstinate, but in some patients moderate diarrhœa takes its place, and at the final crisis, according to West, there may be copious evacuations of liquid and greenish character. Coating of the tongue, indigestion, and vomiting may be early symptoms and may continue for some time, or develop at any period in the disease.

The urine has the characters usually observed in febrile conditions, and sometimes contains a little albumin. Aside from this trivial albuminuria, however, genuine and intense nephritis may develop, as Heubner first pointed out. Previous to this observer, and even before Pfeiffer's contribution, Korsakoff had described cases of glandular enlargement in children convalescent from scarlatina in which nephritis frequently developed; but the exact nature of these cases is doubtful. Nephritis has, however, been observed in cases corresponding exactly with Pfeiffer's description. It develops early—from the second to the tenth day—in most instances, but occasionally not until some weeks have elapsed. The urine is scanty, dark in color, highly albuminous, and microscopically shows epithelial and blood-casts and free blood-cells,—in a word, the characters of a hemorrhagic nephritis. This subsides slowly, and rarely, if ever, occasions severe symptoms.

Occasionally skin-eruptions have been observed, but these are so unusual and various that they cannot be regarded as other than accidental rashes.

Diagnosis.—The disease, as it was described by Pfeiffer, is easily recognized, the peculiar enlargement of the posterior cervical glands, first upon the one side and then upon the other, with or without enlargement of other glands, being the distinguishing feature. When this glandular tumefaction speedily follows an acute fever of irregular type, without marked local disease of the mucous membranes of the throat and independent of recognizable infections, the diagnosis is well-nigh absolute.

Greater difficulty is experienced when the glands in the anterior part of the neck and beneath the jaw are principally involved. In such cases great care must be taken to exclude secondary adenitis of angina and similar disorders and the so-called idiopathic adenitis. More or less extensive epidemicity, the early appearance of pronounced fever and decided glandular enlargement, and the comparatively insignificant development and late appearance of angina, stomatitis, or other local conditions are the points that may serve to make the differential diagnosis. In spite of these distinctions, however, it will often be difficult to assert that the glandular enlargement is not secondary adenitis of ordinary form, unless, as in West's series, a large number of cases of quite uniform character are encountered, without association with other cases in which the glandular condition is manifestly only an attendant symptom.

Cases of this kind must be distinguished from the rare instances of mumps involving the submaxillary glands, or the even more unusual instances (Penzoldt, Alexander) in which the submaxillary and subauricular lymphatic enlargements have occurred without implication of the parotid. In epidemics of this kind, however, distinct communicability has been noted, and some of the cases proved to be typical parotitis.

Hamill calls attention to the cases of idiopathic glandular swelling observed in epidemic form at Hong-Kong by Cantlie and others. These cases were supposed to bear a possible relation to the mild type of bubonic plague, the pestis minor. It is impossible to determine whether such cases have any relation to glandular fever, but it seems at least unlikely.

Prognosis.—There have been but few deaths, and those that have been observed have resulted from unusual complications or occurred in somewhat doubtful cases. The duration of the disease may, as has already been noted, be very protracted, occupying months, extreme anæmia being the ordinary cause of delay in recovery. As a rule, recovery takes place in a week or a few weeks.

Treatment.—Little can be done in the treatment beyond careful nursing, administration of palliative remedies, and the use of tonics during convalescence. Rest and soft diet constitute the useful elements in the general care of the child. Relief of the tenderness and perhaps some aid to more speedy resolution are secured by application of ointments of iodine, mercury, and belladonna, with protection by cotton-wool. Small doses of calomel are recommended by West. High fever is to be controlled by hydrotherapic measures, and the system may be supported by quinine and similar tonics. Iron, cod-liver oil, hypophosphites, and arsenic prove useful during convalescence.

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DIPHTHERIA.

BY LENNOX BROWNE, F.R.C.S., ED.

PREFATORY.

[The writer of this article desires to signify his high appreciation of the article by the late Professor J. Lewis Smith in the original issue of this Cyclopædia, the completeness of which left little to be desired as to the nature or treatment of diphtheria at the date of its publication.

The present contribution must necessarily be limited in its scope, and therefore it has been thought well that, while not ignoring new points in clinical observation and in "classical treatment," as it has come to be called, this article should be chiefly occupied with a consideration of the subject as it has been expounded in the years that have elapsed since the establishment of its microbic origin; viewing it, in fact,—etiology, diagnosis, and treatment alike,—mainly by the increased light that has been afforded concurrently with the advancement of the science of bacteriology.]

DEFINITION.

DIPHTHERIA is an acute infectious disease due to the presence of a specific micro-organism (the Klebs-Loeffler bacillus).

1. Diphtheria is to be considered as *simple* or *pure* when this specific bacillus constitutes the *sole* organism, and it may then be termed **simple bacillary** diphtheria.

2. Diphtheria is to be considered *complex* or *impure* when the bacillus is *associated* with other micro-organisms, which are chiefly cocci, and the disease may in these circumstances be termed **complex** or **cocco-bacillary** diphtheria.

3. The term *pseudo* or *false diphtheria* represents an affection of the throat resembling diphtheria, but distinguished from either the simple or the complex variety by the *conspicuous absence* of the *specific bacillus.* It may thus be termed **non-bacillary** diphtheria.

[Of course in almost every case some form of bacillus is to be found, and the word "non-bacillary" applies only to the absence of the Klebs-Loeffler organism.]

4. The term *pseudo-diphtheria* has also been erroneously applied to an affection of the throat which may indeed be membranous and is characterized by the presence of a bacillus *identical* with the Klebs-Loeffler in every respect *save that of virulence.* A preferable term would be **non-virulent bacillary** diphtheria.

To avoid confusion, the terms "diphtheria" and "diphtherial" will alone be employed in this essay to designate the phenomena caused by the presence of the Klebs-Loeffler bacillus, whether present alone or in association with other micro-organisms.

The term "diphtheritic" should, indeed, have been considered as obsolete since the time that Trousseau showed that the inflammatory state is but mildly evidenced; and the same may be said of the word "diphtheroid," which is now superseded by the terms "pseudo-diphtherial" and "coccal."

It has been made an objection by certain critics that, having defined diphtheria as due to the presence of the Klebs-Loeffler bacillus and applied to it the term "bacillary diphtheria," it is illogical to use the term "non-bacillary diphtheria."

But it has to be remembered that the term "diphtheria" as defined by Bretonneau and Trousseau represents the skin, parchment, or leather-like membrane which is its chief clinical characteristic, and there is no logical reason why this useful clinical definition should not continue.

Thus, having agreed that true diphtheria is due to the Klebs-Loeffler bacillus, we must assume that all other forms of membranous sore throat are pseudo-diphtherial, not so much, or even at all, as regards their morphological, but as to their bacteriological features.

The term "pseudo-diphtheria," however, has, unfortunately, been applied to manifestations in the throat difficult to distinguish clinically or bacteriologically from the true disease. For this the writer ventured some time ago to employ the term "non-virulent" diphtheria, in the belief that the most frequent form of the pseudo-bacillus represents not a different microbe, but simply a true Klebs-Loeffler organism of exhausted toxicity.

Warrant for this may be found in the circumstance that in a recent communication to the Pathological Society of London it was stated that it is quite common to find the true bacilli and pseudo-bacilli in one and the same specimen.

While Roux is responsible for the statement that the so-called pseudo-bacillus is capable of producing membranous sore throats impossible of distinction clinically from the true disease, he has also contended that the only method of determining its specificity is by animal inoculation, a process impossible of practical adoption by the bedside practitioner.

On the other hand, it has been asserted by another eminent authority that "inoculation of animals is not an absolute test." It is certainly a less accurate one in the case of diphtheria than in that of tubercle, for example.

Whether such failure of inoculation be due to the non-virulent character of the bacillus or to non-receptivity of the animal experimented on has still to be proved; but the experience of the variability in the immunization of horses for the purposes of manufacturing antitoxic serum would appear to favor the latter suggestion as of at least occasional application.

We could, it is true, identify these non-bacillary varieties of membranous sore throats by a prefix such as "streptococcal," "staphylococcal," "diplococcal," etc., but this is very undesirable, considering how rare it is to find but one kind of coccal organism in any particular case and how very common to find two or more varieties of cocci equally distributed. Lastly, by making a distinction between the general morbid effects of different forms of membranous sore throat we avoid some clinical confusion, since we thereby emphasize that by certain clinical signs, exclusive of that of membrane, the experienced observer can often predict in advance of a culture whether a case is true (bacillary) or false (coccal) diphtheria. He may even forecast with some degree of certainty the preponderating coccus.

Coming to the question of the morbid products of diphtheria and the distinction between that of bacillary and that of non-bacillary origin, the term *toxæmia* will be employed in this article to represent the systemic effect of the specific bacillary products, especially those attacking portions of the nervous system, as distinguished from the *general infection* to be observed in cases of pseudo-membranous exudation of pharynx, larynx, or trachea due to micrococci, as the ordinary characteristic of any simple inflammatory or pyæmic process.

The writer has spoken of the so-called "pseudo-diphtheria" bacillus as being a true Klebs-Loeffler organism in an exhausted state as regards toxicity. But there are several other bacilli to be found in all forms of sore throat, whose only—or, at least, whose chief—resemblance to the true microbe of diphtheria is a similarity of form. These organisms will presently receive the brief consideration that they merit.

HISTORY.

The history of diphtheria given in the original article is so complete and exhaustive that it is only necessary to take it up afresh from the period when Klebs, of Zurich, discovered the specific bacillus of the disease.

It must, however, be noted that Oertel only just missed the priority of this discovery, for he, in common with Professor Laycock, of Edinburgh, and Hillier, of London, had believed the malady to be due to the presence of a micro-organism; and on all these points, as well as on the early history of the discoveries of Klebs and Loeffler and their contemporaries, Dr. J. Lewis Smith has written at length and with great interest.

It is generally stated that the bacillus of diphtheria was first described by Klebs, of Zurich, in 1883, but Sternberg has drawn attention to its far earlier discovery by the same observer, Klebs having published the fact at a medical congress held at Wiesbaden so far back as the year 1875. The circumstance appears to have attracted but little attention, notwithstanding that on examination of the original reference it is found that Klebs had announced at this date that he had not only detected the rod, but had also made an effort to cultivate it, and, so far as one can judge, successfully. To Klebs, therefore, the credit of having discovered this organism is undoubtedly due. But, since he never definitely announced that he had been able to obtain pure cultures of it, it must be said that he failed in establishing its causal relationship to the disease.

This was effected by Loeffler, who made pure cultures of the bacillus obtained from the throat-membrane, and communicated the disease to guinea-pigs and birds by inoculating them on the pharynx, larynx, and other parts with the products so obtained. On all these grounds, with the name of Klebs that of Loeffler will always be associated, because of his commendable and painstaking investigations of the specific taint-quality of the bacillus and of his reticence in the announcement of his conclusions until he was convinced of their accuracy. This he did in the year 1884.

The following are the grounds for Loeffler's belief as to the identity of the bacillus of diphtheria with the causation of the disease, to which are added a few corroborative experiments:

I. *It is found in all cases of undoubted diphtheria.* In 1888 D'Espine demonstrated the presence of the bacillus in fourteen cases of characteristic diphtheria, and during subsequent years numerous other observers, including Roux and Yersin and Von Hoffmann, have abundantly confirmed this statement. It has also been demonstrated that it can frequently be found in the throats of convalescent patients.

II. *The bacillus can be found* only *in cases of undoubted diphtheria.* D'Espine, when demonstrating the presence of the bacillus in the above-mentioned fourteen cases of true diphtheria, showed, further, that it was absent in twenty-four cases of mild sore throat which were clinically considered to be diphtheria. This observation has also been definitely confirmed.

III. *The inoculation of pure cultures induces the disease in animals.* With regard to this point there was at first some difficulty, for, although it was quite easy to produce a malady with typical throat-membrane, yet the fact that false membranes may be caused by scalding fluids, irritant poisons, or, as in one well-known case, by eau de Cologne, rendered the development of membranes an untrustworthy indication of the disease. The necessary proof, however, was supplied when Roux and Yersin showed that in a pigeon paralysis came on three weeks after the pharynx had been inoculated with the bacillus, and when, further, the membrane had quite disappeared and the bird was to all appearances well.

They also showed that in rabbits the paralysis usually commences in the posterior extremities[1] and then gradually extends over the whole body, causing death by paralysis of the heart or respiratory apparatus. Welch and Abbott confirmed these observations by similar experiments on kittens.

The discovery of the specific bacillus settled what threatened to be an endless controversy as to whether diphtheria is primarily a local or a general disease, in favor of the doctrine that it is in the first instance local. By recognition of its associated organisms we can distinguish the process-changes which are caused by the bacillus from the many complications which are no part of the disease when exhibited in its pure form. We thus find it possible to separate cases of true diphtheria from the various conditions which clinically resemble it, whereas before the causal relationship of the Klebs-Loeffler bacillus to diphtheria was known, observers were obliged to rely upon clinical features only.

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[1] The commencement of palsy in this situation in the rabbit corresponds with the now well-recognized fact that in the human subject the muscles first impaired are those which of necessity are in most constant use.

But the history of this portion of the subject would not be complete without recording the fact that there have always been a few opponents to the unqualified acceptance of this view that the Klebs and Loeffler organism is the absolute exciting factor of diphtheria, it being asserted by them to be the result and not the cause. This has recently been proclaimed with renewed vigor by those who do not admit the logical basis of serum-therapy, and it is therefore necessary to discuss the objection seriously.

Among the earliest writers who cast a doubt on the value of the discovery of Klebs and Loeffler was Oertel, but, with all respect, it may be suggested that his objections were rather of a sentimental than of a scientific nature; for, as already stated, to this eminent authority was due the chief credit of first divining the microbic origin of diphtheria. This he did in his classical article written for Von Ziemssen's Cyclopædia, in which he referred formally to the work of Klebs, who, with him, at first believed the organism to be a micrococcus; but when the bacillus was discovered it is not improbable that Oertel, having failed himself to prove its specificity, did not readily accept the conclusions of his *confrère.* Hence we fail to find any notice of Klebs's views in the American-English edition of Ziemssen's work, published in the year of their announcement; and, moreover, they seem to have been unknown to so ardent a bibliographist and reference-seeker as Morell Mackenzie, for no mention is made of them in his erudite essay on Diphtheria published nearly four years later. In fact, many writers—of whom Kanthack, in 1896, is the latest—even now date the discovery of the Klebs bacilli from 1883–84, the period at which Loeffler published his confirmatory observations.

Billroth and many other distinguished physicians of the time were equally sceptical with Oertel; but Hansemann is probably the most important authority who still remains unconverted as to the identity of the Klebs-Loeffler bacillus with the origin of diphtheria. His objections appear to mainly rest on:

(*a*) The fact of the occasional absence of the bacillus and its frequent association with other bacteria in cases of true diphtheria. It is, however, not true, as he is credited with stating, that this organism is to be found in only three-fourths of all cases of typical diphtheria; for this is contradicted by tables published by various observers in every country.

Opposed to this view is the contention of a physician to a London hospital—and he is not alone—that the fact that "certain cases were not tested for the presence of the bacillus diphtheriæ" does not "invalidate them for the purposes of comparison" with others which were, because "membranous sore throat and membranous laryngitis," which occur "without the bacillus diphtheriæ, are so uncommon that they may be neglected." Nevertheless, such an omission in the present state of our knowledge surely exhibits an enthusiasm of conviction quite as much to be deprecated in practice as the incredulity of the sceptic. It is, moreover, not justified by statistics.

(*b*) Hansemann next states that the bacillus is *never found alone.* Here he again exceeds the truth, although we must admit that in only about ten per cent. of all cases of true diphtheria is the bacillus found in pure cultivation. Nor is this to be wondered at, considering that Millar is credited with having identified some sixty or seventy pathogenic organisms in the normal mouth, while in many cases diphtheria is first exhibited as implanted on an already active coccal sore throat, as on that of scarlet fever.

Dr. S. Gee in his definition of the disease preceding an article on diphtheria written for Clifford Allbutt's "System of Medicine," 1896, says, "for practising physicians the main note of diphtheria is still found in the presence, not of special microbes and morbid poisons, but of false membranes upon certain mucous surfaces or upon abraded skin;" and, again, "even that diphtheria which is characterized by the presence of the aforesaid (Klebs-Loeffler) bacillus is so complicated by the action or co-operation of other morbific microbes that diphtheria is seldom or never due to a simple infection."

It is quite needless to contradict this statement otherwise than by quoting from Dr. Kanthack, who, writing on the etiology and pathology of the same disease and in the same volume, commences with the statement, "There are but few infective diseases the bacteriology of which has been so completely worked out as in the case of diphtheria." And he states, in another place, that "the Klebs-Loeffler bacillus is found in every case of diphtheria; and from the results of investigations made, we may say, all over the world, we must refuse to call any lesion diphtheria unless it is associated with that bacillus; conversely, any morbid process accompanied by this organism is diphtheria." And, lastly, "whatever view we take of the nature of the poison or of the products of intoxication, the laboratory has conclusively demonstrated that the Klebs-Loeffler bacillus is the cause —the specific cause—of diphtheria."

The writer need not say with which of these two somewhat contradictory opinions he agrees.

(*c*) Nor can any one with experience admit Hansemann's next objection, that the Klebs-Loeffler bacillus is to be found, except with great rarity, in diseases not identical, either clinically or anatomically, with diphtheria. For example, experiments—that is, animal experiments—are still wanting to show that the organism to be found in either pharyngitis or rhinitis fibrinosa is truly diphtherial in character, though it is a fact that whilst cases of faucial diphtheria in which the exudation extends to the nares represent a very malignant type of the disease, on the other hand, the deposit which sometimes, though rarely, commences in the nares is at least occasionally of a quite subacute and non-toxic character.

Contrast with these, however, those cases of traumatic membranous sore throat caused by scalding with hot water, strong acids, eau de Cologne, or other local irritants. In none of these do we find the presence of the diphtheria bacillus reported; but, even were it detected, it would still not be unreasonable to argue that a dormant bacillus had been stimulated into activity by trauma, seeing how considerable is the evidence in favor of the presence of these microbes in a quiescent state in throats apparently normal.

And yet again, in those instances where the Klebs-Loeffler bacillus has been detected in cases of phlegmon of the skin, we can be by no means sure that the diphtheria bacillus has not been the primary etiological factor of the inflammation, while the appearance of a lip or an ear excoriated and inflamed by diphtherial discharge bears a strong resemblance to phlegmon or erysipelas. It may be suggested that such cutaneous irritation may be the result of a coccal infection, to which it may be replied that one sees it in a higher degree as a result of diphtherial discharge than in the course of an ordinary purulent otorrhœa or endo-rhinitis.

Several American authors, of whom Stowell may alone be named, and a few others in this and other countries, have followed Hansemann, but none of them appear to have advanced any fresh arguments in support of their statements.

Of quite a different order are the investigations of Dr. Hermann M. Biggs, of New York. His experiments were made with a view to demonstrate that in the large majority of cases of acute angina and follicular (lacunar) tonsillitis, diphtherial bacilli are to be found, and that cultures made from such throats are capable of demonstrating in some instances that these diphtherial bacilli were fully virulent in character, as proved by tests on animals. There is, however, an omission in the chain of evidence,—namely, that the ages are not given,—and the fact that in the majority of the cases the patients were adults is of some importance. This is substantiated by the records of Corbett and Phillips, who, in relating experiments in support of Biggs's investigations, speak of the examination by themselves of eight persons working with them in the pathological laboratory at Cambridge (England), and again of twelve others, all of whom were clearly adults.

The writer long ago insisted on a fact which must appeal to the experience of most practitioners, that while membrane is the almost constant characteristic of acute inflammatory affections of the fauces and larynx in infant life, its absence is to be frequently noted in similar cases, whether diphtherial or not, when occurring in persons of full age.

Moreover, the following case may be quoted from his book on "Diphtheria and its Associates":

> A young Jewess, aged seventeen, who was apparently suffering from faucitis, and in whose throat there had been seen on only one occasion—and that by an exceptionally trained observer—a scrap of membrane on one pillar, not larger than a pin's head; there was, however, an entire absence of knee-jerk, and it was on this ground that diagnosis of diphtheria was made and the patient removed to an isolation hospital, where other diphtherial paralyses fully developed.

Thus, the presence of membrane is sometimes overlooked if the examination for it is not frequent and keen, just as a scarlatina rash may not be noted on account of its occasional evanescence.

The Times (London) for December 21, 1896, gives a brief report of a discussion at a meeting of the Metropolitan Asylums' Board Committee on discrepancies in diagnoses which had been made before and after admission into their hospitals. The statement was made that out of six hundred and fifty-one cases sent to the hospitals with a diagnosis of diphtheria from the attending doctor, five hundred and thirty-three proved on examination and from the subsequent course of the disease to be cases of tonsillitis.

But from the evidence before us it is by no means certain that the former diagnosis was erroneous, for out of forty-eight cases reported by Dr. Hermann M. Biggs, which had been first diagnosed as diphtheria, in twenty-five only did the disease follow the ordinary course of that malady.

One still meets doctors who declare themselves as unconvinced of the specific nature of the Klebs-Loeffler bacillus in view of an occasional clinical experience, thus ignoring the adage that "exceptions prove the rule." Dr. Biggs well states the position when he says "there still remains the firm belief with a large proportion of the profession that only such cases of acute angina are to be regarded as diphtheria as present at some time in their course more or less membrane. . . . Investigations have proved that the presence of membrane does not necessarily indicate the existence of diphtheria, as membranous inflammations of the throat may be produced by other organisms than the diphtheria bacillus." *Per contra*, the absence of membrane does not exclude a diagnosis of diphtheria, since a growing number of cases are on record in which the Klebs-Loeffler bacillus has been discovered with no evidence of membrane in the throat.

The most, indeed, that can be said for Biggs's cases is that the diphtheria, however virulent when inoculated into lower animals, was but slight in the persons examined, though this is but the natural history of the disease when it attacks the adult; a further deduction is that the bacillus is not uniformly active.

The after-history was given in forty-four out of forty-eight of these cases, and here we come to another fallacy in this and other similar records, —namely, that it is too often assumed that the absence of paralytic sequelæ demonstrates that the cases have not been truly diphtherial; this is, we know, by no means the case, for the proportion of paralyses following on diphtherial attacks at all ages is less than twenty per cent.

It is now ten years since Jacobi, of New York, expressed his conviction that many of the cases of so-called "follicular tonsillitis" are of diphtheritic origin and nature, and claimed also that the "herpetic angina" of the French was in many cases nothing but diphtheria. At that time we thought this view was exaggerated, and it is still unrealized by the older school of physicians and by not a few of the new; nevertheless, in the light of recent information, and especially that furnished by Biggs and Koplik, it must in fairness be admitted that the circumstance is of more than exceptional occurrence.

But due credit having been given to these observations, it is not unimportant to suggest that the diagnostic skill of the clinical observer must constitute a factor of some weight in the acceptance of such as representing an ordinary experience; for of one hundred and fifty-two consecutive cases of tonsillitis occurring in the practice of the Central London Throat, Nose, and Ear Hospital (England), which have been bacteriologically examined in recent months,—though not for the purpose of this article,—in no single instance was the Klebs-Loeffler bacillus discovered.

That these were not cursory examinations is proved by the fact that so many as twenty-four different micro-organisms were differentiated, of which those most frequently present were the following:

Streptococci of various kinds in ninety-six cases.
Staphylococci of various kinds in fifty-four cases.
Diplococci in forty-three cases.
Bacillus termo of Vignal in twenty-one cases.
Bacillus coli in fifteen cases.

In any case, it may again be insisted that the clinical course of the disease in such circumstances is, for the most part, a mild one, and it is therefore probable that it may be explained by Sir Richard Thorne-Thorne's theory of the "progressive infectiousness" of diphtheria, as of other specific fevers, and that the bacillus of diphtheria, which we know too often lies dormant in the oral cavities of apparently healthy persons, is suddenly stimulated into modified activity by an inflammatory process of a purely coccal origin.

The practical deduction is that during an epidemic of diphtheria it is imperative to make a bacteriological examination of every sore throat, however apparently mild its objective features may be.

Having, then, shown that the bacillus of diphtheria may be found in inflamed throats of the adult without producing any serious results, it has to be noted that, on the other hand, when diphtheria follows on scarlet fever, measles, or other exanthemata, more especially in the young, the implanted malady almost invariably assumes a highly malignant type. Dr. Albert Wilson appears to believe that a coccal infection generally precedes that of the specific bacillus,—an opinion confirmed by Dr. William Hunter, who says that "diphtheria is the result of a mixed infection."

Evidence, however, is wanting to establish this as being a regular sequence of events in all cases and to justify the assertion made by Terry, that "examination by culture or by the microscope, without other confirmatory tests, is almost, if not quite, useless, or, at any rate, very misleading."

In this connection it is useful to quote a case recorded by Gouguenheim, whose reputation, both clinical and scientific, places the accuracy of his observations beyond cavil. This record, the interest of which merits careful perusal in detail, is briefly that of

An adult, about forty-five years of age, sent to hospital with a diagnosis of diphtheria for the purpose of having tracheotomy performed on account of sudden dyspnœa.

The left side of the soft palate and left tonsil were covered with an extensive, continuous, grayish-white exudation, presenting the aspect of a true diphtherial membrane. The rest of the throat was very red. Laryngoscopic inspection revealed a certain quantity of muco-purulent liquid, but the mucous membrane—slightly tumefied and injected—did not present any false membrane on its surface.

Bacteriological examination of the membranes and of the liquids in the larynx failed to reveal the bacillus of diphtheria in any of the cultures made. Nothing was encountered but chains of streptococci absolutely pure and without admixture.

The dyspnœa soon disappeared completely and the voice became normal; nevertheless, the patient did not improve and the fever continued constant; complications ensued in connection with the pleura and left lung. The urine contained a notable quantity of albumin.

At the moment when the man seemed entering on convalescence, the fever and albumin disappearing little by little, he was suddenly attacked and in an instant carried off by cardiac failure, which the autopsy showed to be due to a purulent pericarditis, the existence of which had not been suspected during life. There was also pulmonary congestion, from apparent vestiges of the purulent pleura of the left side and of pus in the articulation of the left clavicle.

Here was a case in which the clinical course of the disease, especially the termination, resembled that of true diphtheria. But for us the distinctive diagnostic point is the early manifestation of suppuration and its extension to the serous cavities, and this should have tended to bring into harmony the bacteriological and the clinical evidences. A very good case is No. 19 in "Diphtheria and its Associates," in which not only was the writer able to make a diagnosis of its pseudo-diphtherial character by clinical observations alone, but on the same data he was so fortunate as to forecast the bacteriological features of the case,—namely, staphylococcal. (See Figs. 15 and 16.) The opinion was based mainly on the following circumstances: the age of the patient (forty), great œdema of the soft palate and uvula, and an excess of viscid mucus both in the post-nasal space and on the palate, where it formed ropy bands from uvula to tonsils. Lastly, the portions of membrane which were coughed up were much more transparent and friable than would have been the case in true diphtheria.

But a still more striking example, because more difficult of discrimination during life, is another also mentioned in the same work (page 181):

It was that of a male child, aged three years, who was admitted to hospital on February 17, 1895, having suffered from cough and sore throat since the 14th. No fact could be elicited pointing to the probability of a scarlet-fever infection. Albumin was present, but at no time was there more than a trace.

On admission, membrane was seen on both tonsils and soft palate. Croupy cough and marked ensiform retraction suggested laryngoscopic examination, when membrane of a yellowish-gray color and of pultaceous consistence was seen in the larynx.

Bacteriological examination from cultures made on admission and afterwards frequently repeated showed strepto- and staphylococci, but *no* diphtheria bacilli. Death occurred on March 13, and necropsy revealed the lower lobes of lungs airless, especially on the right side; evidences of broncho-pneumonia in the middle lobes; a soft membranous exudation extending from the lower surface of the epiglottis along the larynx and trachea, so as to make an almost complete pulpy cast of this tract. Similar exudation was seen to extend into the left bronchus.

This was clearly a case of coccal infection, or, as we here call it, non-bacillary diphtheria, which in its extension to the larynx constituted a non-bacillary croup. One more example, in which the clinical diagnosis would certainly have been that of diphtheria in pre-bacteriological days, occurred quite recently in the writer's practice:

It was that of a single lady, aged forty-six, who on February 24, 1897, was seized with a rigor and was found to have a temperature of 103° F., which under antipyretics quickly declined to 100°. She complained of pain in her throat, and shortly after developed swelling in the submaxillary region.

She was seen in consultation on February 28. On the whole of the fauces and soft palate was a scattered exudation of thin consistence and dirty grayish color, not unlike that of the early manifestation of secondary syphilis. With a swab the membrane was easily removed, with one or two bleeding points where it was attached to the tonsils, but not from any part of the velum.

The larynx was only slightly congested and exhibited no sign of exudation. A day later the membrane was thicker and was seen on the *posterior surface of the uvula*. The breath was very foul.

The chief symptom was a tendency to pulmonary congestion, and there was none of the characteristic depression of diphtheria. The bacteriological report was to the effect that there was an entire absence of the Klebs-Loeffler bacillus, but an abundant growth of diplococci in almost pure culture.

The after-history of the case confirmed the bedside diagnosis of a diplococcal tonsillitis.

A servant in the same house, who was attacked, a day before the mistress, with extreme nausea and faintness, had been certified to a fever hospital with a diagnosis of diphtheria; but the bacteriological examination, made in the course of the disease, equally established her sore throat to be partially of diplococcal origin, although streptococci were also present. Another patient, a few doors off in the same street, was attacked about the same time, and in her case Klebs-Loeffler bacilli were found

From all that has been stated on this head it appears safe to assert, from figures at our command, that the proportion of cases of membranous sore throat of high grade occurring in the fauces which are not diphtherial (bacteriologically) does not exceed from ten to fifteen per cent. of the whole number of cases recorded, this being the proportion assigned by Billings when the larynx is attacked. A point worthy of note is that diphtherial throats are almost always dry throats, and that purulent sore throats and those accompanied by much expectoration are almost always of coccal origin; the same observation applies to manifestations in the larynx.

Membranous laryngitis or **true croup** has been attributed by many to an organism of a non-bacillary nature. Dieulafoy, for example, has stated that it is of the nature of a diplococcus or of a small grouped staphylococcus,—the Brisou coccus of Martin and Roux; Klein and Councilman give preferential selection to the streptococcus; but at least one case has come under our notice which could confer equal prominence on the staphylococcus as the organism of pseudo-diphtherial laryngitis. In fact, however, there is no reason, logical or bacteriological, why in the case of an extension of the membrane from the fauces to the larynx the coccus should be assumed to change its type. And although Sidney Martin in 1892 expressed the opinion that "there is no evidence at present of any disease other than diphtheria which can produce a false membrane in the larynx," and Ruault, writing in the same year, gave an almost unqualified adhesion to Martin's view, Billings (1894) found that "of two hundred and eighty-six cases in which the membrane was confined to the larynx and bronchi, eighty per cent. only proved to be true diphtheria, fourteen per cent. being undoubtedly not diphtheria." This is probably the proportion; at least it is that represented in the experience of the writer and in that of the few other English practitioners who have always contended against the correctness of the position, so strongly occupied in the Report of the Royal Medico-Chirurgical Committee (1877), that all membranous laryngitis is diphtherial. Thus, here again we find that bacteriology has confirmed the conclusions of general practitioners and clinicians against the dogmas of the schools.

THE KLEBS-LOEFFLER BACILLUS.

The specific micro-organism of diphtheria is for the most part represented as a non-mobile, straight or slightly curved rod, generally exhibiting some clubbing at one or both ends. In addition to the slight single curving, it has sometimes a tendency towards a double curve, like the old-fashioned letter *ſ*, but this may possibly be due to the refractive property of the organism, the ends staining more deeply than the other parts.

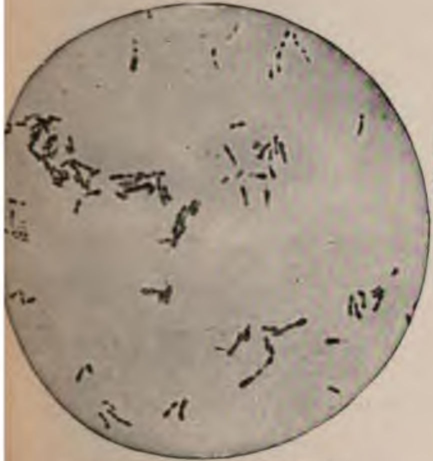
Each bacillus is from one and five-tenths to six micromillimetres in length and five-tenths to eight-tenths of a micromillimetre in thickness; thus, it is both longer and broader than the bacillus of tubercle. Great variation in morphology is exhibited; thus, the outline is not uniformly cylindrical, a bulging being often seen at the ends, while when stained a certain granular or segmented appearance is noticed in the interior. The mistake was at first made of considering that this was an evidence that the organism contained spores, but this view is no longer held. Sometimes patches of the protoplasm appear as if shrinking from the cell-wall (Fig. 3), leaving a more or less regularly uncolored or but lightly colored space at the periphery. The bacillus stains well by Gram's method and with Loeffler's methylene blue.

The organisms occur either singly or in pairs, frequently more or less parallel to each other (Fig. 1), or at an obtuse angle, like a circumflex accent (∧), seldom or never end to end or chain-like (Fig. 2). They are sometimes arranged in small groups of three or four, often in such a manner as to bear close resemblance to letters of the alphabet, such as V, M, N, X, Y, or other forms of wedge-shaped characters.

Several varieties of the bacilli have been described according to their size,—namely, the *long*, the *short*, and the *medium*. They have been also variously described, according to their diameter, as *thick* and *thin*.

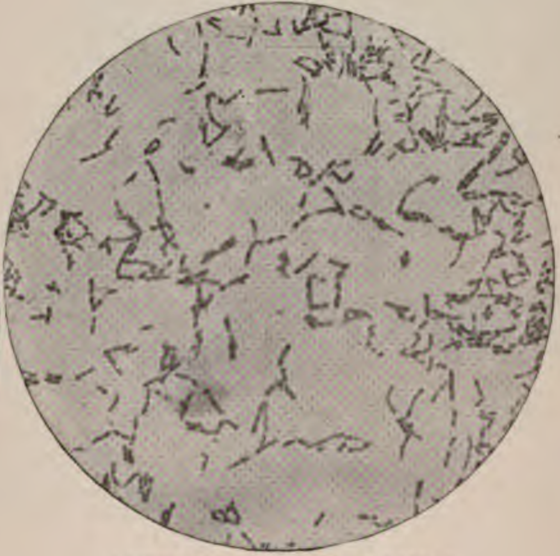
The differences in length and calibre probably represent various stages or varying richness in the growth, for at present definite evidence is wanting to show that variations in the size of the bacilli, either in length or in thickness, represent differences in the degree of their virulence.

Fig. 1.



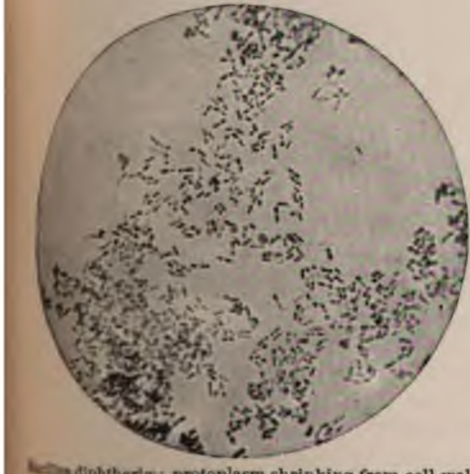
Bacillus diphtheriæ; singly and in pairs.
(For clinical appearance see Fig. 12.)

Fig. 2.



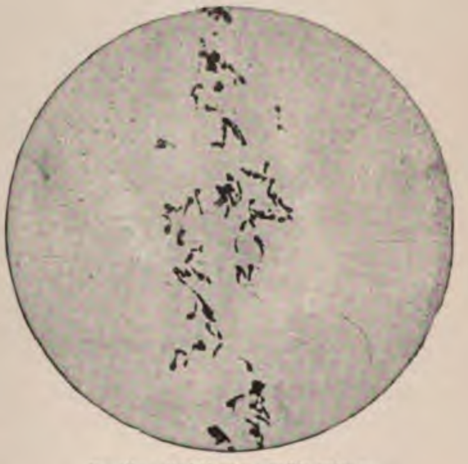
Bacillus diphtheriæ; arranged in chains.

Fig. 3.



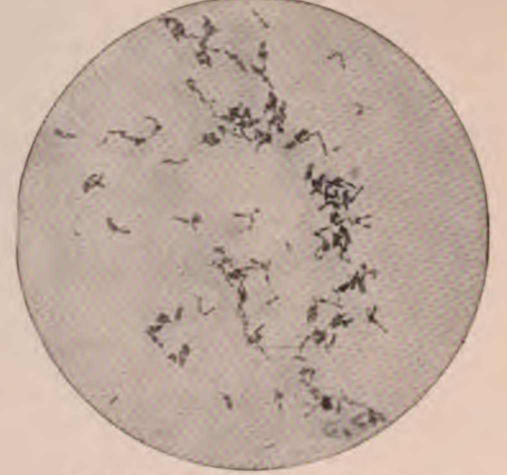
Bacillus diphtheriæ; protoplasm shrinking from cell-wall.

Fig. 4.



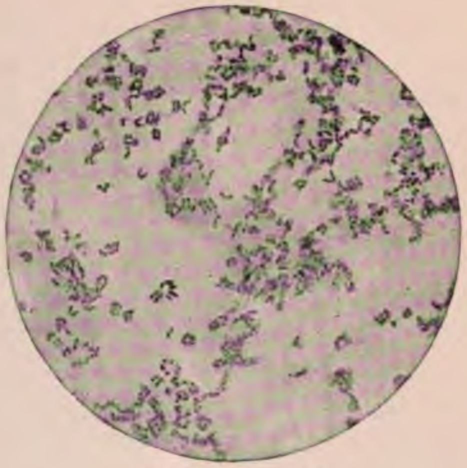
Bacillus diphtheriæ; milk culture.

FIG. 5.



Pseudo or non-virulent diphtheria bacillus.
(From Roux's laboratory.)

FIG. 6.



Non-virulent bacillus.
(From case of author.)

On this point opinions have been numerous and confusing, though the tendency is certainly towards attributing greater virulence to the long variety, since the bacilli examined during convalescence are almost invariably of the shorter kind. Roux and Yersin do not seem to attach much importance to the slight differences they have recognized in the morphological aspects of the organism. Moreover, it would appear that the size of the bacilli and also their method of staining depend somewhat on the medium in which they are cultivated, cultures on glycerin-agar developing less fully and staining less characteristically than those grown on blood-serum. Fig. 4, representing a culture on milk, from a specimen by Professor Klein, illustrates a still more striking difference in form,—so different, indeed, that it would be well, if agreement were more general, to publish only cultures made on one particular medium. Of these, for all purposes of education and comparison, blood-serum is undoubtedly the best.

Kanthack, in his latest contribution to the literature of diphtheria, supports this protest and states the position very well, as follows:

"It is not advisable to be too strict in this division of the bacilli into types according to their size, because, although we may find growths in which all the bacilli are long and clubbed or short and straight, yet they do frequently vary in size and shape in the same culture, and on transferring a long form from tube to tube it often changes in appearance from the long to the short form, and conversely. . . . It has also been stated that cases presenting the long form are more virulent than those presenting the short form. This, however, is misleading and erroneous. After an extensive examination I can say confidently that it is futile to base a prognosis on the type of organism present. Some of the worst cases that I have seen were associated with the short variety exclusively, while many less serious cases exhibited long forms only. Again, I have found colonies of the long and the short form side by side on the same agar-agar surface."

THE ATTENUATED OR NON-VIRULENT BACILLUS.

This is the writer's suggested *synonym* for the so-called *pseudo-diphtheria bacillus*, and the consideration of this microbe has been reserved to the last; it is difficult of distinction from the genuine organism of diphtheria, since in its method of growth, the formation of its colonies, and its microscopical appearance it is absolutely identical with the Klebs-Loeffler. Nor does it readily lose any of these characteristics on further cultivation.

The similarity of the two microbes consists not only in the appearance of the bacilli separately, but in that fantastic alphabet arrangement to which allusion has been made as a special feature in the identification of the true organism. A point of fine distinction is that in the majority of cases the attenuated bacillus exhibits polar staining in a less degree than when the organism is in full virulence. Appended (Fig. 5) is a photo-micrograph of a beautiful example of this "pseudo-diphtheria bacillus," as it has, in these days of exactitude, been so inappropriately named.

The specimen represents a pure culture, but the original contained a few staphylococci. If one compares it with others of admitted virulence, it is impossible to detect the slightest difference between the two in form or arrangement, and any bacteriologist obtaining such a result would not hesitate to give an opinion as to the true diphtherial character of the case from which it was taken. In point of fact, the only method of distinguishing this "pseudo-diphtheria bacillus" from that of the veritable Klebs-Loeffler organism is by an experimental observation of its non-toxic effects when introduced into the bodies of rabbits or guinea-pigs,—that is to say, that although so similar in form, and responsible even for the production of a membranous inflammation of the throat, it is non-virulent in the sense that it is incapable of producing toxæmia either in the person of the individual in whom it is found or by inoculation of lower animals.

It is gratifying to record that these views, first expressed by the writer early in 1895, are confirmed by Kanthack, who in his recent article says, "Bacilli do occur in healthy and non-diphtheritic sore throats which closely resemble the Klebs-Loeffler organism, but yet are not entitled to this name; . . . these have been named 'pseudo-diphtheria bacilli.' The name 'pseudo-diphtheria bacillus' apparently includes several varieties and species, and must be used with caution."

Corbett and Phillips also state (December, 1896) that "the microscope will not help us to distinguish between these two organisms, and, indeed, they differ only in virulence." They "believe that the question must at present remain an open one, and in the absence of more definite evidence than we now possess the views held by individual experimenters are of little importance."

PSEUDO-DIPHTHERIA BACILLI.

There are other micro-organisms to be found in the throat whose only resemblance to the Klebs-Loeffler consists in a greater or less degree of similarity of form, and which may, therefore, be classed as actually pseudo-diphtherial. These are:

Hoffmann's bacillus, the Xerosis bacillus, and several other varieties which have been described by Vignal. A very few words will suffice for their description.

Hoffmann's bacillus is found in normal healthy throats, and has been portrayed as a short, wedge-shaped rod, usually observed in pairs with the bases in apposition. In old cultivations clubbed forms are found which somewhat resemble those of true diphtheria. They stain uniformly, and do not show the same variations in morphology as the Klebs-Loeffler when cultivated on different media. The appearance of the colonies is almost absolutely identical with that of the diphtheria bacillus, but the organism is not pathogenic to animals. It only remains to state that the Hoffmann organism is included by Roux and Yersin under pseudo-diphtheria bacilli which have lost their virulence.

The *Xerosis bacillus* also resembles that of true diphtheria. It was described minutely by Eisenburg (1892), but does not seem to have merited much attention, for at the end of 1895, Eyre, at the Pathological Society, read a paper on "Xerosis Bacillus," defining it as Neisser's and commenting on its close resemblance to the Klebs-Loeffler organism. It is found in the conjunctival secretions in certain forms of conjunctivitis, accompanied by a hypersecretion of the Meibomian glands. It differs from the Klebs-Loeffler bacillus in being more curved, in being often grouped in larger masses, and in its frequent arrangement in rosettes. It grows only at the body temperature and cannot be cultivated on gelatin; it also is non-pathogenic to animals. This comparatively innocent organism is probably responsible for some at least of those cases of membranous exudation observed on the eyelids, which have been diagnosed as diphtherial; but, so far as the writer is aware, paralyses following such cases of ocular diphtheria are almost, if not entirely, unknown. Hermann Cohn, of Breslau, recommends as most efficacious for this form of conjunctivitis hourly pencillings with a five per cent. solution of benzoate of sodium, and the recognized inert effect of this salt on diphtherial false membrane when present in its more ordinary sites gives the *coup de grâce* to the matter.

The *Bacillus striatus albus* resembles the Klebs-Loeffler organism both in size and in manner of staining, but differs from it in the fact that it grows on gelatin at the ordinary temperature. The same may be said of the *Bacillus striatus flavus*, another variety of the same organism. The *Bacillus ulna* of Vignal is rather larger than the *Bacillus diphtheriæ*. It takes the stain evenly. It is straight, with rounded ends, and occurs in pairs. It liquefies gelatin and blood-serum, and the cultures have an odor of putrefaction.

The *Bacillus buccalis minutus* (*bacillus G.* of Vignal) shows polar staining. It is very short, as broad as it is long. It is non-motile, liquefies gelatin, and the colonies are greenish yellow.

The foregoing brief descriptions will demonstrate that there is no ground for confounding any of these bacilli with an active Klebs-Loeffler organism, or even with the microbe generally called "the pseudo-diphtheria bacillus," but which, it is necessary to repeat, is, as understood in this article, a true Klebs-Loeffler exhausted of its virulence.

Different in culture, morphology, and pathogenesis, both to man and animals, it is clear that none of them is entitled to be called "pseudo-diphtherial" at all; and it is a pity that the student should have had so much attention drawn to them in bacteriological text-books.

COMPLEX OR COCCO-BACILLARY DIPHTHERIA.

In our definition we have stated that diphtheria is to be considered *pure* or simple when the Klebs-Loeffler bacillus is the sole organism present in the cultivations, but when associated with other micro-organisms—*e.g.*, cocci—it may be termed complex, *impure*, or cocco-bacillary diphtheria.

The micrococci which are of importance from a clinical point of view, as associated with the specific bacillus, are of three kinds:

(1) Diplococci,—those arranged in pairs (Figs. 7 and 8).

(2) Staphylococci,—those arranged in groups (Fig. 16).

(3) Streptococci,—those arranged in chains (Figs. 7, 8, 10, and 11).

The author's personal experience is that those cases in which *diplococci* are predominant or are associated with other varieties of cocci are "dirty" in character, and are slow to clear away the false membrane.

He has also seen more than one instance of staphylococcal throat as the result of influenza, accompanied by high temperature and a tendency to, but not always with full development of, pulmonary complications, in which the *diplococcus of pneumonia* has been found in association.

The so-called "*Brisou*" coccus, to which some importance was for a time attached,—described by Louis Martin, a colleague of Roux,—is probably a mild form of staphylococcal grouping. It has really no significance clinically, and neither when present alone nor when associated with the bacillus of diphtheria has it any influence on either the course or the severity of the disease.

Streptococci, in relation to our present subject, when found alone generally represent a sore throat associated with the exanthemata, of which scarlet fever is the most usual, for this organism is almost invariably to be found in the throats of scarlatinal patients; not that it is meant to imply that this coccus is the specific micro-organism of scarlet fever, for that at present is undiscovered.

Roux is of opinion that the presence of other micro-organisms with the diphtheria bacillus, especially streptococci, adds to the gravity of the malady. Kanthack, however, states that "personal observation leads him to believe that the presence of streptococci in itself does not influence the prognosis,—indeed, they are rarely found absent, whether the cases be mild or serious;" while others do not consider "the association with streptococci as evidence of unfavorable import." The writer is inclined to think that there is not much to be said on either side, for his own experience, while not confirming Roux's conclusions as to the universality of ill omen of mixed infections from a prognostic point of view, has convinced him that no case of diphtheria can be in any way the better in that it is mixed, because to its own specific toxæmia are added the dangers, both inflammatory and pyæmic, which are characteristic of coccal infections.

Most authorities agree that streptococci are not only the most common of the associated organisms, but that they are also of the gravest omen, being responsible for infectious complications often of a most malignant character. The writer's experience shows that streptococci when associated with the Klebs-Loeffler bacillus in the *conglomerate*—or, as he has called it, the *battalion*—arrangement, in which the cocci are grouped in serried ranks, parallelograms, triangles, or in double files, constitute the most virulent form of complex diphtheria. This variety is especially to be found in those at-

Fig. 7.

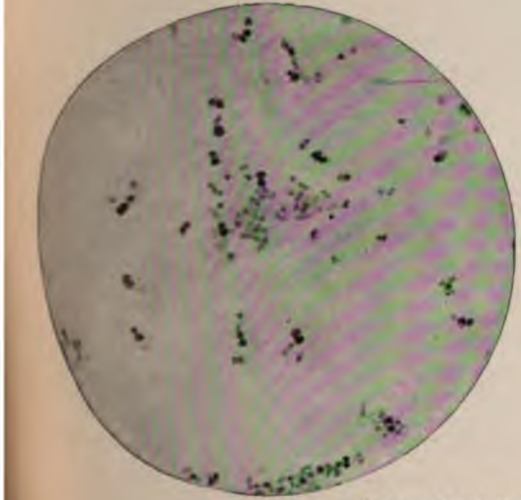
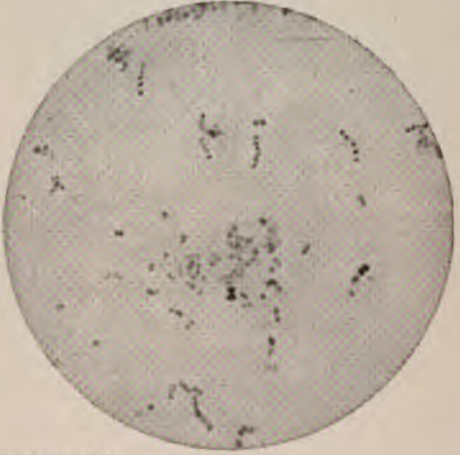
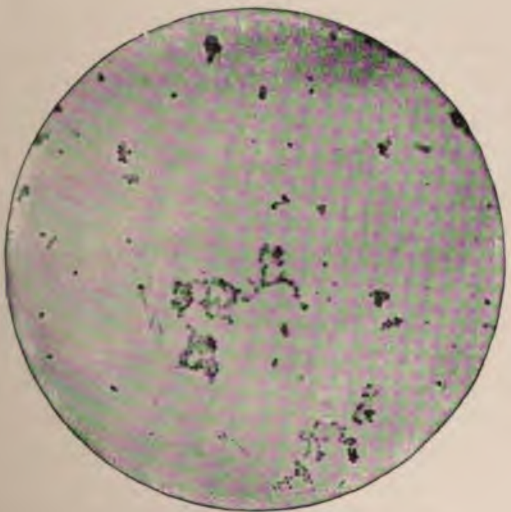


Fig. 8.



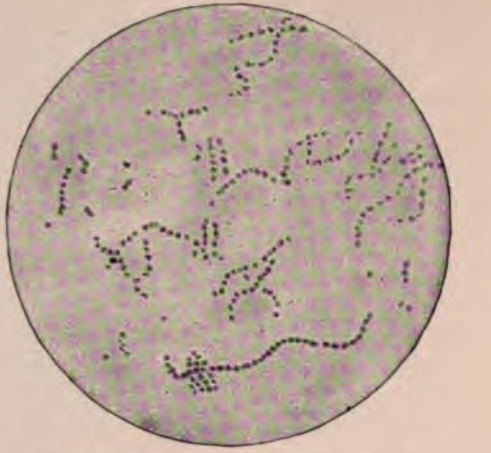
Diplococci and streptococci rigidi.

Fig. 9.



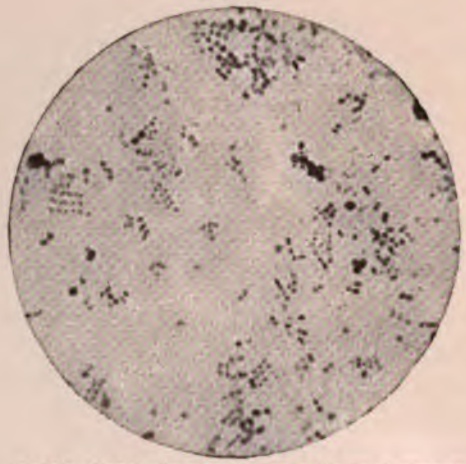
Staphylococci.

FIG. 10.



Streptococci flexuosi from a case of scarlatina.

FIG 11.



Bacillus diphtheriæ and streptococci in conglomerate or battalion arrangement.

tacks of diphtheria which are implanted on subjects already suffering from scarlet fever or measles.

When the streptococci are seen to be arranged in chains which are curved or coiled, they often indicate an exanthematous origin or association. This type of arrangement of the cocci is called *flexuous*, and is of less severity, as regards prognosis, than the battalion.

Lastly, the *rigid* arrangement, in which the chains are short and straight, represents a comparatively mild form of inflammation, and this is the variety frequently found in cases of non-diphtherial tonsillitis of unsanitary origin.

ETIOLOGY.

Since the discovery of the specific bacillus it may be fairly said that diphtheria does not originate *de novo*.

There is no evidence that the disease is limited to any particular country, geological stratum, or even to any particular climate within the extremes of heat and cold. Nevertheless, it may be broadly stated that it is more prevalent in places where the surface soil favors the retention of moisture and organic refuse.

It is now generally admitted that the season at which epidemic sore throat is most frequent is that at which diphtheria is most prevalent.

An interesting example of the liability to draw false conclusions with regard to the influence of the weather on diseases of the throat is afforded in a report issued by one of the London medical officers of health for the exceptionally dry summer quarter 1893, in which belief was expressed that an increase which had been observed in cases of throat-inflammation of an unsanitary type, consequent on long-continued drought and heat, was referable, *first*, to the wind-carrying of dried pathogenic organisms in the form of dust, and, *secondly*, to the want of proper flushing of sewers.

The writer was, however, able to show by figures compiled on a large scale that it is *not during* dry weather that throat diseases of this character are most prevalent, but on the *first occurrence of a light rainfall* after a prolonged period of heat and drought; in other words, that the epidemic wave seems to be greatest when the first rainfalls, being but slight, are sufficient to stimulate the dry and comparatively inert organic matter to activity, and that the prevalence only diminishes or disappears with the thorough flushing of the sewers consequent on heavier and longer-continued showers.

During the last forty years sanitation in city, town, and country has been greatly improved,—in many instances almost perfected,—and the death-rate from zymotic diseases has been considerably diminished. This reduction has been most marked in the case of enteric fever, a disease universally recognized as depending on defective sanitation, polluted water-supply, and bad drainage. Nevertheless, it is an unfortunate fact, which has been already insisted on by Dr. Lewis Smith in the previous edition of this work, that diphtheria, which in its early history appeared to be endemic or epidemic chiefly in the rural districts, now invades towns and cities equally; moreover, each year the mortality from this cause becomes steadily greater.

It may also be taken as accepted that all forms of epidemic sore throat may be aggravated—even if they are not actually caused—by unsanitary surroundings, a point on which the present writer is entirely in agreement with Dr. Lewis Smith.

It has been denied that such causes are sufficient to produce diphtheria. This may be so, but it may with equal confidence be asserted that they at least render a population, as well as an individual, more liable to the implantation and growth of the specific organism of the malady,—so much so that, as Sir George Johnson has aptly said, "not to recognize the frequent filth origin of diphtheria may in practice be as disastrous as to ignore its infectiousness."

It has been already hinted that a sore throat, from whatever cause, is a peculiarly favorable site for the reception and development of the diphtheria bacillus, and that some individuals are particularly liable to such affections when exposed to drain-emanations, sewer-gas, or offensive effluvia from other sources.

It is therefore certain that such non-hygienic conditions must have a considerable influence on the development of diphtheria. Abstract analysis of fifty consecutive reports of the English Local Government Board, constituting those on the various outbreaks of diphtheria occurring in Great Britain between the years 1882 and 1896, showed that in forty-six—*i.e.*, in ninety-two per cent.—there were undeniable evidences of defective sanitation, although in thirty-one only, or sixty-two per cent., was the origin of the outbreak definitely assigned by the medical inspector to have been due to this cause.

In connection with foul emanations as a cause of diphtheria, Dr. Lewis Smith points out the evils resulting from exposure of children to infected sewer-gas,—a point to which others have also drawn attention,—for sewers are now ventilated on the street level, and children have a disposition to play about the gully-holes. Dr. Lewis Smith remarks that when "the diphtheritic virus is established in a sewered city it can probably never be stamped out." These facts may in some measure be responsible for the increased prevalence of diphtheria in urban over rural districts, the converse, as already stated, having been formerly the case.

To the various predisposing causes of diphtheria there is no occasion to refer further in this article, with one exception,—namely, that of the constitutional state of the individual. And first in importance we must place that constitutional state which tends to produce obstructions to the free nasal respiration. The chief cause of this condition is enlargement of the faucial tonsils and hypertrophy of the glandular tissue in the naso-pharynx, which is now known as "adenoid vegetations."

A long experience leads the writer to the conclusion that it is rare to find diphtheria attacking a child under seven years of age unless he or she be the subject of one of these forms of glandular hypertrophy. In a large proportion of young adults who contract diphtheria it is very common to find tonsillar enlargement, and the same has been noted in hospital nurses who have taken the disease.

On the other hand, cases are not wanting to prove that removal of these obstructions to free nasal respiration appears to have conferred a special immunity to the disease on those who submitted to the operation. Lastly, in one thousand consecutive cases of diphtheria tabulated by the writer, the exudation was noted to be limited to the tonsillar region in six hundred and sixty-six cases, and in only eight out of the total number was it not implicated. That this region was the site of the disease in ninety-nine and two-tenths per cent. of the cases is a fact the importance of which, from the etiological aspect, cannot be disregarded.

There is yet one point in the etiology of diphtheria on which a word or two may be said,—namely, the conveyance of the disease from animals to man; but, in the judgment of the writer, the chain of evidence in favor of such a circumstance has in the last few years been rather weakened than strengthened. Taking into consideration the fact that the Klebs-Loeffler bacillus from the human subject, when cultivated on milk or media derived from other animal sources, varies in its morphology, it is at least probable that there are other differences in its development even more essential when inoculated into animals.

Loeffler, for instance, in 1884 described a diphtheria of calves due to the "bacillus diphtheriæ vitulorum," but neither in its morphological nor its biological characters, nor in its pathogenesis is there much similarity between this organism and that of diphtheria in the human subject.

This view of the non-identity of animal diphtheria with that of man has all along been supported by Virchow, and it has recently received powerful confirmation at the hands of Nocard, the well-known veterinary surgeon to the Pasteur Institute, who positively affirms that human diphtheria is entirely distinct from the diphtheria of fowls. The question cannot, of course, be definitely settled until some enthusiastic bacteriologist offers himself as a "*corpus vile*" for inoculation with the so-called diphtheria of domestic animals. The writer believes that, admitting milk to be a most sensitive medium for the cultivation of the diphtheria bacillus, the *materies morbi* is far more likely to come from polluted water, employed either as a cleanser of milk-vessels or as a diluent of the milk itself, than direct from the cow.

THE TOXIC PRODUCTS OF THE DIPHTHERIA BACILLUS.

As the subject of the toxins and antitoxins will be separately considered in another place by Dr. Vaughn,[1] remarks on them here as they apply to diphtheria need be but brief.

The causal relation of the specific bacillus of diphtheria to the disease having been established, it still remained a moot point to what extent the manifestations of the malady could be attributed to the presence of the organism in the membrane, until 1889, in which year all doubt was cleared up by experiments which scientifically determined the process, and established the fact that all the symptoms of diphtheria, except that of the development of membrane locally, are caused by the action of a definite poison.

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[1] See page 93.

Before proceeding further, however, it may be excusable to recall the circumstance that, in the interval between the discovery of the bacillus and that of its power to produce toxins, the present writer had in 1887, on clinical grounds alone, ventured to promulgate the view, which was at the time adversely criticised, that the principal cause of death in diphtheria was a poisoning of the system by fermentative products of the special organism.

Chief among the workers in this interesting field of research are Roux and Yersin, Hankin, Brieger and Fraenkel, Sidney Martin, and Klein. We shall describe as succinctly as possible the various conclusions at which these and other authors have arrived with regard to the toxins produced by the bacillus.

Roux and Yersin first succeeded in obtaining the toxin of diphtheria by precipitation from the original broth culture of the bacilli by means of alcohol and phosphate of lime. The precipitated product was a white or yellowish-white powder, soluble in water, having usually a neutral, but sometimes a faintly acid, reaction.

This product is capable of inducing all the symptoms of diphtheria except the membrane. Its toxic potency is reduced by exposure to a temperature of 58° C. for two hours, and is totally destroyed if submitted to a temperature of 100° C. for twenty minutes.

The views as to the nature of this substance are various and sometimes contradictory. Roux and Yersin inclined to the belief that it is a ferment, or, rather, an enzyme; Brieger and Fraenkel thought it to be a toxalbumin or a mixture of toxalbumins. Sidney Martin thinks that an enzyme is liberated by the bacillus, and that this digests the proteids of the body or of the culture-medium, and thus forms toxic albumoses, the latter producing the characteristic morbid changes. It is now known that the diphtheria toxins do not all originate in the membrane, nor do the albumoses merely accumulate in the tissues; and probably the digestion of the body-proteids, by the action of the enzyme absorbed from the membrane, forms the toxin. These poisonous substances are of two kinds,—namely, albumoses and an organic acid,—which are responsible, by their action on the nervous system, for all the symptoms,—the fever, the cardio-respiratory asthenia, albuminuria, and paralyses.

Uschinsky and Buchner regard the bacterial poisons as direct products of the bacterial cells,—that is, as derivatives from the cell-plasma. In the present state of our knowledge we cannot definitely say whether the toxins themselves constitute the poisonous element or whether they produce poisonous enzymes.

Klein has pointed out that "a definite distinction is to be drawn between the poisons which may be present in the bacteria themselves and the poisonous substances liberated or elaborated by these organisms." For he has shown that when certain micro-organisms are injected into the peritoneal cavities of rodents they produce symptoms of poisoning without any of their metabolic products being present. Moreover, if the microbes are, previous to their injection, killed by heat at a temperature of 70° C., these dead bacteria in certain cases produce the same poisonous effects as the living organisms. Therefore these micro-organisms must contain a poison in their own substance.

THE ACTION OF THE TOXINS.

When these first products of the bacillary action are introduced into the animal body, they produce a local œdema, which subsides at the end of thirty-six hours and is not followed by any destruction in the tissues, although some irregularity in temperature and, in the case of rabbits, distinct febrile conditions are found to follow the injection; the blood becomes darkened and more fluid, and its coagulation is retarded.

Larger doses produce a fatal result from **paralysis** or **coma**, but it is worthy of notice that more lasting and noxious results are obtained from small doses of the poison frequently administered than from a single large dose.

The paralysis at its onset may be rapidly fatal in its effects, or its progress may be slow and protracted; in any case, it is always progressive. The hindrance to the coagulation of the blood offers a suggestive explanation of the delayed occurrence of the paralytic sequelæ of diphtheria, even after all other symptoms of the disease have disappeared.

The paralysis is manifested primarily and chiefly on the respiratory apparatus.

The heart-muscle is in all cases in an advanced degree of fatty degeneration.

Vincent, of Paris, and P. Meyer have found wide-spread parenchymatous changes in the cardiac plexus in two cases of patients dying of heart-failure during convalescence from diphtheria, in which the heart-muscle was unaffected. The changes were exactly similar to those found in the peripheral nerves in ordinary post-diphtherial paralysis. It is desirable that future observations should be directed to the further elucidation of this point.

The voluntary muscles of the body are not by any means necessarily affected at the same time, the ones first impaired being those which of necessity are in constant use. In all cases impairment of activity of voluntary muscles is due to a degeneration of their nerve-supply.

Although there may be at first no apparent atrophy of the muscles, yet the body-weight of the animal decreases steadily.

In diphtherial paralysis both the sensory and the motor nerves are affected in a similar way, and the sympathetic system also may be involved, but, as would be expected, the part most affected in this case is the axis-cylinder.

The central nerve-cells and ganglia in the spinal cord are most probably not the original site of the lesions which take place in the nerve-trunks, for, although morbid changes in the anterior cornua of the cord have been described by some pathologists, none have been discovered in those of the posterior cornua or sensory ganglia, albeit both the motor and sensory nerves share in the degenerative process.

The affection of the nerves must, therefore, be regarded as entirely of a peripheral nature.

The change commences in the white substance of Schwann, which undergoes rapid degeneration, breaking up and finally disappearing altogether. Following on this the axis-cylinder may become ruptured,—**segmental neuritis**,—giving rise to well-marked Wallerian degeneration in the nerve-fibres below the point of rupture. All the nerve-fibres, however, may not be affected to the same extent, and some may still retain their power of conveying nervous stimuli to the muscles which they innervate.

The muscles which are deprived of nervous supply by the rupture of the axis-cylinders in the nerve-trunks undergo a process of fatty degeneration and atrophy.

The albumoses in diphtheria are therefore to be regarded as nerve-poisons, which affect the peripheral nerves, and if administered in small and continued doses produce degeneration of the nerves, leading to paralysis of both motion and sensation.

It is to be borne in mind that these paralyses may occur early in the acute stages of the disease, or in the later stages, when they come under the head of sequelæ.

The *organic acid*, when injected into animals, produces fever to a slight extent, but no paralysis. If, however, an animal be treated with several doses at intervals, the heart, post mortem, shows well-marked fatty degeneration in the muscular elements, and some of the nerve-fibres exhibit stages of degeneration similar to but of a less degree than that produced by very small doses of the albumoses. From these observations the organic acid must be regarded as a nerve-poison of a much weaker nature than the albumoses.

PATHOLOGY AND MORBID ANATOMY.

The *membrane*, being indubitably the primary pathogenic offspring of the bacillus, and the most important clinical indication in the diagnosis of the disease, will now be described.

A superficial exudation is poured out onto the surface of the epithelium of the mucous membrane, and this, being rich in albumin, coagulates into a tough, firm, and elastic layer, containing dead epithelial cells, leucocytes, and often red blood-corpuscles. It consists mainly of a net-work of fibrin, to which fresh layers may be added as the inflammatory process goes on.

In the superficial layers, which are the oldest, numerous indifferent micrococci are usually to be found along with the characteristic Klebs-Loeffler bacilli, which may, however, be detected in all parts of the membrane.

Until quite recently it has been laid down as an axiom that the organism is rarely, if ever, found beyond the immediate vicinity of the exudation.

On the surface, as well as in the stratum between the false membrane and the denuded epithelium, the streptococcus pyogenes and staphylococcus aureus and albus are also very frequent companions of the bacillus, and these organisms may travel into the deeper tissues, lymphatics, and blood-vessels, when they constitute the cause of secondary infective processes in the lymphatic glands, lungs, spleen, and other organs.

The lower strata of the mucous membrane are also invaded by the exudation, and the coagulation process extends deeply into the submucous layers, which become infiltrated with small inflammatory cells, leucocytes, and micrococci. By this process the membrane becomes progressively firmer and tougher by each fresh deposit from beneath. Thus the external or upper layer of the diphtherial false membrane, which is oldest, when it becomes disintegrated, is thrown off in the form of a slough. The subjacent epithelium soon disappears, and on this denuded surface a layer consisting of small cells, pus-cells, and granular débris is formed, separating it from the dead material above.

The destructive process, extending deeper to the submucous tissues, involves the blood-vessels and the lymphatic channels.

The blood-vessels become constricted from pressure; local thrombosis and embolism follow, with ulceration, necrosis, gangrene, and extravasation of blood. The lymphatics of the part are filled with leucocytes, pus-cells, and cocci, which, passing into the nearest lymphatic glands, produce swelling from infiltration of inflammatory cells, and finally suppuration.

The diphtherial exudate is adherent, and can be removed only by the employment of some force, when a raw, inflamed, and bleeding surface is exposed.

The membrane varies much in its color, from that of white, faintly tinted with a pearly gray or lemon, to deep grayish green, brown, or almost black, the intensity of hue depending partly on the age of the exudate and largely on the amount of blood extravasated.

The diagnostic characteristic of a true diphtherial exudate is that it contains the Klebs-Loeffler bacillus, the presence of this organism being an irrefragable indication of the nature of the disease. When the malady marches towards cure, the bacilli diminish in number, and very often disappear with the membrane. On the other hand, they may persist long after the individual is apparently cured.

Pseudo-diphtherial membrane due to coccal infections is characterized by an excessive production of fibrinous material, which forms a net-work of delicate strands and fibres, enclosing in its meshes leucocytes, granular débris, and pus-cells. The false membrane is attached by thin fibrinous threads to the surface of the tissue beneath. The exudate is readily soluble in dilute acetic acid.

The epithelial cells become altered in character, swollen, and undergo fatty degeneration; they desquamate freely.

The membrane, being only loosely adherent, can easily be stripped off, revealing a hyperæmic but not a bleeding base. The necrotic process does not involve the entire epithelial surface, for on removal of the exudate some patches will be found to be normal in character and unaffected.

As extravasations of blood into the interstices of the membrane are exceptional, pseudo-diphtherial exudation rarely presents a brown or black color, but is usually of a whitish-yellow or grayish appearance.

The criterion of differentiation between truly diphtherial membrane and pseudo-varieties is the presence or absence of the specific bacillus.

Diphtheria must be regarded as primarily a local infection, for the bacilli are found chiefly at the seat of infection,—namely, in the false membrane.

In fact, as mentioned by Dr. Lewis Smith in the early history of this subject, the bacillus was not found in the blood-vessels, lymphatics, or internal organs, and this was accepted as decisive evidence that its pathogenic action must be localized on the surface.

Thence arose the idea that the systemic infection was due to a chemical poison generated by the specific microbe.

But it is now known that the bacilli may escape from the site of the first deposit and find their way into the vascular system or into the lymphatic vessels, by which channel they may invade the spleen, kidney, lung, and so forth, or the various lymphatic glands.

Frosch (1893) and Wright (1894) discovered the Klebs-Loeffler bacillus in the lungs and spleen; later Bulloch and Schmorl (1894) found it in the submaxillary, bronchial, and mesenteric glands; and still more recently (1897) Kanthack and Stephens have confirmed the accuracy of these researches by independent investigations.

The mortality, when there is true diphtherial infection of the lung, spleen, and other organs, is, according to the last-mentioned authorities, exceedingly high, and in almost every case in which the larynx is involved and tracheotomy becomes a necessity, the diphtheria bacillus can be demonstrated in the lungs.

THE BACTERIOLOGICAL DIAGNOSIS OF DIPHTHERIA.

The presence or absence of the Klebs-Loeffler bacillus can usually be decided in a very short time by removing a portion of the membrane or suspicious exudate by means of a swab of sterilized cotton-wool, a platinum wire loop, or a pair of forceps. This small fragment of membrane is rubbed over the surface of a clean cover-glass, and the film so obtained is dried and stained with Loeffler's blue staining solution, or, preferably, with Roux's combined stain of dahlia violet and methyl green.

The cover-glass, being prepared, is mounted in the usual manner and submitted to microscopic examination. The bacilli should at once be recognized, if present, by their typical characteristics. If bacilli are not found by the above method, a streak culture may be made on blood-serum, when the characteristic colonies of the diphtheria bacillus will, if present, be evident after an incubation of twenty-four hours at a temperature of 37° C. Moreover, the presence or absence of other organisms—*e.g.*, strepto-, staphylo-, or diplococci—may be determined, after more than twenty-four hours have elapsed, by the appearance of colonies which are characteristic of each.

Finally, the microscope will confirm the diagnosis and establish the presence or absence of any particular form of micro-organism which may be present in the particular cultivation.

Presuming that our colonies have developed, how are we to know that they are those of the diphtheria bacillus?

After the culture-tubes have been in the incubator at a temperature of from 35° to 37° C. for from fourteen to eighteen hours, colonies of the diphtheria bacillus appear as whitish-gray specks, each about the size of a pin's head; the contour is regular and the surface dry. By transmitted light the centre of the colony is seen to be thicker and more opaque than the periphery, which is translucent.

An almost positive diagnosis of the presence of the diphtheria bacillus is therefore established,—that is to say, a **positive naked-eye** diagnosis has been obtained.

The **streptococcus** is of much slower growth, and its colonies may be distinguished from those of the bacillus by the fact that they do not begin to form until the lapse of at least twenty-four hours. They appear as white colonies, very much smaller than those of the bacillus, and resemble so many pin-points as compared with the pin-head colonies of specific organisms themselves.

The **staphylococcus**, although more rapid in development than the streptococcus, resembles it in its slow rate of growth as compared with that of the diphtheria bacillus; its colonies are much larger than those of the bacillus diphtheriæ. They are of a flocculent or snow-white appearance, darker in the centre, but thinner at the edges, and the halo-like effect at their periphery is somewhat increased over what is observed in a colony of the Klebs-Loeffler organism. These colonies often take from two to three days to develop properly. A yellowish or golden tint may appear in some cases, but this is by no means either an early or a constant occurrence.

Preference is given to blood-serum as a culture-medium, because the diphtheria bacilli grow more rapidly and preserve more of their special characteristics on this than when cultivated on other media.

Hydrocele fluid has been advocated as being of special value for the culture of the Klebs-Loeffler bacillus. It is not easy to obtain, except the worker be near to a large hospital. The advantages claimed are that the fluid is perfectly transparent, and the bacilli grow rapidly on it and preserve the same characters as they do when grown on blood-serum.

Moreover, the growth of many other associated bacteria is stated to be more than usually retarded in this medium, streptococci requiring three or four days to develop, and staphylococcus aureus growing still more slowly, and producing a yellow coloration which is quite diagnostic. As already hinted, nothing short of animal inoculations—an impracticable measure in the case of a human patient—will satisfactorily distinguish between the virulent and the non-virulent bacillus. Neisser has recently pointed out a sure and rapid method of distinguishing between these two microbes, based on the staining properties belonging to the granules present in the active and virulent, and absent in the attenuated or non-virulent organism.[1]

The practitioner must be cautioned against accepting too readily a negative diagnosis as decisive; for not every bacteriologist, any more than every practitioner, is equally skilled in making his preparations and cultures, or equally capable of accurately interpreting the evidence they may afford.

Added to this, the swab employed to remove the secretion may not always reach the site of the bacilli. Some such explanation seems to afford the only possible excuse for the following circumstance which happened in one of the writer's own cases:

Repeated bacteriological examinations of the throat had given negative results as to the presence of the Klebs-Loeffler bacillus, but on the twelfth day of the attack otorrhœa occurred, and a culture taken from the aural discharge so soon as it was observed, and before any local treatment was commenced, demonstrated the presence of the specific bacillus.

The following are the formulæ of the staining solutions for microscopic preparations which are most generally used:

Loeffler's Solution.

Take of Concentrated alcoholic solution of methylene blue, 30 parts;
Solution of caustic potash (1 in 10,000 water), 100 parts.
Mix.

Place the cover-glass in this solution for from three to five minutes, wash, dehydrate, and mount in balsam.

Another method of employing Loeffler's stain is as follows: the cover-glass preparation is strongly stained in Loeffler's solution, and is then placed in a watch-glass of water with about two drops of glacial acetic acid, by which it is decolorized; it is then dehydrated and mounted in the usual manner.

The decolorizing process must not be carried too far, as the oil of cloves used for clearing, and the xylol balsam used for mounting, will still further reduce the strength of the stain.

Roux's Double Stain.

Take of Dahlia or gentian violet, 0.5 gramme;
Methyl green, 0.5 gramme;
Distilled water, 200 centimetres.
Mix, and filter before use.

Sections are placed in this stain for twelve hours, then washed, dehydrated, cleared, and mounted.

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[1] Berl. Klin. Woch., December 13, 1897.

Gram's Method.

Take of Aniline water, 100 parts;
Concentrated solution of gentian violet, 11 parts;
Absolute alcohol, 10 parts. Mix.

Then rinse and transfer to Gram's solution as follows:

Take of Iodine, 1 part;
Iodide of potassium, 2 parts;
Distilled water, 300 parts. Mix.

Place sections in this solution for from one to three minutes.

Some pathologists recommend to simply drain off the gentian violet, and to immerse direct in the iodine solution, without previous rinsing, until the sections acquire a brown color; this takes place in from one to three minutes. The sections are then washed in alcohol until they are of a pale-yellow color, dehydrated, cleared, and mounted in balsam.

When the micro-organisms are contained in tissues, it may be necessary to counterstain in eosin.

CLINICAL DIAGNOSIS.

Details of those points of clinical diagnosis to be found in the first issue of this work and in other treatises need not be repeated here, and but little need be added in this article from the point of view of purely bedside observations.

But those who have not been in the habit of comparing what can be seen at the bedside with the results to be obtained in the bacteriological laboratory will be surprised to find how much confirmation, or it may be correction, the one is capable of affording to the other. Many illustrations have been already given, and it is from this aspect that our remarks on diagnosis will be based.

The diagnosis of the malady, as its name suggests, rests mainly on the presence of a false membrane; but membranes—at first sight very similar to those of true diphtheria—may result from the action of various irritants, such as strong acids, ammonia, eau de Cologne, scalding water, caustics, and so on.

The bacteriological test is therefore essential. Moreover, as has been already stated, such an examination is the more necessary since each day fresh facts are recorded to warrant a belief that diphtheria may arise and proceed through all its stages without the detection of membrane. But this conclusion is not as yet generally accepted by practitioners, for, as Hermann Biggs has well said, "there still remains the firm belief with a large proportion of the profession that only such cases of acute angina are to be regarded as diphtheria as present at some time in their course more or less membrane."

Of a quite separate category are those inflammations of the throat accompanied by exudation of pseudo-membrane which occur during the course of the specific fevers. Until the elucidation afforded by bacteriology, these were considered by the majority of authors from the time of Home, in 1765, to be truly diphtherial, notwithstanding many vigorou and weighty arguments which were to the contrary. We now know that the membranous inflammations of the throat exhibited previously to or during the early stages of any of the exanthemata are not, as a rule, of the nature of true diphtheria; while such as are implanted on them in defervescence or as sequelæ are almost invariably proved to be absolutely of that nature by the presence of the Klebs-Lœffler bacillus.

In many cases these doubts can be solved, in a large degree, by a careful comparison of the symptoms, both physical and functional, of those membranous sore throats which are characterized by the micro-organism recognized as specific of diphtheria either alone or in association with cocci, and by due recognition of other more special evidences of the different diseases in which they are found.

The following are the particular points to which attention may be usefully directed:

Age.—The writer has long insisted on a fact which must appeal to the experience of most practitioners, that while membrane is the almost constant characteristic of acute inflammatory affections of the fauces and larynx in infant life, its absence is to be frequently noted in similar cases occurring in persons of full age, and, curiously enough, it is seldom to be seen in infants at the breast, though even in the newly-born the system may be infected with the specific toxæmia.

Such a fact may tend to harmonize recent but established experience with the apparently contrary opinion of Dr. Lewis Smith, who believes that "under certain circumstances the newly-born infant exhibits considerable susceptibility to diphtheria."

The following table of one thousand consecutive cases analyzed by the writer shows the frequency and the proportionate mortality of diphtheria according to age:

Males and Females.

AGE.	NUMBER OF CASES.	DEATHS.	MORTALITY. PER CENT.
Under 1 year	11	5	45
Under 5 years	392	176	45
Between 5 and 10 years	317	83	26.2
Between 10 and 15 years . . .	102	12	11.7
Over 15 years.	178	7	3.9

It is thus seen that in these one thousand cases the occurrence of diphtheria in patients under one year of age was 1.1 per cent.

An analysis of nearly six thousand cases demonstrated the frequency as not exceeding 1.5 per cent. The youngest patient in whom the writer has seen diphtheria with membrane was six weeks of age.

The degree of inflammation present in the throats of patients attacked with pure diphtheria is of a lower grade than that of throat manifestations in connection with scarlet fever or measles.

Experience leads the writer to believe, although this opinion has been contradicted, that œdema of the uvula and velum is an indication of the malady being non-diphtherial, or at least of a mixed character. We have never seen it in the case of a pure diphtherial infection.

A point worthy of note is, that unmixed diphtherial throats are almost always dry throats, as likewise are the cultivations of the diphtheria bacillus on blood-serum. An excess of mucus or saliva always suggests a coccal infection or complication, and the same may be said of local suppurations.

As to the site of the membrane,—

From the following table the relative frequency of site in one thousand cases will be at once apparent, and, to save space, the relative mortality according to situation is included:

Table of One Thousand Cases of Diphtheria, showing the Relative Frequency of Sites of the Membrane and Proportionate Mortality.

PART AFFECTED.	NUMBER.	DEATHS.	MORTALITY. PER CENT.
Fauces (alone)	666	81	12
Larynx (alone)	4	1	25
Nostrils (alone)	2	1	50
Fauces and larynx	112	51	43.7
Fauces and nostrils	165	106	64 2
Fauces, larynx, and nostrils	49	30	61.2
Labial or buccal only (involved in 6)	1	2	33.3
Hard palate only (involved in 12)	1	11	91.6

Faucial.—The *tonsils* were the sites in all the cases where the situation is described as that of the fauces, and this disposition to attack the tonsillar tissue is evidenced not only in the faucial region, but in the pharyngeal vault, where is situated the **pharyngeal tonsil**, at the orifice of the Eustachian tubes (**tubal tonsil**), at the base of the tongue (**lingual tonsil**), and, finally, in the ventricles of the larynx, the glandular tissue of which may be held to represent in some degree a **laryngeal tonsil**.

It is a peculiarity of the diphtherial exudation to start in some small recess, as in the lacunæ of the tonsils, or on some little prominence, such as the uvula, the free edge of the epiglottis, or the eminences of the cartilages of Wrisberg and Santorini.

Laryngeal.—The frequency of *laryngeal* extension, as shown in the above table, is doubtless underestimated, for the laryngoscope was not used to aid the diagnosis in these cases. It is a matter of regret that in these days, when the art of laryngoscopy is so generally cultivated, it should be so seldom employed in cases of suspected diphtheria of the larynx. For by its aid diagnosis would be rendered more complete, and, intra-laryngeal treatment being more early adopted, a better chance of life would be given to many a patient. In the case of intubation, both introduction and extraction of the tube would, by the aid of the mirror,—an easy matter when the mouth is propped open,—be accomplished with greater accuracy and speed.

Nasal.—When the nares are involved the mortality is so high that an attack thus characterized has been aptly called "malignant diphtheria;" indeed, of the cases in the one thousand tabulated in which the nasal fossæ were implicated the death-rate amounted to 63.4 per cent.

According to Dr. Lewis Smith, "the occurrence of pseudo-membrane in the nares is common," and this opinion, although not generally recognized, is confirmed by the foregoing tables.

For, though diphtheria but rarely invades the nares primarily, and is limited to that situation,—only two in one thousand are here recorded,—nasal diphtheria, as represented by an extension of the exudation from the pharynx, occurs in 21.3 per cent. of all cases.

It may, indeed, be generally stated that the nares are almost always involved when membrane is exhibited on the posterior surface of the uvula. Its presence is further indicated by a nasal discharge, which is usually of a peculiarly fetid, sanious, and irritating character, causing excoriations and sores round the nostrils and on the upper lip. The fetor of the flux in the case of diphtheria is of itself quite distinctive, for it is never to be observed in cases of ordinary acute endo-rhinitis or coryza; so that this symptom of foul odor has led to the adoption of another synonym of diphtheria, "putrid sore-throat." The discharge is often tinged with blood, which symptom is occasionally intensified by sudden and profuse attacks of epistaxis. This form of the malady is always to be regarded as of the gravest import, since systemic infection rapidly ensues, and fatal prostration, asphyxia, and pulmonary complications are almost certain to follow.

The reason for the exceptionally intense toxæmia of nasal diphtheria is to be found in the fact that the nasal passages offer more than any other site, both in their extensive area and in their readily absorbent surfaces, the conditions most favorable for the production of the toxic enzymes, the turbinals constituting not only an extensive and highly absorbent area, but also a source of direct cerebral infection through the cribriform plate. In fact, the arrangement of the apparatus employed in the laboratory for the manufacture of the toxins bears a striking resemblance to that of the contents of the nasal cavities.

Bacteriological examination of the membrane from the nasal cavities demonstrates that the disease is always of a "mixed" nature, the character of which may often be provisionally foretold according to the nature of the local symptoms. The associated organisms are most frequently streptococci of various degrees of virulence according to the severity of the local evidences. The exudate may in some cases be discharged from the nares as a large plug taking the shape of the turbinals with their corresponding choanæ. In others shreds of membrane may come away, but in the majority of cases the fluid discharge is the main feature, and any argument to the effect that the disease is not diphtheria, because no membrane is either seen or liberated, is based on ignorance of this fact.

When the nasal discharge is sanious and very irritating, the streptococci are found in large numbers, and are usually of the most virulent form, especially in those cases where profuse epistaxis is a prominent symptom.

In purulent and sanguineo-purulent discharges staphylococci may also be found. In one case diplococci were likewise discovered, and the presence of these organisms would suggest or confirm a diagnosis of pulmonary complication. Extension of the inflammation along the Eustachian tubes is not uncommon, although by no means so frequent as in scarlet fever or measles, even if allowance be made for the relative frequency of the two diseases.

The *aural* complication of diphtheria, unlike that of these last-named exanthemata, does not appear to be characterized by a great degree of pain, nor is its prognostic importance in regard to meningeal inflammation or mastoid suppuration great, although periostitis, which requires relief by incisions, does from time to time occur. It is not out of place to remark here that among the sequelæ of diphtheria, deafness, both in the middle and in the internal ear, is sufficiently frequent to occupy our prognostic consideration. It may also here be mentioned that in one case of nasal diphtheria death ensued from meningitis, and no aural complication was to be found. This circumstance offers a not improbable explanation of the gravity of nasal diphtheria. For not only, as already mentioned, do the turbinals constitute an extensive and readily absorbent surface, but there is a liability to direct cerebral infection through the cribriform plate by a process similar to that sometimes observed in regard to cerebro-spinal meningitis, when the specific organisms have been found in the anterior meninges.

Experience of non-diphtherial purulent otitis would lead one to expect that in diphtheria the staphylococcus would be the most commonly associated organism of the bacillus; but in the few cases of diphtherial discharges from the ear examined by the writer the streptococcus has been found quite as frequently, and even more abundantly.

Ocular.—In some instances, rare though they may be, the inflammation may pass from the nose along the lachrymal duct to the eye. In other cases the bacillus may become implanted directly on the conjunctiva.

Dr. Lewis Smith has pointed out that children who are suffering from simple conjunctivitis or from granular lids are very liable to contract diphtheria when exposed to infection, the malady having a decided predisposition to attack surfaces already inflamed. True diphtheria of the conjunctiva is a serious complication, and often results in permanent opacity of the cornea, and even total destruction of the sight.

We have already described an organism—"the Xerosis bacillus"—resembling the Klebs-Loeffler organism, as found in some cases of hypersecretion of the conjunctiva, and this is probably responsible for many cases erroneously diagnosed as ocular diphtheria which run a mild course without either systemic infection or subsequent paralyses.

Adenitis.—The enlargement and tenderness of the lymphatic glands in the neck constitute not only a very early and constant symptom, but also one of considerable diagnostic value. It may precede the appearance of the membrane in the throat, or may indicate its presence in some part not visible on examination. To a certain extent the amount of glandular enlargement is proportionate to the virulence of the disease. There is a distinct difference in the portion of the glandular region affected.

In **pure** or **simple diphtheria** the **cervical** glands are those mostly attacked. This may be in the form of enlargement and tenderness of the whole chain of glands, or, in the gravest cases, as one large swollen mass in the neck, in which the **parotid** may also be involved. In **complex diphtheria**, both the **cervical** and **submaxillary** glands are affected, and in **pseudo-diphtheria**, where streptococci are predominant, of which scarlet fever is a striking example, the submaxillary glands are those most frequently affected. In diphtheria following scarlet or other specific fever—in other words, when streptococci are associated with the diphtheria bacillus—both sets of glands are inflamed concurrently.

Adenitis is much more common in scarlet fever than in diphtheria; indeed, it may be taken as one of the ordinary symptoms of the former disease. It is far more intense, and suppuration, to which it is also more prone, takes place at a much earlier date. Nevertheless, suppurating adenitis and cellulitis are far more frequent in true diphtheria, especially in the cocco-bacillary varieties, than is generally taught, for this complication—or, as it may almost be termed, this sequela—was found in 9.75 per cent. of our one thousand cases.

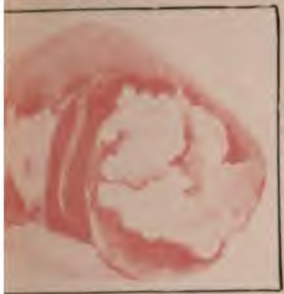
THE MEMBRANE IN DIPHTHERIA.

The clinical diagnosis of diphtheria depending, as it does, so greatly on the presence of false membrane in the throat, makes it necessary to devote a brief space to consideration of the different forms of pseudo-membrane which occur in other acute specific fevers, and the other signs by which these may be distinguished from diphtheria.

Fig. 12 represents what may be called the gradual and creeping type, and also the circumstance that the membrane generally commences as a fine, semi-translucent, pellicular exudation, increasing to a thick, opaque membrane, the density of which, however, is variable. The bacteriological characteristics of this case are depicted in Fig. 1.

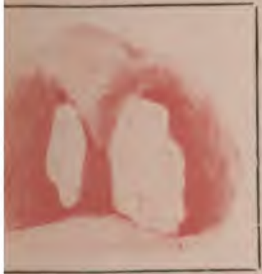
A picture less frequently seen, but more characteristic of the name which has been given to the disease, is shown in Fig. 13, in which the exudate from the first was plastered on the tonsils as if "laid on with a trowel." Fig. 14 shows the bacteriological appearance in this case. It is of special interest when compared with Fig. 1, for while in both the clinical diagnosis was thereby satisfactorily established, and the two cases were of equal gravity, the variations in the dimensions of the bacillus confirm what has been already said as to the untrustworthiness of a prognosis solely or mainly based on this particular feature.

FIG. 12.



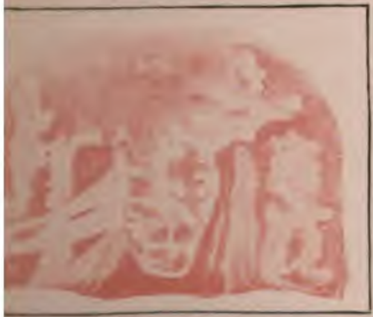
mbrane of diphtheria on tonsils.

FIG. 13.



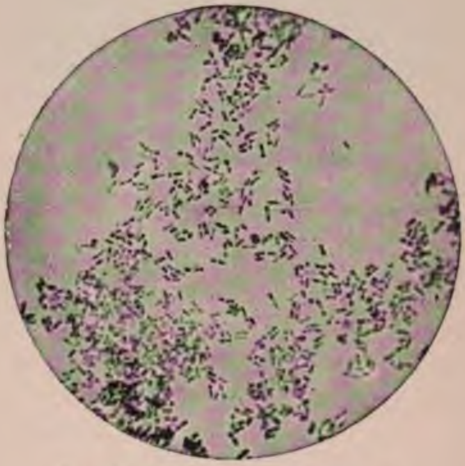
ne of diphtheria; emplastered exudate.

FIG. 15.



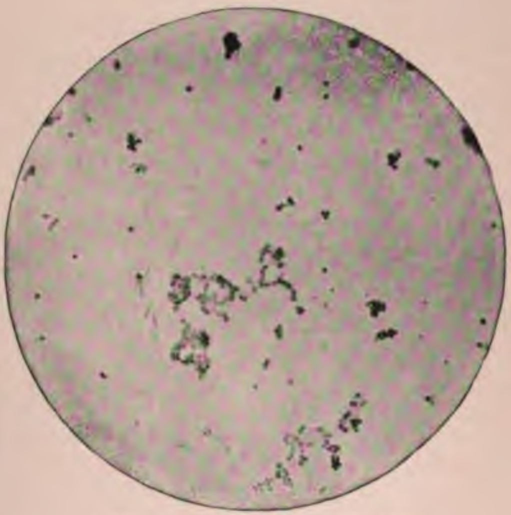
al appearance of staphylococcal throat. (See page 194.)

FIG. 14.



Bacteriological appearance of same case.

FIG. 16.



Bacteriological appearance of same case.

DIFFERENTIAL DIAGNOSIS.

Scarlet Fever.—False membrane is rare in scarlet fever throats. The exudation in such cases is yellowish in color, dirtier, or grayer than in the early stages of diphtheria. It tends to become pulpy, pultaceous, or purulent, and to leave deep and destructive ulcers, producing characteristic and unhealing perforations in the pillars of the fauces. Scarlet fever is responsible for suppurative inflammation of the middle ear; this is attended by great pain, and is liable to extend to the mastoid antrum and the meninges. Otorrhœa is less frequently a complication of diphtheria, is not as a rule painful, and is less obstinate to treatment. No membrane is present in the nose in scarlatina, but there is often a purulent discharge. Epistaxis is not common, but hemorrhages from the throat are by no means infrequent, on account of the deep ulcerations. The larynx is but rarely attacked as compared with diphtheria and measles, and when it is, the inflammation is usually of the nature of an acute œdema. The temperature is nearly always higher and the onset more sudden than in diphtheria.

Lastly, the rash, which in scarlet fever is of almost constant recurrence and punctiform in character, is only occasional in diphtheria, and is then erythematous. This of itself may often afford a conclusive criterion of discrimination.

Measles.—Membrane on the fauces is very rarely observed during the course of an attack of measles, although true diphtheria is not infrequently implanted on this exanthem as a sequel, in which circumstance the diphtheria is manifested in a very fatal type. Coryza is an early symptom, and great redness of the conjunctivæ is a well-marked feature. This is sometimes accompanied by manifestations of false membrane on the eyelids.

Measles is particularly liable to be characterized by general implication of the air-passages. Hence we frequently find as a complication or sequel, laryngitis of an acute but non-membranous character, pneumonia, and bronchitis. There is less tendency to severe suppuration and ulceration in the throat, but middle-ear troubles are very common, and usually commence as a serous inflammation, while those observed in scarlet fever are almost always suppurative in character, as they are also, but not so constantly, in diphtheria.

Typhoid Fever.—A certain amount of pharyngeal and laryngeal inflammation and even serious ulceration is not uncommon in enteric fever, and, if looked for, a faucial exudation, easily to be distinguished from aphtha, may be seen about the third week. Independently of the special commemorative signs, the characteristic point about the throat is that the membrane does not become thicker nor separate more easily than in scarlet fever; the prevailing organism is the staphylococcus rather than the streptococcus. Lastly, it is altogether of a milder nature. The characteristic course of the temperature, headache, diarrhœa, etc., in most cases materially assist in forming a differential diagnosis. It must not, however, be forgotten that true diphtheria sometimes, although rarely, supervenes on an attack of typhoid fever. A diphtherial enteritis is also not unknown, and the author has recorded two cases.

Small-Pox.—In very severe cases of diphtheria, especially those in which the nares are the sites of membrane, epistaxis is a frequent symptom, similar to such as occurs in cases of hemorrhagic small-pox.

Exhaustive inquiries from superintendents of infectious-fever hospitals prove that membranous exudation of the throat is practically unknown in small-pox, and has only been occasionally observed in the malignant form of this disease; in these rare cases deep ulceration and necrosis, leading to œdema of the glottis, have occurred.

Non-Diphtherial Tonsillitis.—This affection of the throat is perhaps that which is most frequently mistaken for true diphtheria, and therefore demands more than passing notice. Indeed, there can be but little doubt, as already mentioned, that many cases of lacunar tonsillitis, especially in the adult, are really diphtherial in an early stage, the true nature being revealed only subsequently by the characteristic paralytic sequelæ of diphtheria. The membrane—though it hardly deserves that title—in ordinary tonsillitis can be readily detached by the swab without any hemorrhage. It consists mostly of necrosed epithelial cells; it is soft and friable, with a fetid odor, and occurs in small, soft, rounded masses very different from the firmly adherent fibrinous exudation of true diphtheria. It is almost always accompanied by much mucous secretion of a viscid, ropy, and clogging character, diphtheria being a much drier affection of the throat. The diagnosis is helped by the sudden onset, the high grade of temperature, and, above all, the pain. Difficulty of opening the mouth and extreme dysphagia are also important symptoms of distinction in acute tonsillitis long before suppuration occurs.

Lastly, the exudation in tonsillitis is almost always confined to the tonsillar region, and does not spread to the fauces.

The accompanying figure (15) illustrates the appearance of a non-bacillary diphtherial throat due to coccal infection,—in this instance the staphylococcus. The ropy cloying mucus bands are almost alone sufficient to distinguish it from the dry throat with well-defined exudate in true diphtheria.

ELEMENTS OF PROGNOSIS.

I. *Bacteriological.*—The first point to determine is, whether the case is one of true diphtheria or pseudo-diphtheria. The presence or absence of the Klebs-Loeffler bacillus will decide this question.

The morphological character of the bacilli present will not appreciably influence our prognosis as to the virulence of the attack.

Secondly, is the specific bacillus associated with one or more of the cocci which have been shown to exert an influence on the character of the disease? If cocci are present, then the nature of those which predominate must be taken into account, and their significance must be duly considered according to their variety, numbers, and arrangement.

The general statement may be made that the less the Klebs-Loeffler bacillus is associated with other micro-organisms the more favorable will be the prognosis.

The writer's own experience leads to the opinion that when streptococci are associated with the diphtheria bacillus it may be predicted, if they are not already present, that enlarged submaxillary and parotid glands, phlegmonous inflammation of glands and tissues, broncho-pneumonia, nephritis, and other septic complications will present themselves in the course of the malady, which, if they do not prove fatal, will materially retard recovery and convalescence.

The presence of staphylococci in large numbers is an indication that suppurative processes may be expected.

II. *Clinical.*—The prognosis of diphtheria is always difficult, and should in every case be a guarded one, for every epidemic of the disease differs in character and in severity of type.

First, it is necessary to consider the *age* of the patient, for there can be but little doubt that there is an age disposition to diphtheria, as well as an age mortality, due to two causes, one of which is the disposition to nasal obstruction and enlargement of the tonsils (allusion to which has already been made in discussing the etiology of the disease), and another the greater tendency in the infant to membranous exudations in all acute inflammatory conditions of the throat, quite irrespective of contagiousness, as compared with the submucous infiltration with œdema in analogous affections when they occur in the adult.

There is also as much hypersensitiveness to diphtheria in the young as there is to scarlet fever, measles, and the like, which, moreover, are themselves so frequently the forerunners of an attack of diphtheria. And it is worthy of notice, in this connection, that in all epidemics of diphtheria, as well as in the large majority of sporadic cases, children are the first to be attacked.

In the case of an epidemic the prognostic data of any individual case should be regulated with due regard to the character of the prevalence, as judged by other cases in the neighborhood. The source of primary incidence and personal infection must also influence one's forecast, for undoubtedly the virulence of a case may be modified in its intensity by such general and special circumstances.

The *character, amount, and extent of the false membrane* present in any individual case are also important factors in the prognosis. The larger the surface affected and the more abundant the exudation, the graver will be the forecast; for it must be evident that under these circumstances the prognosis is rendered more grave from the twofold point of view of increased mechanical obstruction to respiration and the greater opportunity for the generation and absorption of the toxic products of the specific organisms present in the membrane.

On the other hand, we know by recent bacteriological investigations that, although the membrane may be present in only very minute quantity, or even entirely absent so far as ocular inspection can determine, yet the specific bacillus may be present in the throat in its most virulent form, and for toxic reasons a grave prognosis would then be indicated.

The relative mortality of diphtheria according to the *site of the membrane* has already been considered, and it may be generally stated that when the surface involved is of large extent, the membrane very thick, of a fetid odor, and with a tendency to become gangrenous, and, lastly, associated with a foul discharge from the nose, indicating the presence of membrane in the nasal passages, the case is one of great severity, and the prognosis must be most unfavorable.

It is a peculiarity of diphtheria, apart from almost every other disease, that the exact date at which the patient can be declared "out of the wood" is most variable and uncertain; for death often occurs suddenly after a comparatively mild attack and at a period when the patient seems to be convalescent.

Death from *convulsions* and *coma*, rare as it is, may safely be prognosticated to be the result of uræmic poisoning, though the possibility of cerebral complications must not be lost sight of. A peculiarity of the uræmia of diphtheria is the clearness of intelligence up to the moribund stage, or even up to cessation of life itself.

The mortality due to *nephritis* and its results, according to our tables, was 2.7 per cent., and more detailed examination would appear to show that there is some relation between this renal complication and nasal obstruction, which will readily be understood by those who recognize the influence of the last-named condition on the oxygenation of the blood. Moreover, there is strong probability that the frequency with which suppression of urine is associated with epistaxis is more than accidental.

A possible explanation of the frequency of nephritis may be found in the fact that a considerable proportion of cases of diphtheria follow on scarlet fever, in which, however, albuminuria occurs at a later date than in diphtheria, though in the latter it often continues long and may be responsible for permanent renal mischief.

Sudden death may occur from suffocation, asthenia, and cardiac, renal, pulmonary, neurotic, and other causes. In the first week it may ensue from obstruction to respiration and its consequences, either with or without operative interference, when the larynx is involved. Paralysis of the respiratory or cardiac functions may be responsible for a fatal termination at a quite early or at a later date. Again, the formation of a fibrinous clot in the heart or great vessels may lead to a sudden and unexpected end.

LOCAL, GENERAL, AND CONSTITUTIONAL TREATMENT.

The various lines of treatment which were generally accepted and adopted prior to the discovery of the Klebs-Loeffler bacillus and to the introduction of "antitoxic serum" have been so completely considered in the previous issue of this Cyclopædia that there is little new to add.

But since it is now fairly established that no element of treatment, whether constitutional, topical, or operative, can be ignored even by those who counsel serum therapy, it may not be superfluous to give in full detail the measures which would be adopted by the writer in cases of diphtheria independently of, or as adjuvants to, the employment of antitoxic serum.

At an early date measures should be adopted to facilitate the elimination of the toxic elements of the disease by the excretory channels. For this purpose, at the onset of an attack the bowels should be freely opened by the administration of calomel in the adult patient, or in the case of children, even though diarrhœa is a symptom, by the same drug, or by gray powder, preferably combined with small doses of antimonium,—to wit, the old James's powder. To reduce local inflammatory symptoms, even before any membrane is manifested, the salicylate of sodium, combined with chlorate of sodium and decoction of cinchona, is very useful; when membrane is seen, and is recognized as diphtherial in character, iron should be given in full doses (combined with chlorate of sodium or of potassium), for this drug has proved to be, from general experience during the last forty years, the sheet-anchor of the constitutional treatment of diphtheria.

In cases of cardiac depression, strychnine should be added to the iron mixture, or it may be given hypodermically. To produce the best results the drug must be given in full doses; as much as five minims of the standard solution of the British Pharmacopœia may be administered three or four times in twenty-four hours without risk even to young children.

As to topical remedies, the application of pure lactic acid of the strength adopted in the British Pharmacopœia—practically the same as that of the United States—on a swab of cotton wool once or twice a day with sufficient force to detach the membrane at its edges, or even to remove it, has proved so satisfactory in the writer's own practice that he has rarely had to resort to any other measure. A diluted—1 to 2 or 1 to 4—solution of the same remedy may be employed twice or thrice daily by the nurse in the intervals of the doctor's visits. Of the local and constitutional effects of calomel, insufflated or internally administered, albeit much lauded by many, especially in the United States, he has had little experience. Sulphur and the sulphites have given good results, for the twofold reason that they act both systemically and locally, but they would appear to be more useful in cases of coccal infection than in those of truly diphtherial origin. Peroxide of hydrogen as a local application should not be neglected, for its value has been proved by the experience of many American colleagues. Theoretically it is an almost ideal germicide, but it is both unstable in composition and variable in its results.

As a local germicide after the detachment of the membrane, the biniodide of mercury is to be preferred to the bichloride, for it is much less liable to coagulate the albumin in the secretions of the mucous membrane or of the blood. It must, however, be used with caution, and only in a strength of 1 in 2000 to 5000, for, as Roux has said, "there is already sufficient poison in the system of a patient suffering from diphtheria to justify us in hesitating to employ remedies which are in themselves capable of producing systemic effects of a toxic nature." This is also an objection to the use of mercury as an internal remedy, for its effects, in what may be termed reasonable or justifiable doses, are too slow to be depended on for the arrest of so acute and rapidly fatal a disease.

Among other germicides of a less active and irritating character we may mention carbolic acid, boric acid, and chinosol, the last of the strength of from two to five per cent. Irrigation of the mouth by means of a specially constructed syringe is in every case to be preferred to the old-fashioned and useless method of gargling; for the latter method, while it cannot be used by children and is seldom efficiently carried out by adults, not only necessitates the patient's rising from the recumbent position so essential to be maintained in diphtheria, but locally throws the muscles of the throat into irregular action, and thereby favors palatal and faucial paralyses.

Loeffler has made investigations into the power of certain substances to destroy quickly the bacillus of diphtheria, and has found that the salts of iron—to be administered also internally—are particularly active, especially when combined with certain essences, notably benzol, toluol, creolin, and metacresol. A solution containing these ingredients, and now known as "Loeffler's,"[1] has been tried in the writer's practice with uniformly good results, which have, however, been more evident in cases of pseudo-diphtheria than in the true bacillary disease.

When the nares are the seat of membrane every effort should be made to keep the nasal choanæ patent. Membrane must be removed by forceps or by a cotton-wool swab passed along the inferior meatus. The nares should be freely irrigated with some antiseptic and germicidal solution, followed by the application of an oily spray of menthol or thymol, and supplemented in certain cases by the use of an antiseptic ointment.

For the reduction of external inflammation and for the prevention of suppuration in the cervical and maxillary glands, the application of continuous cold by means of a "Leiter's coil" is of the greatest service; for by this means inflammation is reduced and separation of the membrane is materially favored.

Operative.—It is only necessary to mention tracheotomy and intubation; the latter procedure, primarily adopted by Bouchut in 1858, has been reintroduced and perfected by O'Dwyer, of New York, who first employed it for the relief of laryngeal dyspnœa in the year 1880. Intubation is said to be pre-eminently successful in infants under four years of age.

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[1] *Loeffler's Solution:* ℞ Menthol, 10 parts; toluol, 26 parts; solution of perchloride of iron, 4 parts; absolute alcohol, q. s. to measure 100 fluid parts. Mix. To be applied to the affected parts by means of a brush or swab.

When there is any obstruction to free nasal respiration from enlarged faucial tonsils or from the presence of adenoid growths in the naso-pharynx, the writer has had a uniformly favorable experience of operative measures for their removal. Indeed, the immediate improvement in the breathing, limitation of the exudation, and obviation of the necessity of opening the trachea are important advantages which cannot well be overlooked, and in this he has the support, among others, of Lefferts, of New York, and Macintyre, of Glasgow.

THE ANTITOXIN TREATMENT OF DIPHTHERIA.

It is probable that during the last few years no question in any branch of medical science has undergone more full discussion or more changes of opinion and pronouncement than that of the nature of the toxins—the specific and poisonous products—of bacterial organisms; and as a consequence it is not difficult to understand that the same may be said with regard to the substances which are supposed to neutralize their noxious effects,—in other words, the so-called antitoxins.

These toxins, to which the specific origin of tetanus, typhoid fever, anthrax, and other infectious diseases is admittedly due, have been well described and considered in a special article by Dr. Vaughan (page 93 of this volume); while the extent of our present knowledge especially relating to those of diphtheria has been already indicated in the section on the pathology of that disease in the present contribution. There is no occasion to go over the old ground. Suffice it to say that the labors of Behring, Buchner, Brieger, Fraenkel, Roux, Yersin, Sidney Martin, and others, valuable as they undoubtedly have been, are by no means entirely conclusive as to the exact constitution of the toxin of diphtheria. As a matter of fact, it is at present undetermined; and, as already hinted, there are few, if any, among the scientific investigators of this disease, either from a bacteriological or a chemical point of view, who have not found it necessary to correct or modify their original opinions as to the nature and action of the diphtheria toxin. This fact is amply illustrated in Dr. Vaughan's contribution.

Thus, we are not only ignorant of the exact biological process by which these poisons are elaborated, but chemists have up to now failed to determine their exact compositions either by analysis or synthesis; nor can we express them by any formula representative of their constituent elements. It naturally follows that, whatever the amount of success—and it has been considerable—which may attend the antitoxin treatment, it cannot be admitted that the remedy is founded on so accurate and scientific a basis as is often claimed.

As to the toxins, the most recent observations tend to prove that they are not of the nature of albumins, as first stated, nor are they formed by any chemical reaction on the proteid elements of either the medium used for culture purposes or from the proteids existing in the living being, both of which theories have been put forward. This incompleteness of data may in these days of logical therapeutics account for the disinclination of many sober-minded and experienced physicians and surgeons to subscribe fully to the claims of laboratory workers as to the value of antitoxic serum; and, until some more definite knowledge is arrived at, the treatment of diphtheria by its means must be considered purely on the basis of empiricism, a stand-point sufficiently solid, perhaps, for the majority of practitioners, whose sole aim is to heal, but somewhat insecure from the aspect of the "pure" science worker.

It is candidly admitted that, although yet by no means universally adopted, there are thousands of practitioners who do accept the antitoxin treatment of diphtheria on its curative merits, and in view of this fact there are good grounds to justify at least a more extended trial of the remedy in the future.

The author of this contribution is well aware that he has been credited with antagonism to the serum treatment of diphtheria from the date of its first introduction; but in point of fact he was quite willing to accept it enthusiastically, and was led to maintain an attitude of scientific scepticism only by the results of his own observations and experience. In view of what has presently to be said, he believes himself to be more than justified in having held this position.

For when the records of those observers who are in its favor are investigated, hardly one of them can be considered entirely satisfactory on comparison of the results with those of cases in which the older and classical treatment of diphtheria was adopted.

It is not unfair to add that, largely as the result of much severe criticism, the remedy has undergone so many radical modifications in its manufacture and method of administration as to constitute an entirely different remedy, both as to material, dosage, and adjuvant measures, from what it was when first introduced less than three years ago.

The *history* of the serum treatment of infectious diseases dates from the year 1860, when Pasteur first published the results of his investigations upon ferments. But this subject is elsewhere so ably dealt with by Dr. Vaughan that it is unnecessary to do more here than record the fact and date. Briefly, in December, 1890, Behring and Kitasato stated that if the blood-serum of an animal immunized against diphtheria by introduction of gradually increased doses of its toxins into the circulation were injected in sufficient quantity into another one, not only would this second animal be protected against an attack, but a cure would be effected by similar treatment of any other one the subject of the disease.

Roux, at the Congress of Hygiene and Demography held at Budapest in 1894, confirmed Behring's statement by his own experience, and recorded many successful cases in the human subject. The enthusiasm displayed in this communication and his subsequent demonstrations in Paris enforced the attention of the whole medical world, and this new discovery in the treatment of diphtheria was quickly accepted by the majority as an accomplished fact.

The writer commenced his experience of antitoxin by comparison of a series of one hundred cases of diphtheria treated with the remedy under his own personal observation in one of the Metropolitan Asylums' Board Hospitals (for infectious diseases) of the City of London during the first four months of the year 1895, with another series of one hundred cases treated on the old lines, and, of course, without serum, in the same hospital during the corresponding period of the previous year. The two series of patients were thus placed in exactly similar circumstances in regard to season, hygienic conditions, building, food, and nursing: it would be difficult, therefore, to have a more equal basis for comparison.

The serum with which these one hundred cases were treated was prepared by the British Institute of Preventive Medicine, the principal and certainly the most trustworthy source of all that was manufactured in England at that time.

The deaths in the two series proved to be equal in number—namely, twenty-seven—in each of the one hundred cases, and this mortality of twenty-seven per cent. in the cases treated with antitoxin was found to be practically identical with that of the same hospital during the previous two years out of a total of nearly two thousand five hundred cases.

As to the mortality at age periods, the figures were not altogether favorable to the serum treatment, nor was any advantage observed in regard to the following points, on each of which careful comparisons were made: (1) the prolongation of life in cases which ultimately terminated fatally; (2) the day on which the membrane commenced to separate; (3) relapses and re-formation of the membrane; (4) complications,—albuminuria, nephritis, anuria, cardiac failure, and paralytic sequelæ.

The only advantage of the antitoxin treatment which was at all apparent was a comparatively minute reduction of mortality in the laryngeal cases, a point which has certainly been made increasingly clear in other subsequent and independent records.

At a meeting of the Clinical Society of London, in December, 1894, the writer was the first in England—Hansemann had preceded him in Berlin—to make the suggestion that under the serum treatment patients seemed to be more liable to suffer from severe complications of diphtheria. He particularly instanced anuria, nephritis, and cardiac failure. For out of the first eight cases which were treated under his personal supervision with antitoxin, six died, all with suppression of urine, and in the kidneys of all the six there was post-mortem evidence of interstitial changes.

About this time Dr. Benda, of Berlin, reported that on necropsy of thirty-nine cases of diphtheria treated with antitoxin, only six were free from nephritis; three showed severe and twenty-five slighter parenchymatous inflammation.

Baginsky had also suggested (*Lancet*, January 2, 1895) that "attention should be paid to symptoms of cardiac failure, which seemed to be disproportionately frequent in the recent epidemic; for most of those dying under the [antitoxin] treatment died from cardiac asthenia." Before the introduction of antitoxin, a careful analysis by the writer of one thousand consecutive cases of diphtheria, which had also been treated in the same Metropolitan Asylums' Board Hospital as that in which his comparative study had been made, showed the proportion of those cases in which albumin was present in the urine to be 40 per cent. The mortality due to nephritis was 2.7 per cent. of the same series. When the report of the whole of the Metropolitan Asylums' Board Hospitals for the year 1896 was examined, it was found that every single complication noted therein occurred in a greater proportion in those cases which were treated with antitoxin, as the following table clearly demonstrates:

COMPLICATIONS.

Cases treated in 1896 with Antitoxin, 2764; without, 1411.—Total, 4175.

COMPLICATIONS.	WITH ANTITOXIN (TABLE XVI. IN REPORT). PERCENTAGE MORTALITY.	WITHOUT ANTITOXIN (PAGE 9 IN REPORT). PERCENTAGE MORTALITY.
Albuminuria	60.1	40.1
Nephritis	0.57	0.56
Paralysis	25.7	12.6
Pneumonia—lobar	0.67	0.35
Pneumonia—lobular . . .	3.1	1.48
Relapse of disease	1.8	1.06

The writer's prediction that the serum treatment would be liable to increase the proportion and the severity of the complications of diphtheria has thus, unfortunately, proved only too well founded, at least in the cases treated in London. More recent observations, especially those of Spronck, indicate that the action of antidiphtheria serum on an albuminuria is inconstant, which explains at once the position of those who affirm equally with those who deny any injurious effects of the serum in this direction.

Nor can we accept the often advanced excuse, that the reason why a greater number of serum-treated patients exhibit either severe complications or sequelæ is because a larger proportion survive, for, in truth, most of these complications—all except a portion of the paralyses—occur during the acute stages of the disease, and the frequency of paralytic sequelæ in serum-treated cases only goes to prove that the antitoxin has no power to prevent their development, a point now admitted by all serum-therapeutists.

In addition to these complications proper, so to speak, to the disease, we have to consider others "probably connected with antitoxin," and admittedly peculiar to and directly due to the administration of the serum.

The tables given in the report of the Metropolitan Asylums' Board for 1895 and 1896 include a sufficient number—something like fifty per cent.—of these in the shape of skin-eruptions, joint-pains, and local abscesses; and granted that these may not be vitally serious, they are at least very inconvenient, and are largely responsible for retarded convalescence.

When we refer to reports on the antitoxin treatment of diphtheria for the two complete years 1895 and 1896, published by the superintendents of these hospitals, we find that a claim is made for a considerable reduction in the combined general mortality—that is, those treated with and those treated without antitoxin—" by the use of the serum," and this is supposed to be proved by statistical tables; but, unfortunately, a critical analysis of these and the basis of comparison on which they are founded do not show any improvement in the results in regard to a lowering of the mortality under serum treatment.

Percentage Mortality.

YEAR.	WITHOUT ANTITOXIN.	WITH ANTITOXIN.	COMBINED.
1894 (before) . . .	29.6		
1895	13.4	28.1	22.5
1896	10.8	25.1	20.8

These figures show that the reduction in the mortality is much greater in those cases which were not treated with antitoxin, and this reduction accounts for most of the (combined) lower death-rate in the last two years. The answer that is always offered to this is that the antitoxic serum was employed only in severe cases, with the inference that those not treated with serum were of an extremely mild character. Nevertheless, all patients admitted to the hospitals in a hopeless and moribund condition, quite beyond the reach of any treatment, to whom antitoxin was not administered, —amounting to nearly fifteen per cent.,—were included in the list of cases in which serum was not employed, but were excluded from those in which it was. And although by this means the mortality in the non-antitoxin class is unduly swelled, it is still lower than that of the serum treatment.

Moreover, the basis of classification into mild and severe cases of diphtheria is, to say the least, almost universally fallacious, for no case can be said to be of a mild nature until the termination of the attack. Especially is this the fact in children under five years of age,—a period when the disease is notoriously fatal. Nevertheless, in the reports under consideration not only were nearly a third of the cases of this age-series treated without antitoxin, but their death-rate was lower as compared with those injected.

To summarize the writer's experience: the particular objections to a comparison of the old with the new serum treatment, in his judgment, are:

1. The exclusion from the tables of cases not treated with antitoxin which were admitted to hospital in a moribund state, and the inclusion of them with those to whom the serum was not administered; these should at least be treated as neutral.

2. The question of dosage. This ought to be considered with reference (*a*) to the actual amount of fluid given and (*b*) with regard to the very vari-

able proportion of the active ingredients contained in different samples. On this simple but necessary and vital point three stages have already been arrived at. Claims for recoveries were advanced (at least in Great Britain) in the earliest days of the serum treatment, in which only ridiculously minute doses—a few minims—of the remedy were administered; then the dose, still given by measurement of fluid, became equally ridiculous when considered in the light of still more recent pronouncements. The actual quantity of the serum recommended for injection is now considerably less,—a concession to the criticisms that the large quantities of blood-serum injected might of itself be noxious,—but its strength, as measured by immunizing units,—the newest basis of calculation,—is enormously increased, so that either the first and second doses were quite insufficient or the present ones are equally excessive. All the same, the tendency is to still fuller doses, with a corresponding increase in the concentration of the serum so as not to increase the bulk of the fluid introduced.

3. The statement that diphtheria antitoxin is useful only in cases of pure diphtheria—that is, where the Klebs-Loeffler bacillus is unassociated with other organisms of a maleficent nature—would, if still maintained, limit the administration of the remedy to a very small proportion of cases of diphtheria. But we now almost daily read reports in which antitoxin is said to have effected a cure not only in complex diphtheria, but in many cases in which bacteriological examination has altogether failed to demonstrate the presence of that bacillus which is not only specific of the disease, but also asserted to be the active principle of the remedy. The *reductio ad absurdum* would seem to have been reached in the recommendation and, indeed, acceptance of diphtheria antitoxin for the cure of ozæna (atrophic rhinitis), a disease as dissimilar from diphtheria as can well be imagined.

This objection trenches closely on the mode of action of antitoxin, a point which will presently receive separate attention.

4. In the early days of antitoxin treatment all other measures, either general or local, were declared to be not only useless, but even harmful to the success of the serum. Alcohol was also prohibited. Witness, by contrast, the careful attention and strict regulations which at the present time are enforced as to the topical use of antiseptics and germicides, the internal administration of drugs similar to those formerly prescribed, and the return to free exhibition of alcohol as an integral part of the treatment.

Lastly, the early recognition, especially by Roux, that the antitoxin treatment absolutely demands for its success the most rigorous attention to all matters relating to general hygiene, *personnel* of the patient, and unwearying watchfulness of nursing, must be held to play some part, and not a minor one, in the improved results.

For all the foregoing reasons it should be conceded that antitoxin serum in the treatment of diphtheria has resolved not into a specific independent of other remedies, but as a useful adjuvant to those other measures which have come to be regarded as classical in the combating of this malady.

Having thus recorded this personal experience as well as that of the most important representative hospital organization in this country (England), notwithstanding that the inferences are different, it has to be frankly admitted that far more favorable reports of the results obtained from serum treatment of diphtheria have been received from the Continent, albeit some have been generally qualified by cautious reservations.

But to America, the country where an organized bacteriological examination of the suspected diphtherial exudation was first introduced, must we turn for information that is accurate, impartial, and, it may be added, encouraging. We will take as at once the most complete and as a type of the whole the records of the American Pediatric Society, at a meeting of which, held in Montreal in May, 1896, a report was presented of the results obtained from a collective investigation of five thousand seven hundred and ninety-four cases of diphtheria treated with antitoxic serum in private practice.

Reports were received from no less a number than six hundred and fifteen different practitioners, and the diagnosis of diphtheria was confirmed by bacteriological examination in about two-thirds of the whole number of the cases.

The mortality in the total number of cases treated was 12.3 per cent. Of these, however, two hundred and eighteen were recorded as moribund on admission or as having died within twenty-four hours from the time of injection; and if these moribund and (presumably) hopeless cases be excluded, the total mortality is reduced to 8.8 per cent.

The report states that the mortality was lower the earlier the injection was given, for after the third day it rose rapidly. Nevertheless, "it must be said that striking improvement has in some cases been seen even when the serum has been injected as late as the fifth or sixth day. The duration of the disease, therefore, is no contra-indication to its use." The figures show also improvement in the results obtained in adult cases.

The laryngeal cases were twelve hundred and fifty-six, or 37.5 per cent. of the whole number treated. In six hundred and ninety-one no operations were performed, and five hundred and sixty-three recovered, giving a percentage of 16.9 of the whole number and 44.8 for the laryngeal cases. (The writer's analysis of one thousand cases of diphtheria treated prior to the days of antitoxin gives a mortality of 44 per cent. in this class.)

A second report from the same American society for a period of eleven months ending April 1, 1897, furnishes statistics of seventeen hundred and four cases of laryngeal diphtheria, with three hundred and sixty deaths, a mortality of 21.12 per cent., thus showing a progressive improvement in results, with, presumably, corresponding improvements in matters of administration.

Of this number thirteen hundred and six cases were not subjected to operation, with a death-rate of 17.8 per cent.; while that of the six hundred and sixty-eight cases in which an operation—tracheotomy or intubation—was performed was 27.24 per cent., or about half of that formerly obtained by the most successful operators.

The society's report claims that, whereas before antitoxin was used only 27 per cent. recovered, now even in the most severe cases of diphtheria treated with serum the recoveries are 73 per cent., an increase of fully threefold. With regard to complications and sequelæ, "the proportion of cases of broncho-pneumonia, 5.9 per cent., was very small. . . . There was little evidence to show that nephritis was caused in any case," concerning which it may be suggested that if these highly concentrated solutions were employed, the amount of increased blood-pressure leading to renal distress would naturally be less than in the early days of serum treatment, when the fluid was weaker in antitoxic strength but larger in amount.

In three thousand three hundred and eighty-four cases treated with antitoxin, paralytic sequelæ occurred in three hundred and twenty-eight, or 9.7 per cent.,—a low proportion,—the average having been formerly something like 14 per cent. The report says,—

"The proportion of cases in which paralysis occurred was rather lower, but the frequency of fatal cardiac paralysis (thirty-two cases) was high." This last is surely an important admission, and not quite consistent with another statement contained in the same report, that "in two or three instances the serum was believed to have acted unfavorably upon the heart, but in a large number there was a distinct improvement in the heart's action." Again, "in three cases the injections were believed to have hastened death."

Of the six hundred and fifteen doctors who contributed to the returns of the American Pediatric Society "more than six hundred pronounced in favor of the serum treatment, the great majority being enthusiastic."

Although these reports furnish some items of evidence calculated to qualify the laudatory results, yet, allowing for some slight exaggeration due to enthusiasm, it is impossible to deny that there is ample testimony in favor of the efficacy of the antitoxin treatment of diphtheria, and that its benefits have in the United States greatly surpassed those in London. It must not, however, be assumed that there has been no opposition to its adoption in the United States or on the European Continent; for, indeed, there is hardly a scientific centre that has not furnished writers of repute who either express themselves as definitely adverse to the treatment or deprecate the extravagant enthusiasm of some of its advocates.

A difficulty in the way of its general adoption still exists in the fact that, in spite of every effort to establish a universal standard of strength of the serum, up to the present time no definite decision has been arrived at, nor can we rely on the uniformity of any particular sample of antitoxin, wherever it may be prepared.

The results of *The Lancet* commission "on the relative strengths of diphtheria antitoxic serum," published in July, 1896, prove that the various specimens obtained from different manufacturers, home and foreign, exhibit great discrepancies between the amount of antitoxin and the "immunizing units" contained in the several samples, when carefully analyzed, with those which are notified on the labels attached to the samples submitted for trial, while there was only one firm (that controlled by Behring and Ehrlich) which supplied serum with any real, albeit not constant, approach to conformity with the strength stated on the label. A sample from another laboratory (an English one) contained no antitoxin element whatever and, so far from being innocuous, was crowded with infectious micrococci.

In a leading article which appeared so recently as August 14, 1897, the editors of *The Lancet* deem it necessary to repeat the conclusion of the commissioners that "the astounding variation in the quality of the serum supplied by different makers and the puzzling way in which the antitoxin from certain sources varied from time to time were constantly making themselves evident, and that the results obtained by the use of these serums in the treatment of diphtheria should have varied is what must be expected if the antitoxin does exert any influence at all in the course of the disease."

On these grounds the editors of *The Lancet* think it "intolerable that the medical man should be kept in ignorance of the strength of the serum which he is making use of, when the want of such knowledge may be but too often a matter of life or death to his patient."

This criticism does not, of course, apply to the same extent as was formerly the case, but "there is still evidence that there is a real necessity for a comparison of the serums that are made."

The editors continue by saying that "there has been a marked rise in the antitoxic value of the serum that has been put on the market in this country, which may now be held to compare very favorably with the antitoxic serum prepared abroad. In fact, with the exception of the strongest forms prepared in the German laboratories, it may be said to be equal to any on the market." But so long as there is an exception it is not possible to compare the results of the serum treatment in different countries. "The protest—for protest it was—that the serums obtained in this country were not only of comparatively low antitoxic value, but that they differed very greatly from one another, and were not sent out, even from the same establishments, of constant strength, with the result that, as there was not strength marked on each sample bottle, the practitioner was left in grave doubt as to the dose to be administered and the antitoxic value of each dose," has borne satisfactory results, and it is believed "that most of these objections have now to a great extent been removed." With this we agree. There has not, however, so far as we know, been published any independent testimony as to the standard strength of the serum manufactured in the New York laboratory or in that of the English College of Physicians and Surgeons, which represent those at present in largest use in these two countries, and there cannot be a doubt that these discrepancies in the quality of the remedy would go far to account for the variations of the success attained in different countries; nor can we see how this state of things is likely to be remedied until the state undertakes to regulate the output of antitoxin in our own country according to its value, as is ordained in the two continents of Europe and America.

But there is another difficulty: the variations in the methods of testing the actual strength of the serums and, consequently, of comparing their therapeutic value caused much difference of opinion as to their relative merits, and, says *The Lancet*, "Professor Ehrlich, who has been working at the subject for the last four or five years, has only recently [August, 1897] published a method differing very materially from his original method, by means of which he is able to get more accurate and constant results than he has hitherto been able to obtain."

Nor have we yet finished our list of objections, and there still remain matters of controversy which require discussion. One important point is that, allowing that the injection of antitoxic serum is of benefit, there is still great doubt as to the exact mode of its action.

We have shown that its value has not been limited to those cases characterized by the presence of the specific bacillus; indeed, it is said to have cured cases which were not of bacillary origin at all; nor is its beneficial effect less evident in others in which a large amount of the noxious influence of the malady is due to the presence of hardly less dangerous micro-organisms; for example, a diphtheria implanted on a scarlet-fever throat,—a uniformly mixed form of the disease,—in which cases the serum treatment has been so especially successful. We are therefore driven to the conclusion, and in support thereof we may quote the names of Soerensen and Oertel, that it is probable that the serum supplies dynamic force, enabling the patient to withstand the prostration due to the toxæmia, and thus—admitting that the powers of assimilation in children vary—we can understand why large doses are required by some, and why comparatively small doses produce noxious effects in others.

We can also understand the better results obtained in those cases which are earliest treated, although this is a point which has been insisted on with regard to serum with a *naïve* oblivion of its application to every treatment of every disease.

We can likewise appreciate that the serum may be beneficial at a date prior to that at which brandy or strychnine would be indicated, and it is not impossible that an anatomical reason could be given why the larynx when attacked with diphtheria is more susceptible to improvement under the serum treatment than the fauces.

This suggestion as to the value of albumin *per se* is strengthened when we remember that antitoxin in which the bulk has been reduced to a minimum by desiccation has been found to give less satisfactory results than those obtained by the use of liquid serum. We would not venture to contradict those who may ascribe these differences of result to a deleterious effect on the antitoxin produced by the desiccating process, for, ignorant as we are of the exact chemical nature of the toxins and antitoxins, no one is in a position to dogmatize on this point; but it may be remarked that the *antidiphtherin* of Klebs, a non-albuminous fluid obtained from cultures of the bacillus, has, after due trial, been abandoned as of no value. We are, indeed, almost justified on all these grounds in ignoring the whole question of an antitoxic influence, especially when we once again call to mind that the proportion of cases claimed for benefit under the treatment is considerably greater than those in which the Klebs-Loeffler bacillus is found unmingled with cocci,—these last being declared by Loeffler to be not favorably influenced by antitoxin; and that in no case have the actual toxic results of diphtheria, such as cardio-respiratory paralysis, nephritis, and neuroses, been in any way diminished—some statistics, indeed, show that they have been increased—in those patients who survive injections.

In view of all these facts, it is once more urged, as Soerensen has done, that at least equal benefit to that obtained by antitoxin might result from the injection of normal saline solutions, or, although that is more hazardous, by the injection of simple sterilized blood-serum, as advocated also by Grawitz.

The last points to be considered are those of *administration* and of *dosage*. As to the first, the whole procedure is one well understood by every well-educated student, and it is not necessary here to occupy space by dilating at length on the urgent necessity for absolute cleanliness and general antiseptic precautions, nor is there any reason to be advanced for a preference of the flank to the abdomen as the site of injection.

For reasons already considered, it is extremely difficult to define the exact dose of the serum, and the writer's remarks on this head will be for the most part confined to quotations from different authorities.

According to Roux, speaking from the Pasteur Institute, his first recommendation was to administer twenty cubic centimetres (representing about five fluidrachms) to every patient above one year of age on first admission to the hospital. In very severe cases he gave as much as thirty cubic centimetres. In 1895 the dose of serum prepared by the British Institute of Preventive Medicine was twenty cubic centimetres as a first injection for an adult and ten cubic centimetres for a child under one year, as employed at the Metropolitan Asylums' Board Hospitals. During this year the average dose given was 41.2 cubic centimetres. The superintendents report that they "think the best results may be obtained by giving a dose of one thousand Behring's immunization units every twelve hours for the first twenty-four, thirty-six, or forty-eight hours (that is, of two, four, or six drachms to each patient), according to the gravity of the case, and, if necessary, a subsequent injection of half the amount daily for such time as the exudation may remain adherent."[1]

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[1] According to Variot, the latest writer on the subject, antitoxin is apparently still administered in France by the cubic centimetre and not by the unit.

In the reports of the same institution for the year 1896 the average number of injections given had, however, been greatly reduced, and amounted to only 2.3 per patient.

The serum used during this year was of higher immunization value than that used in the year previous, but the average dose given at one injection was nineteen hundred and eighty-three and three-tenths Behring's units. For ordinary mild cases two thousand units were advocated, but the report states that, "as regards units, there is, as far as we know, *no limit to the number that may be injected.*

"With the strongest serum [four thousand units in ten, and in a few instances in five, cubic centimetres] eight thousand units can easily be given in one injection."

From these two reports it will be seen how enormously the immunizing dose of serum has been increased in the period of only twelve months.

In the report of the American Pediatric Society for 1896 the dose for a child over two years in all laryngeal cases with stenosis and in all severe cases is stated to be from fifteen hundred to two thousand units for a first injection, to be repeated in from eighteen to twenty-four hours if no improvement is evident, a third dose being given, if necessary, after a similar interval.

In their second report (May, 1897) they recommend the selection of the most concentrated form of the antitoxic serum. In all cases of laryngeal diphtheria in patients of two years of age and over, the first dose should be two thousand units, and from eighteen to twenty-four hours after a second dose of two thousand units if no marked improvement occurs, and a third dose of two thousand more units twenty-four hours after the second if there be still no improvement in the patient's condition.

It will be seen, therefore, that in the practice of our American *confrères* also there is a tendency to increase the dose of serum, although not to such a great extent as in Germany, whence we learn "that as much as ten thousand units may be, and have been, used beneficially and without apparently doing harm." The recommendation to employ a more concentrated solution must not be overlooked when we make a comparison between the new and the old calculations for administration, for if more antitoxin is now introduced the bulk of fluid serum is less.

Another question often asked is, Should antitoxin be injected before any bacteriological confirmation has been made? True, it is stated that it is innocuous to persons not suffering from the disease; on the other hand, it is a curious fact that recovery is claimed for it in many cases which bacteriological examination has proved to be pseudo-diphtherial. But can it really do no harm? The recorded cases in which fatalities admittedly due directly to the procedure have occurred are both too numerous and too wide-spread to leave any doubt that the serum is by no means harmless, whether it be administered to cure the disease or as a prophylactic.

Granted that these cases are small in number as compared with those in which a cure was effected, it is by many considered a grave responsibility to employ a remedy that may by any chance lead to fatal results.

The writer has no need to state in which direction his answer would be, but in fairness he records the fact that the administration of antitoxin for the purposes of prophylaxis has been viewed with high favor in the United States, especially by Dr. Hermann Biggs, to whom not only America but the whole civilized world is largely indebted for his splendid services in the cause of bacteriological science and serum-therapy.

THE HYGIENE AND PROPHYLAXIS OF DIPHTHERIA.

The preventive measures to be adopted during the run of a case of diphtheria are simply those which are required for the routine treatment of any other infectious or contagious fever, and a very few words will meet the question, for it has been so excellently detailed in a former volume of this Cyclopædia that there is little to add.

The preventive points to be insisted on here as being of special application to diphtheria are the prompt surgical treatment of every case in which the patient is the subject of enlarged faucial tonsils, adenoid growths in the naso-pharynx, or other causes which lead to mouth-breathing in children; an early examination of the throats of those at school or of families whenever an epidemic of sore throat occurs; and the rigorous segregation of those attacked.

Finally, although it cannot be denied that, so far as the city of London is concerned, diphtheria is steadily increasing, if not in fatality at least in frequency, and in an inverse ratio to the general improvement in drainage and sanitation, attention to these matters must not be neglected, for, as before quoted, "not to recognize the frequent filth origin of diphtheria may in practice be as disastrous as to ignore its infectiousness."

SCARLET FEVER AND MEASLES.

BY FLOYD M. CRANDALL, M.D.

SCARLET FEVER.

SCARLET FEVER is an acute, infectious, contagious disease, typical cases of which present the following features. After a period of incubation of from two to four days there is a sudden onset of sore throat, vomiting, and fever; within twenty-four hours an eruption appears on the neck and rapidly spreads over the body and continues for about six days, when it terminates in desquamation, which continues for three weeks or longer. The complications are numerous and grave, and render the disease one of the most fatal of childhood.

Etiology.—The chief predisposing cause is age. I have seen an undoubted attack of scarlet fever in an infant of one week, but it is rare under one year. From that age to five years the susceptibility steadily increases; after eight years it diminishes, and is very slight during adult life.

That scarlet fever is an infectious disease does not admit of doubt, but the specific germ has not yet been discovered. It has, however, been fully demonstrated that streptococci play an important *rôle* in the causation of many of the symptoms, but they do not cause the primary disease. They are the cause of the pseudo-membranous exudations of the throat, the otitis and adenitis, and probably the nephritis, pneumonia, and joint-lesions. They are so constant in their presence and so active in the causation of the more serious symptoms that they must be regarded as important factors in the production of the clinical picture which we know as scarlet fever. The disease as it commonly appears is a mixed infection, the more malignant and fatal symptoms being due not so much to the primary as to the secondary infection. Staphylococci and diphtheria bacilli are sometimes found in conjunction with the streptococci. The specific germ exists in the blood, for inoculation of serum into susceptible animals produces a typical attack of the disease. It is also found in the various secretions, as shown by their power to generate the disease. Susceptibility of the human organism to scarlet fever is less decided than it is to measles, exposure being far less frequently followed by illness.

Sources of Infection.—Scarlet fever may be contracted by direct exposure or through intermediate infection. The chief source of direct infection is the patient himself, but the area of infection is limited to a few feet; hence in direct exposure alone close contact is necessary. Infection by the breath is doubtful, but it is not certain that it may not occur. The scales thrown off during desquamation are extremely infectious. The retention of the scales by clothing, bedding, and the walls of rooms is one of the most common causes of infection. The various discharges of the body, particularly the purulent secretions from the throat, nose, and ear, are also very infectious. Scarlet fever is spread by indirect infection more frequently than any other disease except diphtheria. Its specific micro-organism is more tenacious of life than that of any other disease except, perhaps, small-pox. Hence the disease may be conveyed long distances by clothing, carpets, bedding, books, and toys. It may be conveyed in the fur of cats and dogs, and it is probable that these animals may themselves suffer from the disease. The conveyance of scarlet fever by milk and other articles of food is undoubted. There are numerous authentic cases in which the disease has been conveyed by letters written by hands in the stage of desquamation. Infection through a third person is not infrequent. Epidemics of scarlet fever usually spread very slowly as compared with those of measles.

Period of Incubation.—The incubation period is very short. The extremes in authenticated cases range from a few hours to fifteen days. In eighty-four per cent. of cases Holt found the period to be less than six days, and in sixty-six per cent. between two and four days.

Period of Infection.—While the period of incubation is short, the period of infection is long. The disease is not infectious during the period of incubation, but it may be so from the appearance of the first changes in the throat. The most actively contagious period is at the height of the febrile stage, on the third, fourth, and fifth days. The infectious power then diminishes, but increases again during the stage of desquamation. The period of contagion continues until the last evidences of desquamation have disappeared. It is an error, however, to accept this as the only measure of the period of isolation. The purulent discharges from the throat, nose, and ears are capable of infecting others, and isolation should not be relaxed until they have disappeared.

Pathology.—The lesions of uncomplicated scarlet fever are confined to the skin and throat. The lesions of the skin are those of acute dermatitis. The production of epithelium is greatly increased during the acute stage, which results later in profuse exfoliation of the superficial layers. In addition to this, according to Neumann, there is also an acute development of exudative cells, particularly among the ducts and follicles, which easily reach the epithelial surface,—a fact which accounts for the great infectiousness of the desquamating cells. The changes in the throat of uncomplicated scarlet fever are catarrhal in nature and are an essential part of the disease. A tenacious semi-membranous exudate not infrequently appears.

The croupous and diphtheritic pseudo-membranes must be regarded as complications, and will be so considered. The post-mortem findings in those who die early in the disease are confined to the throat, the lesions of the skin being but faintly marked. After the disease has been in progress for several days changes are found in the viscera and other tissues. They are, however, the lesions common to all febrile and septic diseases.

Symptoms.—Scarlet fever is one of the most variable of diseases in its clinical manifestations. From the attack so mild that diagnosis is difficult to the fiercely malignant form, we see every possible grade of severity. Notwithstanding this variability of type, the majority of cases pursue a fairly uniform course, and may with propriety be called ordinary cases. Other types may be denominated as mild, severe, and malignant.

Ordinary Type.—The *invasion* of scarlet fever is usually sudden, and is marked by vomiting, fever, sore throat, and rapid pulse. In older children a chill is sometimes the first symptom; in younger children, a convulsion. The vomiting is usually repeated several times, and is not accompanied by nausea. The child may be restless, with flushed face and bright eyes, but is more commonly dull and heavy. The intensity of the period of invasion is usually indicative of the severity of the attack, though this is a rule subject to many exceptions. Intense symptoms at the outset are usually followed by a severe attack, but mild initial symptoms are sometimes followed by very grave ones as the disease progresses.

Temperature.—The normal temperature of scarlet fever is high. It is frequently found to be 103° F. at the first visit, and may reach 104° or 105° F. on the first day. A temperature on the first day above 104½° F. gives warning of a severe attack, below 102° F. of a mild attack. The highest point is commonly reached at the height of the eruption on the third and fourth days. It then begins to subside, and becomes normal at a varying period, ranging from the ninth to the fifteenth day. The fever is frequently remittent, and in mild cases almost intermittent, in character. An abnormally rapid pulse is quite characteristic of scarlet fever. It is often 150 on the first day, and continues rapid throughout the disease.

Angina.—Sore throat is one of the first symptoms complained of by the child. The fauces, tonsils, and pharynx are of a uniform bright red color, and on the hard palate numerous dark red macules may be seen. In mild cases the throat symptoms may be very slight; in very severe cases the tonsils may be studded with follicular spots or smeared over with a tenacious exudate closely resembling a pseudo-membrane. There is frequently a discharge from the nose which may consist of clear, tenacious mucus or muco-pus. The glands at the angle of the jaw become enlarged. After the fourth day the tongue, which is at first coated, often becomes clean and red with prominent papillæ. This appearance, following a coated tongue, is peculiar to scarlet fever, and is known as the strawberry tongue.

The *eruption* usually appears within twenty-four hours after the initial vomiting. It is not infrequently seen within twelve hours, is sometimes delayed for thirty-six hours, and in rare cases to the fourth or fifth day. It is usually well developed during the second day of its appearance. It continues with little change for from four to six days, when it gradually subsides in the order in which it came. It generally appears first over the front of the neck and upper part of the chest, and spreads rapidly until the whole body is covered. It consists of minute points of bright scarlet color closely grouped together on a slightly reddened skin. They become confluent in places, forming bright scarlet patches; but over most of the surface they remain discrete throughout. Being hyperæmic in nature, the rash disappears on pressure, leaving for a perceptible time a white spot. An eruption of very fine vesicles is seen in rare instances. There is usually at first much itching or burning of the skin.

Desquamation is one of the most characteristic symptoms of scarlet fever. It rarely begins before the fifth day of the eruption, and is frequently delayed until the seventh or eighth. It appears first on the neck and between the fingers. At first it occurs in fine branny scales, but soon assumes the form of large lamellar scales. Sometimes the skin can be peeled off in long strips. It continues from ten to thirty days, and is most persistent where the skin is thickest. It frequently continues about the fingernails after other portions of the body are clear. When the skin has received careful attention the desquamation is sometimes almost imperceptible. In rare instances a second desquamation occurs.

The Urine.—During the febrile stage the urine becomes scanty and high-colored, and frequently contains a slight amount of albumin, and sometimes blood and hyaline casts. Except in the more severe forms, suppression is rare and dropsy still more so. These symptoms usually subside as the fever falls, and rarely prove serious. The more serious kidney lesions occur later, and will be considered as a complication.

Mild Type.—Scarlet fever is sometimes so mild as to render diagnosis very difficult. As a rule, there is an onset of vomiting, fever, and sore throat, as in the ordinary type, but none of the symptoms are urgent. I have seen an undoubted case in which the temperature never rose to 101° F. The eruption is often very faint, and may not appear on the face. It may, however, be bright and distinctive for twenty-four hours and then fade away so rapidly as to have disappeared by the fifth day. In rare cases it is an evanescent rash which disappears entirely within twenty-four hours. Complications are rare, but nephritis is a common sequel, due in many cases to exposure and lack of care. Owing to lack of isolation, the patient may become very dangerous to others. It is by these mild cases that the disease is sometimes sown broadcast.

Severe Type.—During the invasion of this form of disease, the symptoms are those of the ordinary type intensified. Not only are the symptoms severe, but the various stages are prolonged. The fever may continue for twenty days or more, and the stage of desquamation may continue for even a longer time. A fatal termination is common, death occurring usually during the second week. It may, however, occur much later, as the result of nephritis or other late complication. The chief peculiarity which distinguishes this from the ordinary type is the presence of septic symptoms due to streptococcus infection. The throat is usually the first to show the evidences of streptococcus invasion. On the third day, and in some instances on the first or second day, a membranous exudate appears on the tonsils and soon invades more or less completely the pharynx and naso-pharynx. A purulent nasal discharge appears, and the lymphatic glands at the angle of the jaw begin to swell, the cellular tissues being so involved as often to cause immense enlargement. The Eustachian tubes are involved, and purulent otitis media follows. The urine contains albumin, and perhaps blood-cells and hyaline and epithelial casts. The pulse becomes weak and rapid; there is low delirium or stupor, and the child refuses nourishment. Broncho-pneumonia may develop, with, perhaps, other signs of septic infection. The child may die from exhaustion or sepsis, but sudden death is not uncommon. Others, after overcoming one complication after another, slowly recover after a long and tedious convalescence.

Malignant Type.—This type is, fortunately, rare. It begins suddenly with convulsions and hyperpyrexia. The scarlatinal poison may be so intense as to cause death within twenty-four hours without the development of characteristic symptoms. More commonly death does not occur before the third or fourth day, the patient being comatose or delirious. The nervous symptoms are so marked that some writers have given to this type the name of cerebral scarlet fever. In a case of my own the initial symptoms were convulsions, hyperpyrexia, and hæmaturia.

Surgical Scarlet Fever.—Patients who have undergone surgical operations are unquestionably very susceptible to scarlet fever, and contract the disease on the slightest exposure. The scarlet fever of surgical cases, however, is not essentially different from the usual disease. It is no doubt true, as Osler has shown, that the eruption which has frequently led to a diagnosis of scarlet fever is nothing more than the red rash of septicæmia.

Complications and Sequelæ.—*Angina.*—A catarrhal condition of the throat is normal to scarlet fever, but membranous exudates are not essential to it. The true nature of these exudates was long a subject of discussion which has been settled by the bacteriologist. With few exceptions, the angina of the early stages is pseudo-diphtheria;[1] that of the later stages, true diphtheria. While primary pseudo-diphtheria is a very mild disease, the death-rate being rarely over five per cent., secondary pseudo-diphtheria is very dangerous and fatal. In scarlet fever it is particularly so. The membrane may appear in the throat on the first or second day, but it is not usually seen before the third day. It may be confined to the tonsils, but frequently spreads rapidly, so as to fill the whole throat and naso-pharynx. It shows a rather marked tendency to invade the ears and nose and to shun the larynx. It reaches its height about the sixth or seventh day. It frequently presents all the local characteristics of diphtheria, together with the general symptoms of septicæmia. It is usually impossible to make a differential diagnosis between diphtheria and pseudo-diphtheria from clinical evidence alone. The exciting cause of this membranous inflammation is the streptococcus pyogenes.[1] It is occasionally associated with the staphylococcus aureus or albus.[1] It not only occurs in the pseudo-membrane and the tissues underneath it, but is found in the blood in large numbers. Through the agency of the toxins which it generates, it is unquestionably the direct cause of the complications and general septicæmia. The pseudo-membranes which appear late in the disease are usually associated with the Klebs-Loeffler bacillus, and are, therefore, diphtheritic. Diphtheria is, in the fullest sense of the word, a complication, and is not an essential symptom of scarlet fever. In rare cases gangrene occurs, involving the various tissues of the throat and being accompanied by extreme prostration, and almost invariably resulting in death.

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[1] Illustrated in the article on Diphtheria, p 198.

Otitis is a very frequent complication, and in its results is one of the most serious, as it is a common cause of deaf-mutism. It results from extension of the inflammation from the throat to the Eustachian tubes. The tendency to ear involvement varies greatly in different epidemics, but it is always more common in young patients. It does not usually occur until the second week, and generally involves both ears. Although perfect recovery frequently results, the process is prone to be a destructive one, and to result in necrosis and long-continued suppuration. It sometimes leads to a rapidly fatal meningitis.

Adenitis and *cellulitis* are common results of streptococcus invasion of the throat. Not only are the lymphatic glands themselves enlarged, but there is more or less extensive inflammatory œdema of the surrounding tissues. Streptococci are found in great numbers, both in the nodes and in the œdematous tissues around them. The cellulitis may be so extensive as to extend from ear to ear. Enlargement of the nodes may be detected during the first week, but serious cellulitis does not, as a rule, occur until later in the disease. Suppuration and extensive sloughing, or even gangrene, may occur, and is a complication of the utmost gravity.

Joint-Lesions.—Although acute articular rheumatism sometimes occurs as a complication of scarlet fever, the joint-affection often called scarlatinal rheumatism is in most instances a synovitis. It is a mild condition, and is frequently confined to the wrist. It appears early in the second week, continues for three or four days, and disappears, suppuration being very rare. It is seldom seen under four years, and its frequency varies in different epidemics. Pyæmic arthritis occurs in extremely rare instances and affects the larger joints, the lesions being multiple. Marsden has recently offered the following excellent classification of the joint-affections of scarlet fever:

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[1] Illustrated in the article on Diphtheria, p. 200.

(1) synovitis, (2) acute or chronic pyæmia, (3) acute or subacute rheumatism, (4) scrofulous disease of the joints.

Nephritis.—Albumin may be found in the urine during the acute stage, but it is febrile albuminuria, due to simple degenerative nephritis, which subsides as the temperature falls. In the septic type of disease kidney lesions may occur, to which the term septic nephritis has been given. The urine contains albumin, but blood and casts are not necessarily present, neither do the rational symptoms of uræmia appear. The kidney affection is essentially the result of the septic infection.

The most common kidney lesion is post-scarlatinal nephritis, and is, pathologically, a diffuse nephritis. It develops during the third or fourth week, and may follow a severe or mild attack. If it follows a severe case there may be no interval of apyrexia between the primary attack and the onset of the nephritis. It may be so mild as almost to escape notice, or it may be so severe as to cause speedy death by uræmia. Between these extremes is every variety of clinical type. Recovery may be complete, or it may be incomplete, the patient dying with chronic Bright's disease months afterwards. The first symptom to be noticed is usually œdema of the face, which is frequently accompanied by feverishness at night and restlessness. Dropsy and all the characteristic symptoms of acute nephritis rapidly develop. The urine usually shows a small amount of albumin for a number of days before the advent of definite symptoms. As the disease develops the urine becomes scanty and high-colored, and may be completely suppressed. It contains a large amount of albumin, and is loaded with blood-cells and casts. The first evidence of albumin after the second week should be a warning of danger, and should receive immediate attention.

Pneumonia, although it is usually found at the autopsy in patients who have died with septic symptoms, is frequently not recognized before death. It is a less common complication than in measles. Endocarditis and pericarditis, though uncommon, are sometimes encountered. Degeneration of the heart-muscle occurs in prolonged cases, as in all febrile and septic diseases. During the height of the disease murmurs are frequently heard, which disappear as the active symptoms subside. Permanent organic lesions sometimes develop in conjunction with the late kidney complications.

Second attacks of scarlet fever are extremely rare. They no doubt sometimes occur, but in most supposed cases there has been some error in diagnosis. Relapses are more common than second attacks. They result from auto-infection, and usually occur during the second or third week.

Prognosis.—The mortality of scarlet fever varies greatly with the age of the patient and with the character of the epidemic. The younger the patient the greater the mortality. Holt, after a study of a large number of American and European cases, concludes that the general mortality may be assumed to be from twelve to fourteen per cent., while under five years it is from twenty to thirty per cent. The majority of fatal cases occur in children under seven years. The first cases of an epidemic are invariably more fatal than the later ones. Prognosis is rendered unfavorable by the appearance of the following symptoms, the gravity being in proportion to their severity: violent onset, high temperatures, convulsions, extensive pseudo-membranous or gangrenous pharyngitis, diphtheria, croup, pneumonia, excessive cellulitis, superficial gangrene, nephritis, and exhaustion with general septic symptoms.

Prophylaxis.—In few other diseases are preventive measures more effectual than in scarlet fever. In view of the gravity of the disease, therefore, prophylaxis assumes unusual importance. The most important of all prophylactic measures is isolation of the sick. This isolation should be absolute, and should apply to the nurse as well as to the patient. The room in which isolation can be accomplished most perfectly should be selected as the sick-room. All hangings and unnecessary furniture should be removed. If possible, one person should be selected as an intermediary between the nurse and the family. The doctor's visits should not be unnecessarily prolonged. He should always have in readiness a gown of muslin or calico, fastened close at the neck and wrists, and long enough to completely cover his clothes. This he should wear during all visits to the sick-room. A stethoscope should be used in making physical examinations of the chest.

The period of isolation should not be less than forty days; it should be as much longer as the period of desquamation or purulent discharges may demand. Upon abandoning quarantine the child should be thoroughly bathed with soap and water, and then with a sublimate solution 1 to 5000. The nurse should take similar precautions. All the discharges of the patient should be disinfected with strong sublimate solutions.

The bedding, carpet, and clothing should be disinfected with boiling water or steam. The mattress should be destroyed. The room itself should be thoroughly washed—floor, ceiling, and walls—with a 1 to 1000 sublimate solution. It is a wise plan to furnish one room on the top floor of every house for a sick-room. The mouldings should be plain and the floor of hard wood; the walls and ceiling should be painted or covered with washable paper. The bedstead should be of enamelled iron.

Treatment.—There is no specific for scarlet fever. Many have been proposed, but have been tried and found wanting. Much may be done to avert complications and render them less serious when they occur, and many lives may be saved by judicious management. Mild cases require little or no medication. The child should be kept in bed for at least three weeks, and should receive a fluid diet for not less than two weeks, a soft diet being given thereafter. Milk is by all means the best diet for scarlet fever patients. It may be given peptonized or plain, as may be indicated by the case. It is usually best to dilute it with Vichy water or lime water. Later in the disease, broth, eggs, or meat jellies may be given. The stomach should never be overcrowded.

The initial vomiting usually requires no treatment, but the bowels should be acted upon mildly by small repeated doses of calomel. Later they should be kept acting, if possible, by means of enemata rather than by use of cathartic drugs. Stimulants are sometimes demanded in severe cases, but are rarely required in uncomplicated ones. In malignant cases they should be pushed to the point of tolerance. Strychnine is of great value in septic cases with prostration; it may often be combined to advantage with digitalis. Bathing of the surface with warm water, followed by thorough anointing with plain or carbolic vaseline or a five per cent. ichthyol ointment, should be begun as soon as the first signs of desquamation appear, and should be continued through the course of the disease. It gives comfort to the patient and aids materially in limiting infection.

Burning and soreness of the throat during the first few days may be mitigated by giving cool water or bits of ice. Later, in the simpler forms of pharyngitis, hot drinks may be given, or irrigation of the back of the throat with weak hot saline or boric acid solutions may be employed. Chlorate of potassium should be avoided. Its beneficial effects are doubtful, and its known irritating effect upon the kidneys contra-indicates its use. Nasal syringing should be avoided unless it is clearly indicated. Jackson, in a large experience as aurist of the Boston City Hospital, has seen less otitis when it has not been generally employed. It is indicated by a purulent nasal discharge or obstruction of the naso-pharynx. More harm than good may result from over-zealous attempts at local treatment of the throat and nose. Peroxide of hydrogen is, in my opinion, an unsafe remedy in such conditions. It is an irritant, even when rendered alkaline, and has the power to prolong indefinitely the presence of pseudo-membrane. The most successful treatment consists in the use, not of active and poisonous antiseptics, but of mild and cleansing washes freely and frequently applied.

It should not be forgotten that a high temperature is normal to scarlet fever. It may be allowed to run, therefore, without interference, to a somewhat higher point than in most other diseases. A temperature continuously above 104° F., however, demands treatment. It is best reduced by means of the cold bath, but this, for obvious reasons, is less practicable in private than in hospital practice. The cold pack or cold sponging is almost equally good, and is more available. An effective method of applying cold has been adopted at the Willard Parker Hospital, and is thus described by Northrup: "The tendency in all cooling processes is for the feet to become cold. To obviate this, the patient is placed upon blankets, but the legs, feet, arms, and hands are wrapped in warm, dry blankets, and hot bottles are enclosed in the wrappings. An ice-bag is placed to the head. The face and trunk are freely sponged in warm water and alcohol, evaporation being hastened by fanning, so long as it cools the patient, clears the cerebrum, gives force and rhythm to the heart, and leaves the patient to a quiet sleep. The warmth to the extremities seems to exert a favorable influence upon the heart's action and to favor a free superficial circulation."

Antipyretic drugs should be given with great caution. Quinine is the least objectionable, but is difficult to administer; it is frequently vomited, and sometimes causes unpleasant nervous symptoms. The coal-tar antipyretics are capable of doing great harm if injudiciously administered.

Anti-streptococcus serum has recently been proposed as rational treatment for scarlet fever, and some very favorable results have been reported from its use. At the present writing, however, very little is known of the treatment, and no expression of opinion is possible. As the more serious symptoms are due to streptococcus infection, the theory underlying this method of treatment is not irrational.

Emaciation and anæmia are very frequent results of scarlet fever. During convalescence, therefore, active tonic treatment should be instituted, the chief reliance being placed upon iron. The patient should be particularly protected from cold, for exposure not infrequently seems to precipitate nephritis long after its usual period of occurrence.

MEASLES.

Measles is an acute, infectious, contagious disease which presents, when it pursues a typical course, the following characteristics. After an incubation of twelve days there is a gradual invasion, marked by feverishness, coryza, suffusion of the eyes, and dry cough, followed on the fourth day by a maculo-papular eruption, which appears first on the sides of the face and neck and slowly spreads over the body. This eruption continues for about five days, when it fades away and is followed by a bran-like desquamation, which is usually completed within a week.

Etiology.—Analogy leads to the belief that measles is due to a specific micro-organism, but it has not yet been isolated. While it must be an extremely diffusible germ, its vitality is evidently small. It is at least a fact that measles is the most contagious of all the infectious diseases except small-pox. It is uncommon under six months, but above that age every child who has not already had it may be expected to contract it upon exposure. Adults are rather more susceptible to it than to the other infectious diseases. Measles is endemic in all large towns, but at intervals becomes epidemic, and usually spreads over a wide area before it expends itself.

Sources of Infection.—The measles poison is usually conveyed by direct contact, but the area of contagion is large. It may be conveyed by clothing or may be contracted by a susceptible person entering a room recently left by a measles patient. This, however, is far less common than with scarlet fever. Although intermediate contagion may occur, it is comparatively rare. The infectious power of the poison is quickly lost, so that sick-rooms soon become safe for occupancy. While it is possible that the contagium may be conveyed by the breath, it is certain that it resides in the sputa and the discharges from the nose and eyes. If it resides in the desquamation scales, it is far less potent than is the poison carried by the desquamation of scarlet fever.

Period of Incubation.—This may range from nine to twenty-one days.

Among one hundred and forty-four cases, Holt found it to be between eleven and fourteen days in sixty-six per cent. I have repeatedly seen the initial symptoms appear twelve days after exposure. From all the evidence available, it would seem that twelve days is the most common period of incubation.

Period of Infection.—Measles is most contagious at the height of the attack. It may be contagious from the first appearance of the catarrhal symptoms, cases being recorded in which the disease was transmitted four days before the eruption appeared. The contagiousness seems to diminish as the active symptoms subside, and is slight during the stage of desquamation. Except in complicated cases in which the catarrhal symptoms are prolonged, the period of infection is not over twenty-eight days.

Pathology.—The lesions of uncomplicated measles are confined to the skin and the mucous membranes of the conjunctivæ, nose, pharynx, larynx, and the larger bronchial tubes. The morbid changes of the skin are those of acute hyperæmia; on the mucous membranes they are those of acute catarrh. In complicated cases pseudo-membranous inflammation may occur.

The complications are due to mixed infection, the germ most commonly present being the staphylococcus. Streptococci are, however, frequently present, and, as a rule, cause more serious lesions than do staphylococci. The mucous membranes are rendered by measles very susceptible to the assaults of these germs. As they are invariably present in the wards of hospitals, the disease in such institutions is always a dreaded one, for it is prone to be complicated.

Symptoms.—Very mild cases of measles sometimes occur, but the disease not infrequently runs a severe course. In rare instances a malignant type is encountered.

Invasion.—A chill followed by high temperature may be the initial symptom, but, as a rule, the disease begins gradually with feverishness, sneezing, coryza, suffusion of the eyes, and photophobia. Within twenty-four hours a cough of peculiarly hard, dry character appears, and the attack presents all the symptoms of a catarrhal cold. The coryza, however, is usually more marked than that of an ordinary cold. The fever often falls somewhat after the first day, but the coryza and cough do not correspondingly diminish. The eruption is usually first seen on the afternoon of the fourth day, and is accompanied by increasing fever. It sometimes appears as early as the second day, particularly in young children, and is in rare instances delayed to the fifth or sixth day. Drowsiness is not uncommon during the stage of invasion, but there are no characteristic constitutional symptoms.

Koplik describes a symptom (*Archives of Pediatrics*, vol. xiii., No. 12) which he believes to be of the greatest value in making an early diagnosis of measles. He says that on the first day of the invasion an examination of the buccal mucous membrane in a good light will reveal a scattered eruption consisting of small, irregular spots of bright red color, in the centre of each of which is a minute bluish-white speck. This he regards as pathognomonic of measles.

Temperature.—The temperature does not usually range as high in measles as in scarlet fever. Occasionally it will be found at 103° F. or 104° F. on the first day; more commonly it is not above 102° F. It frequently falls somewhat below that point on the two following days, but increases as the eruption appears, and reaches its height on the second day of the eruption. It then gradually falls and becomes normal between the seventh and ninth days of the disease. Not infrequently there is a sudden fall about the sixth or seventh day, forming almost a crisis.

Eruption.—The eruption usually appears on the fourth day, but in some cases is seen on the third day and in others is delayed until the fifth. It is first seen on the temples and sides of the face, on the neck, or behind the ears. At its earliest appearance it commonly consists of small red spots having no strictly characteristic appearance. They rapidly increase in size and form small macules or very slightly elevated papules on a slightly reddened base with normal skin between. They are circular or crescentic in shape, and, being hyperæmic in nature, disappear on pressure. As the eruption develops it tends to become confluent in places, particularly on the face, where it assumes a blotched appearance. There is usually a certain amount of œdema, particularly about the cheeks and eyes, which further tends to change the appearance of the patient. The eruption usually reaches its height at its first site of appearance at the end of thirty-six hours; it remains stationary for about two days and then rapidly fades away. It extends over the body somewhat slowly, appearing on the trunk and limbs on the second day. The wrists and backs of the hands are commonly the points last involved.

On the first day the spots form simple macules, but later they become flat papules that can be readily felt by the finger. They are sometimes almost shotty to the touch. The rash presents its most typical appearance on the chest. Miliary vesicles are not uncommon, and in rare cases petechiæ appear. Occasionally the rash, instead of assuming the usual hyperæmic form, becomes distinctly hemorrhagic. This may occur in limited areas or may extend over the whole body. In the latter case it presents the type known as "black measles," a condition extremely rare in private practice. The spread of the eruption is sometimes very rapid, the whole body being covered in a few hours, but this is rare. In other rare instances the rash is so slight and of such short duration as to be almost overlooked. The constitutional symptoms in such cases are, as a rule, correspondingly mild. Occasionally, in malignant cases marked by sudden and severe initial symptoms, the rash scarcely makes its appearance or is greatly delayed.

Constitutional Symptoms.—These reach their height during the stage of eruption, being usually at a maximum on the sixth day of the disease. They remain stationary for about two days, when the fever abates and all the symptoms begin to subside. During the height of the disease the patient presents a very characteristic appearance. The face is covered by a patchy eruption, and is swollen and œdematous; the eyes are red and sensitive to the light, and are filled with a mucous or muco-purulent secretion; the nose is swollen and discharges a similar secretion; there is a dry, metallic, and very troublesome cough; the tongue is coated, the appetite is completely lost, and the bowels are frequently relaxed; the child lies in a heavy and stupid condition, but is restless and irritable when disturbed. The glands at the angle of the jaw are frequently enlarged, and sometimes also the post-cervical glands. As the fever subsides the cough rapidly changes its character, becoming looser and less irritating. It frequently disappears within a week, but sometimes the evidences of bronchitis continue and the cough long proves a troublesome symptom. The photophobia in most cases subsides rapidly, but the eyes are prone to remain weak and watery. The child becomes brighter and less irritable, the appetite returns, and the evidences of illness rapidly disappear.

Desquamation.—Desquamation begins as soon as the eruption has faded. It rarely continues longer than ten days in any given area, and may be of much shorter duration. It is most marked where the eruption has been the most intense. It occurs in fine branny scales, quite unlike the lamellar desquamation of scarlet fever. It is often so slight as to be completely overlooked, particularly where inunctions of the skin have been practised. Desquamation is usually completed by the twentieth day from the onset of the fever.

Irregular Forms.—Although a much larger proportion of cases of measles will run a typical or regular course than will a similar number of cases of scarlet fever, the disease is quite capable of assuming very irregular and atypical forms. Such irregular types are most common in children under three years.

The disease may be extremely mild, the eruption being faint and the fever slight. Such cases present no variations from the usual type, except that of mildness in degree. Although the catarrhal symptoms may be slight, the diagnosis of *morbilli sine catarrho* should be made with extreme hesitation.

A severe form is sometimes seen, marked by high temperature, intense eruption, and severity of all the symptoms. Except in young children, the uncomplicated disease, even when thus severe, is rarely fatal. But it should not be forgotten that a temperature that reaches an unusually high point, or continues unabated as the eruption fades, is usually due to some complication, commonly pulmonary. Any marked variation from the usual type demands particular attention, for it probably indicates a complication.

Malignant measles, marked by intense and overwhelming symptoms from the outset, is fortunately rare outside of institutions. The same is true of hemorrhagic or "black measles." Relapses in measles are extremely rare, and are of doubtful occurrence. A secondary rise of temperature after a normal fall indicates a complication, usually otitis, pneumonia, or gastro-enteritis.

Complications and Sequelæ.—The most common and serious complications of measles are broncho-pneumonia, membranous laryngitis, and otitis; the most common sequelæ are tuberculosis and conjunctivitis.

Pneumonia.—Bronchitis is an essential part of measles, but inflammation easily extends from the smaller bronchi to the alveoli, thus transforming a normal condition into a serious complication. The younger the child the greater is this danger. It occurs chiefly in children under three years, and is comparatively rare over four years. It is very common in institutions, and renders measles the most dreaded of all the contagious diseases in infant hospitals, diphtheria being no exception to the rule. In a recent epidemic of measles in the Infants' Hospital of New York every case in children under eighteen months was complicated by broncho-pneumonia or croup, and eighty per cent. died. The pneumonia usually made its appearance soon after the eruption reached its height, but developed in a few cases during the stage of invasion. According to Holt, ten per cent. of all cases are complicated by broncho-pneumonia. He agrees with Henoch that a certain amount of pneumonia is found at autopsy in almost every fatal case. Lobar pneumonia is an occasional complication of measles in children over four years. Empyema is sometimes a sequel of such complicating lobar pneumonia.

Croup.—Catarrhal pharyngitis is an essential part of measles; pseudo-membranous pharyngitis sometimes occurs as a complication. Instead of invading the nose and ears, as in scarlet fever, it shows a strong tendency to invade the larynx, but croup frequently develops without the appearance of membrane in the pharynx. As in scarlet fever, the pseudo-membranes which develop during the height of the attack are usually due to streptococci, and are, therefore, not true diphtheria. Those which develop later are usually due to Klebs-Loeffler bacilli, and are true diphtheria. As in scarlet fever, the streptococcus disease is quite as fatal as the bacillus disease. Not only is the child in imminent danger from laryngeal complications, but is in serious danger also of broncho-pneumonia, which occurs as the direct result of streptococcus infection. Fortunately, in private practice both complications are rare in children over four years.

Otitis occurs somewhat rarely. Both ears are usually involved, but the disease presents in its symptoms and course nothing unusual. Diarrhœa is of frequent occurrence, and may be so severe as to prove a serious complication. It may be due to simple intestinal indigestion, or it may be the evidence of enterocolitis. Febrile albuminuria is not infrequent in cases with high temperature, but nephritis is very rare. Nervous symptoms, excepting the occasional occurrence of convulsions at the outset, are rare. Meningitis may follow otitis. Cellulitis and suppurative adenitis are very rare, but moderate enlargement of the cervical glands is common and sometimes persists for months.

The occurrence of measles simultaneously with other infectious diseases is not very infrequent. There seems to be a particular tendency to the simultaneous occurrence of measles and pertussis.

Tuberculosis is the most serious sequel of measles. It commonly occurs as a tubercular broncho-pneumonia, general miliary tuberculosis, tubercular adenitis, or tubercular joint-disease. These conditions may result from primary infection or from the lighting up of some old tubercular process. Measles unquestionably puts the tissues into a condition very susceptible to the development of tubercle bacilli, so that infection may result from slight exposure. Acute miliary tuberculosis may follow measles at once, the temperature range being continuous from the onset of the primary disease to death from the complication. General tuberculosis with grave pulmonary involvement may follow so close upon measles as to leave no appreciable interval between. It is sometimes the cause of a secondary fever which develops soon after the subsidence of the primary fever. Tubercular disease of the bones and joints subsequent to measles is usually of late occurrence.

Prognosis.—Death from measles in private practice is rare in children over four years of age. The mortality of the disease is probably from four to six per cent., but under two years it is often twenty-five per cent. or more. It is highest between one and two years; but even at this age uncomplicated measles is not a highly fatal disease. Pneumonia is the cause of death in almost ninety per cent. of fatal cases. A violent onset of high temperature warrants a guarded prognosis. A rising temperature with a fading eruption warrants an unfavorable prognosis. The same is true when the eruption is excessive in amount and confluent over wide areas. Grave constitutional symptoms with faint eruption is a serious condition. The same is true of a hemorrhagic or black eruption, but it is not so necessarily fatal as is commonly supposed.

Measles has a marked tendency to leave serious conditions behind. Treatment should not be directed solely to saving the life of the child, nor should the prognosis be given solely with reference to that end. The strong tendency to tubercular invasion should not be forgotten; and when the fever persists after the tenth day, even if it is not high, the prognosis should be guarded. The list of chronic affections left in the wake of measles is a long one,—bronchitis, pharyngitis, rhinitis, adenoid growths, and enlarged tonsils and mesenteric glands are among the number.

Prophylaxis.—The high mortality of measles before three years suggests the advisability of taking particular precautions against the exposure of infants. Delicate children of the so-called scrofulous type and those with hereditary tendency to tuberculosis should be especially guarded against exposure. Early and absolute isolation of the sick is imperative. Quarantine of the patient should not be less than twenty-eight days, and as much longer as purulent discharges may continue. The period of quarantine after exposure should not be less than fifteen days, and twenty days would be preferable. Children who have been exposed should be isolated from other children for that period. The sick-room is less liable to prove dangerous than is the scarlet fever sick-room. Thorough cleansing and ventilation for two weeks after the patient has left it is sufficient to insure safety. The infection of measles is not persistent, nor is intermediate infection common, so that prolonged precautions are not necessary, but during the height of the disease the same measures should be taken as in scarlet fever.

Treatment.—The measles patient should be placed in as large and well ventilated a room as is available. The temperature should not be kept at too high a point, nor should the child be forced to swelter under too heavy coverings. It accomplishes no good and renders the child restless and irritable. The room should be kept very dark, and no direct light should be permitted to fall on the eyes. As inflammation of the eyes subsides the light should be gradually admitted, but full light should not be permitted until the conjunctiva has become normal in appearance. Itching of the lids may be relieved by cold cloths or by the application of cold cream or some bland oil. If a purulent discharge appears the eyes should be kept clean by frequent applications of a warm solution of boric acid.

Even in the mildest cases the child should be put to bed and kept there until desquamation is practically completed. The diet should consist of milk and broth during the febrile stage; during the height of the disease the child should not be over-urged to eat. Applications of plain or carbolized vaseline will do much to reduce the irritability of the skin. As soon as the eruption begins to subside, inunctions of plain or carbolized vaseline or ichthyol ointment should be practised daily. A daily warm bath does much to hasten desquamation. The hard, metallic cough is one of the most troublesome symptoms of the disease. Very little relief, however, can be afforded by treatment before the fever begins to subside. Nauseating expectorants tend to render the child more irritable and to increase the anorexia, and have but slight effect on the cough. Small doses of opium aid in allaying the cough and are quite permissible. Brown mixture in the form of tablet triturates is as effective as any treatment, and is easy of administration.

Though hyperpyrexia is uncommon in measles, the fever sometimes requires attention. The effect of the fever upon the patient is a better guide for treatment than is the thermometer. If the child becomes comatose or restless and delirious, small doses of phenacetine are admissible. Only enough should be given to reduce the temperature moderately and to allay the restlessness. Cold sponging is the best treatment for high temperature, and far preferable to the administration of large doses of antipyretics. Uncomplicated cases do not require stimulants. Broncho-pneumonia requires the same treatment that it would receive under other conditions. Other complications must be treated as they arise.

RUBELLA.[1]

By WILLIAM A. EDWARDS, M.D.

THE statement made several years ago that rubella is a distinct disease is here reaffirmed; indeed, it is now almost universally recognized as such, although even at this late day a writer occasionally questions its specific nature. Donald Hood, before the Royal Medical and Chirurgical Society,[2] in affirming the identity of this disease with measles, endeavored to offer a scientific explanation of his statement of the identity of rubella and rubeola. His assumption is that rubella is nothing but a modified form of rubeola, in which the contagium has undergone an evolutionary modification in consequence of profound alteration in the soil and the conditions of its environment.

That rubella does not protect from rubeola is explained by saying that rubella, being a milder form of the disease, does not confer the protection afforded by a previous attack of measles of the usual severity,—an argument the fallacy of which is shown at once by mentioning how at variance it is with all our accepted knowledge on the conference of immunity by attenuated virus. Putnam has also recently asked the question, Is German measles (rötheln or rubella) an independent disease?[3]

The consensus of opinion now is that in rubella we have a disease occurring almost universally in epidemics and due to an unknown but specific contagion. The existence of the disease is doubted only by those who have never seen it. Its propagation is similar to that of all exanthematous affections, but it is peculiar in the rapidity with which it spreads and in the extensive nature of the epidemics. Of recent years I have observed that it attacks adults very frequently, and Ashby thinks that in some epidemics adults suffer more in proportion to their numbers. This writer, in company with Dr. Hutton, observed an epidemic in the Children's Hospital, Manchester. Out of twenty-seven cases, eight were those of lady probationers and nineteen were of children, so that the adults suffered far more in proportion to their numbers.

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[1] The present communication is an addendum to my article (volume i. p. 684) of this Cyclopædia. My contributions to the study of rubella were among the earlier American papers of the last decade. The ground then taken that rubella is a distinct entity is now generally accepted, although the argument at times has become warm and pointed. It has been thought best to adopt the term "rubella" instead of "rötheln." The former is more generally used in America, although the latter is still the reference-word in several standard books and in some of the catalogues, notably the Index Catalogue of the Surgeon-General's Office.

[2] Medical Press and Circular, London, March 22, 1893.

[3] Boston Medical and Surgical Journal, 1893, cxxix. 30–33, Discussion 37–40.

The statement that a previous attack of either measles or scarlatina does not protect from the contagion of rubella is verified by more extended experience. Observers continue to report epidemics of rubella followed by measles as a sequela. A recent communication is that of Kramsztyk,[1] and Fiessinger[2] has seen rubella preceding measles. Stone and Davis[3] report an extremely interesting condition in an orphan asylum. Rubella appeared first and spread through the institution, but was hardly well under way before genuine measles was introduced, an opportunity being thus afforded of seeing the two diseases running side by side. A dozen of the children had first one disease and then the other, not in a way that could be accounted for by relapses, but often with a clear interval of a fortnight of sound health. Some had measles first, and then rubella; others rubella first, and then measles. Theodore[4] records a case in which measles followed rubella on the fifth day. The cervical glands, enlarged during the course of rubella, resumed their normal size while the measles was at its height. Leflaire (Sajous's Annual) reports four children who had measles and six weeks later were down with rubella, and Powell[5] an attack of measles very rapidly following one of rubella.

We may now state that the following facts are in accord with almost all the writers:

1. Rubella is a distinct disease, although sometimes bearing a striking resemblance to measles or scarlet fever. I must confess that in a very large series of cases the resemblance to scarlet fever was never to me personally very well marked, but that its clinical aspects were very often, indeed, so similar to measles that it was with difficulty differentiated. I am rather inclined to think that the statement that rubella resembles scarlatina has to some extent been copied from writer to writer and is not always an independent opinion. For many years this resemblance has never been very apparent to me, and I have never encountered any difficulty in deciding between the existence of rubella and scarlatina, but many times have I encountered extreme difficulty in the differential diagnosis between rubella and measles. Just here we must consider whether or not there really exist two forms of rubella,—one that mimics measles, and the other the scarlet fever mimic. Personally, as I have already stated, I have had little

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[1] Przegl. lek., Krakow, 1891, xxx. 625-637, Index Medicus; also Archiv für Kinderheilkunde, Stuttgart, 1894, B. xvi., H. i., 2.

[2] Méd. Mod., Paris, 1896, vii. 721.

[3] Northwestern Lancet, May 15, 1890; Sajous's Annual, 1891.

[4] Archiv für Kinderheilkunde, Stuttgart, 1894, B. xvi., H. i., 2.

[5] American Journal of Obstetrics, New York, 1896, xxxiii. 671.

experience in that form which is said to resemble scarlatina, and, if it exists, it is certainly rare. It is but fair to state, however, that writers have recently recorded their experience with this form of rubella. Holootschiner[1] writes on the question of rubeola scarlatinosa, and Squire[2] gives some clinical observations on the diagnosis of scarlatina from rubeola and rubella, while Sayer[3] notes a clinical resemblance to scarlatina.

2. Rubella does not protect against measles or scarlatina.

3. Neither measles nor scarlatina confers protection against an invasion of rubella.

4. Epidemics of rubella prevail without any regard to the existence of cases or epidemics of either measles or scarlatina, although the epidemics may run side by side, as in the St. Paul cases of Stone and Davis.[4] There is little doubt that occasionally sporadic cases of rubella are mistaken for rubeola; indeed, it is often a most difficult matter to make this differential diagnosis of a single case of rubella, as epidemicity is one of its most marked characteristics and diagnostic features. West aptly expresses the resemblance of rubella to measles when he says "they resemble each other somewhat as varicella and variola—alike but not the same,—not twin-sisters, indeed, but half-sisters, at any rate." I agree with Holt that it is never entirely safe to make the diagnosis of rubella unless the disease is prevailing epidemically. He further says that when the diagnosis is made in a sporadic case it is usually erroneous, and the disease is, in truth, mild measles or scarlatina. First cases in an epidemic are difficult of recognition and are often overlooked.

It has been observed that the features of an epidemic of rubella will be modified if either measles or scarlatina is prevailing at the same time. This modification will assume the characters of one or other of the modifying diseases in accordance with their prevalence and proximity. I have several times observed this curious phenomenon.

While I have always contended for the distinct nature of rubella, and insisted that it was an entity just as defined as the other exanthemata, still it is not improbable that the striking resemblance to measles is more than a coincidence. With our increased knowledge of biology and bacteriology, it seems at least probable that the germinal cause of measles and rubella may in times gone by have been derived from the same parent stock, and that the diseases as we find them to-day are the result of development and cultivation under different conditions.

The period of incubation is still somewhat indefinite, but it is certainly within from seven to fourteen days. No new evidence since my last publication has been accumulated to place it more definitely. Prodromal symptoms are rarely observed, but adults may complain of backache and headache for a few hours or even a day before the rash appears. The skin of the face sometimes tingles. The rash varies considerably; the color is at first usually rose-red, certainly much lighter than measles; the confluence is variable, and the crescentic arrangement not well marked; indeed, it is often entirely wanting. It reaches its maximum in forty-eight hours, and in my experience is almost always followed by more or less branny desquamation. Sometimes this is very slight, and may easily escape notice. The skin often remains in a more or less stained condition, which may last several days or a week.

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[1] Meditsina, St. Petersburg, 1893, v. 430, Index Medicus.

[2] Clinical Journal, London, 1892–93, i. 145–148.

[3] Columbus Medical Journal, 1897, xviii. 162–166.

[4] Ibid.

Dukes[1] notes three eruptions which bear a close resemblance to rubella: the roseola simplex which arises in hot weather or follows chill or indigestion; another, that occasioned by handling caterpillars; and the last, a medicinal eruption caused by copaiba, which Hutchinson has called the morbilli sine catarrh.

There is great variety in the severity of the attacks and in the clinical manifestations of the disease, some cases being most atypical. The rash may be so ill defined as to render a positive diagnosis impossible. I have described cases of undoubted rubella in which the course of the disease was that of a serious ailment, high temperature, albuminuria, pneumonia, earache, and other complications and sequelæ arising during the course of the epidemic.

The milder cases may be so mild as to be overlooked until the occurrence of other cases calls our attention to the existence of rubella. The later cases are apt to become more typical as the epidemic advances. As stated in my earlier papers, a characteristic symptom is enlargement of the posterior cervical glands,—that is, those along the posterior edge of the sterno-cleido-mastoid muscle. I have also observed enlargement of the submaxillary and occipital glands. Osborn has noted enlargement of the glands at the edge of the hair on the lateral aspects of the neck. Da Costa has found swelling and even suppuration of the cervical glands to be not uncommon sequelæ. This has not been my experience. I have never seen the glands suppurate.

Holt is about the only recent writer who does not consider swelling of the post-cervical glands an important symptom for the differential diagnosis. He remarks that it is not uncommon in measles. In my experience, however, it is a most valuable aid, because it often comes very early, and it usually enlarges the glands in such a manner as to allow of their separate and distinct recognition. It is rarely the agglutinated tumefaction seen in measles. Furthermore, the enlargement seems to be to a certain extent independent of the throat involvement, and does not bear the same relation to the intense catarrhal engorgement of the throat and fauces as it does in measles.

Other writers, as Townsend, Swift, and Griffith, have noted the frequency of enlargement of the lymphatic glands of the neck in measles, and sometimes the absence of enlargement in this situation in rubella. The treatment, which is very simple, is fully considered in the original article. Usually none whatever is required for the disease itself, complications sometimes demanding active therapeutic measures.

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[1] Lancet, London, March 31, 1894.

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PERTUSSIS.

By WILLIAM FITCH CHENEY, M.D.

The etiology of this disease long remained an unsolved problem and a source of controversy. Even to-day the solution is not wholly clear, but the only explanation of the cause that will fit all the known facts is the existence of a specific germ, that finds entrance into the body of the child, lodges and grows there, and by its development sets up the series of characteristic phenomena. Convinced of this by deductive reasoning, scientists set to work to furnish positive proof by finding the specific germ.

Bacteriology.—Especially connected with the last decade's researches in the bacteriology of pertussis are the names of Afanassiew, Cohn and Neumann, Kurloff, Koplik, and most recently Czaplewski and Hensel.

Afanassiew announced in 1887 that he had found in the sputum of children with pertussis a characteristic bacillus. This bacillus was subsequently found again by Szemetzchenko, by Genser, and by Wendt, and it became for a time pretty generally accepted as the genuine pertussis germ. But in 1893 Cohn and Neumann,[1] after exhaustive examination of a series of cases, announced that they could not find Afanassiew's bacillus, and expressed the opinion that this bacillus, even if constantly present, was only one of a number of accidental saprophytic forms, occurring even in normal sputum.

In place of the deposed bacillus, Cohn and Neumann put forward the claims to supremacy of a diplococcus, which they found more constantly present than any other microbe in the sputum of pertussis. This diplococcus was very small, and while in some cases it was abundant, in others it was rather scarce. Ritter, just about the same time as Cohn and Neumann, also announced that a diplococcus was much the most common form of germ found in the sputum of pertussis; but his was a larger diplococcus than that described by Cohn and Neumann, and the two could not be reconciled with each other.

In 1896 Kurloff[2] described a ciliated body resembling the protozoa, which he had found in the sputum of children with whooping-cough. The size of this parasite varied greatly. Some were very small, only half as large as the red blood-corpuscles, while others were of a circumference so great that they exceeded in size the largest forms of white blood-corpuscles.

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[1] Archiv für Kinderheilkunde, Band xvii., 1893.

[2] Centralblatt für Bacteriologie, Band xix., Nos. 14, 15.

They were covered usually over one-half, but sometimes entirely, with long cilia, by means of which they were able to move about at will. As the parasites increased in size they showed within their structure glittering spores, apparently arranged concentrically, that were finally set free by the bursting of the larger forms. These minute spores gradually increased in size, themselves developed spores, and finally burst. Kurloff claims that the parasite he describes was previously seen and described in 1886 and again in 1889 by Deichler.[1]

In September, 1897, Dr. Henry Koplik, of New York, published in the *Centralblatt für Bacteriologie* a report of sixteen cases of pertussis in which he had made bacteriological examination of the sputum, with the discovery in thirteen of a bacillus which seemed to correspond to the bacillus of Afanassiew. As described by Koplik it is a slender, minute bacillus, more delicate than the diphtheria bacillus and not more than one-third or one-half the latter's length. When stained it has a finely dotted appearance, and in this resembles the stained diphtheria bacillus; but here the resemblance ceases, for it is much thinner and shorter. When cultures of this germ were injected into animals (white mice, guinea-pigs, and dogs), none showed lesions of the lungs or characteristic convulsive symptoms; but only a few experiments with animals were made.

The most recent contribution to the bacteriology of pertussis is that made by Czaplewski and Hensel, in the *Centralblatt für Bacteriologie* for December, 1897. In forty-four cases examined they invariably found in the sputum a small, short bacterium, with oval, rounded ends. With moderate stains this bacterium takes on a distinctly darker coloring at the poles, while the middle connecting portion remains almost colorless, thus making the germ resemble a diplococcus. The bacteria are only two or three times as long as they are broad, and are very small. They are found most often free, rarely intra-cellular. Inoculations of animals with this germ did not produce processes analogous to those of pertussis; but Czaplewski was seized with a violent coryza during his work, and in his nasal secretions were found almost pure cultures of this bacterium. His attack, however, lasted only a week, and was not accompanied by the typical cough of pertussis. Czaplewski and Hensel believe that the bacterium isolated by them is the same as the bacillus described by Koplik, but they are not certain as to its identity with that of Afanassiew.

In the *Johns Hopkins Bulletin* for April, 1898, Koplik in a later article accepts the identity of the bacillus isolated and described by him with that isolated by Czaplewski and Hensel, but is inclined to leave its identity with that of Afanassiew an open question.

Thus it will be seen that several different germs have been found in the sputum of pertussis with sufficient regularity to be looked upon as the exciting cause of the disease, but that no one germ has yet been found which is generally recognized as the true one. We are probably nearer the truth than we were ten years ago, but we have not yet reached the solution of the problem.

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[1] Zeitschrift für wissenschaftliche Zoologie, Band xlviii., 1889.

Pathology.—What tissues are affected and what changes are produced in them by pertussis are by this time fairly well known. The changes consist in a catarrhal inflammation of the mucous membrane of the larynx, trachea, and bronchi, usually also of the nose and naso-pharynx, with the formation, as a product of this inflammation, of a tough, adhesive mucus. So far the morbid alterations produced by pertussis do not differ especially from those produced by other inflammations of the upper respiratory tract; but in pertussis we have besides these the characteristic spasmodic cough, indicative of a nervous excitability that distinguishes the disease from other respiratory catarrhs. The explanation of this peculiar feature is suggested by Drs. Wells and Carré[1] to be the fabrication by the pertussis germ of a virus which by its absorption acts as an irritant poison to the nervous system, especially to the respiratory and vagal centres, making them abnormally excitable. From what is known of the action of other germs in producing a local lesion and a general intoxication, the following is probably the essential pathology of this disease: a catarrh of the upper respiratory tract due to some microbe that makes its habitat there, and a hypersensitiveness of the terminal nerve-filaments in respiratory and gastric mucous membranes, due to some poison generated by the microbe and absorbed into the blood.

Clinical History.—The symptoms and signs of pertussis, its typical course, and its possible complications have long since been carefully observed and accurately described. There is nothing new to be said in this connection, except to explain how the modern theories regarding the etiology and pathology accord with the facts in the clinical history as for years past they have been presented. (1) The first or catarrhal stage of the disease means the presence and growth of the specific germ upon the laryngeal and tracheal mucous membrane; the second or paroxysmal stage means that the germ has flourished long enough and has developed virus sufficient in amount to affect the nervous system in the way peculiar to itself; and the third or declining stage means the overpowering of the invading germs and the gradual elimination from the system of the toxic products of their activity. (2) The primary object of the cough, which is the characteristic feature of the disease, is the removal of the tough, viscid plug of mucus that accumulates from time to time in the larynx or trachea, in consequence of the catarrhal inflammation that the specific germ has set up; the frequency and violence of this cough are due partly to the sticky nature of the secretion, but mainly to the hypersensitive condition of the mucous membrane on which this secretion is formed; and the vomiting that so often ends the paroxysm is likewise due to hyperæsthesia, this time manifested in another branch of the pneumogastric nerve. (3) The paroxysms of coughing are likely to be more frequent and more violent at night, because the patient is then shut up in a room where oxygen is not so plentiful and the microbe finds conditions more favorable to its growth; or, secondly, because at night, on account of the patient's position during sleep, the secretion more readily accumulates and sets up irritation. (4) The complications that sometimes occur with pertussis are simply accidents in its course: the hemorrhages (from nose, mouth, conjunctiva, ear, or meninges) being mechanical and dependent upon the general venous congestion that accompanies the violent paroxysms of coughing; the pulmonary inflammations (broncho-pneumonia or lobar pneumonia) arising from secondary invasion by staphylococci, streptococci, or pneumococci upon a soil made ready for them by the pertussis germ; and the disturbances of the nervous system (convulsions, coma, paralysis, aphasia, loss of sight or hearing) occurring because of the irritable condition of all the nerve-tissues, or because of the accumulation of carbonic acid gas in the blood during a paroxysm, or because of an intra-cranial hemorrhage.

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[1] London Lancet, June 8, 1895.

Diagnosis.—The position of the profession as regards the diagnosis of pertussis is still as humiliating as it has always been. During the first stage, before the characteristic whoop appears, we cannot say positively whether the disease is whooping-cough or simple bronchial catarrh; while after the whoop appears any mother can make the diagnosis without a physician to aid her. There seems no prospect for release from this embarrassing situation until the special germ that causes pertussis has at last been definitely determined; then by examination of the sputum we may be able to decide before the whoop occurs. Czaplewski and Hensel, in fact, succeeded in finding in five cases of bronchitis the bacterium above described by them before any clinical diagnosis of whooping-cough had yet been made, and so were able to predict the typical paroxysms which sooner or later in every case appeared.

Prognosis.—The mortality of pertussis has heretofore been altogether too high, because of the popular tendency to neglect the disease and to consider it too trivial to require medical attention. It is well to remember what the statistics show, as Dr. W. W. Johnston[1] has pointed out,—viz., that in the United States whooping-cough holds its own with scarlet fever and measles in the number of lives it destroys each year. The prognosis is good with proper care and attention; but so common is it for the physician never to be summoned until some alarming complication has arisen, that many children die from pertussis whose lives might have been saved.

Treatment.—Out of the mass of suggestions that have been made and continue to be made constantly concerning the management of whooping-cough, it will suffice to select a few of the therapeutic agents that at the present time are generally accepted as useful. These can best be classified under the heads of (1) Prophylaxis, (2) Hygiene, and (3) Drugs.

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[1] Archives of Pediatrics, April, 1895.

(1) *Prophylaxis.*—Pertussis is contagious, and one child gets the disease from another. It is rational, therefore, to keep cases of whooping-cough isolated; they must not be sent to school nor taken into public halls or conveyances. The people are fairly well educated by this time about other contagious diseases, but about whooping-cough they still seem ignorant or careless.

(2) *Hygiene.*—Where drugs accomplish so little, resort must necessarily be had to every hygienic aid. First in importance under the head of hygiene comes *fresh air*. Much dispute has taken place upon this point in years past, but to-day the best authorities agree that as the disease is due to a germ, and oxygen is one of the best germicides, the patient should be allowed as much oxygen as possible. In pleasant weather the child should live almost continuously out of doors, and even in cloudy or windy weather it is generally better to take the child out, well wrapped and protected from cold. Some judgment must naturally be exercised, but the rule should be "all the fresh air possible." A second requisite is *a well-ventilated sleeping apartment.* The lesson taught by increased cough at night is commonly overlooked; it means, as a rule, foul air in the child's bedroom. Oxygen is as good a germicide by night as by day, and should be furnished as abundantly as the arrangements for ventilation will permit. A third important point is the *nutrition* of the patient, which must be carefully kept up. If paroxysms are accompanied by vomiting, the child must be fed soon afterwards, without waiting for regular meal-hours; and if vomiting is so frequent that no food given by the stomach has time to digest, then resort must be had to rectal feeding.

(3) *Drugs.*—These may be best considered under two heads,—(*a*) antiseptics, and (*b*) antispasmodics. (*a*) *Antiseptics.*—The rational medication of a disease produced by germs is the use of remedies that will destroy germs, and these remedies are most efficacious when applied directly to the site at which the germs have colonized. Within recent years efforts have been made by many experimenters to reach the germ of pertussis in the upper respiratory tract by means of swabbing the parts within reach, by insufflations, by sprays, or by vapors. As for *swabbing*, Raubitschek[1] tried the use of a solution of corrosive sublimate, 1 to 1000, applied thoroughly every day with a brush or absorbent cotton to the pharynx and epiglottis; he claims that this treatment never failed to arrest the disease after a few applications. Moncorvo[2] recommends the similar use of a one per cent. solution of resorcin; and by other observers good results have been claimed for a one per cent. solution of carbolic acid, or a saturated solution of boracic acid applied as described for the sublimate solution. As regards *insufflation*, the powders recommended most highly have been quinine, resorcin, and boracic acid; these are to be blown into the nose or into the larynx with a powder-insufflator once or twice a day. For *sprays* the

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[1] La Semaine Médicale, April 26, 1894.

[2] La Médecine Infantile, November 1, 1895.

[illegible] used as for swabbing. While all of the preceding [illegible] of great value, they are practically of little use, because [illegible] by small children to their employment. *Vapors* are [illegible] brought into contact with the respiratory mucous membrane [illegible], because the air of the room can be impregnated with [illegible] takes them in unconsciously; but at the same time the anti[illegible] because of its greater dilution. Of all the substances [illegible] inhalation by vaporization, most observers agree that a [illegible] of carbolic acid gives the best results; but the vapors [illegible], from creolin, and from thymol are all exceedingly useful.

Under the head of antiseptics should be mentioned also the internal administration of quinine, for its germicidal action is the one especially [illegible] for it. The dosage recommended is one centigramme (three-twentieths of one grain) for every month, and one decigramme (one and one-half grains) for every year of the child's age, given three times a day [illegible] syrup of licorice. There is no doubt of its value, if the child can be [illegible] to take it and to retain it; but practically it is of limited usefulness, because of its bitter taste and its tendency to upset the stomach.

(*b*) *Antispasmodics.*—If we cannot kill the germ, we can still try to overcome the effects of its development in the body; and first among the drugs that will do this comes bromoform, one of the most recent and one of the most efficient of the remedies for pertussis. It should never be prescribed in solution, but always alone; and it is best administered by dropping it on a little sugar in a spoon, beginning with a dose of one drop every four hours, and increasing the dose by one drop each day until some effect is produced. The number of the paroxysms and their severity are certainly diminished by bromoform.

Second in usefulness among antispasmodic drugs comes antipyrin, either alone or combined with bromide of sodium. Dr. Charles G. Kerley,[1] during an epidemic of pertussis in the New York Infant Asylum, divided his cases into groups of twenty and treated each group in a different way. He obtained the best results from a mixture of antipyrin and sodium bromide. The dosage he recommends is: for an infant aged eight months, gr. ss of antipyrin with gr. ii of sodium bromide; aged fifteen months, gr. i of antipyrin with gr. iiss of sodium bromide; aged from two and a half years to four years, gr. ii of antipyrin with gr. iii of sodium bromide. In each case the dose to be repeated every two hours.

Finally, the only one of the old remedies that stands the test of time and is still considered valuable as an antipasmodic is belladonna, in the form either of the fluid extract or of a solution of atropine. Whatever preparation is used, the dose should be small at first, and gradually increased until the physiological effects of the drug are produced,—viz., dilatation of the pupils, dryness of the throat, and flushing of the cheeks.

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[1] New York Polyclinic, 1896, vol. viii., No. 2.

VACCINATION.

By R. G. FREEMAN, M.D.

DEFINITION.

VACCINATION is the conference of artificial immunity by the inoculation of the micro-organism (as yet unknown) of small-pox, modified by its passage through a relatively insusceptible animal.

HISTORY.

The literature of vaccination during the past eight years, since the original volumes of the Cyclopædia were published, furnishes further evidence of the efficiency of vaccination as a protection against variola, and the safety of the procedure if carefully carried out with proper virus. The complications following vaccination at rare intervals can in all probability be almost entirely eliminated by such improved methods of procuring and preserving virus as will eliminate pyogenic bacteria, and by protection of the site of vaccination from infection.

Singularly enough, one of the most imposing works on vaccination, published recently by a man of considerable scientific attainment, is intended to throw doubt on the efficiency of vaccination and discredit on the practice. The weakness of the position taken by Crookshank[1] in this work is evident from the fact that it has apparently had no influence on enlightened public opinion.

That the mortality from small-pox has been reduced to relative insignificance by the practice of vaccination is evident when one compares the mortality from that disease before and after the introduction of vaccination.

Almost all statistics concerning vaccination emphasize the following facts:

(1) The mortality of the vaccinated is markedly less than that of the unvaccinated.

(2) The mortality among the vaccinated having good scars is less than that among those having poor scars.

(3) The mortality among those having several vaccination scars is less than that among those having only one vaccination scar.

(4) The mortality from small-pox in any particular locality is, for the

most part, inversely proportional to the stringency and effective execution of the vaccination laws.

1. A marked diminution in the mortality of the vaccinated as compared with the unvaccinated is evident in practically all reports, the difference being more marked in some cases than in others.

Blyth[2] concludes from a study of statistics that the mortality of all ages in a vaccinated community will be 5 per cent., while in an unvaccinated community it will be 49 per cent. In ninety-four thousand cases of small-pox tabulated by Abbott[3] the average mortality among the vaccinated was 30 per cent., and among the unvaccinated 5.35 per cent. The mortality from small-pox among the vaccinated depends on the efficiency of the virus, the technical conduct of the operation, and the time interval between vaccination and exposure; that is to say, it depends on the degree of immunity which one possesses when exposed to small-pox, this diminishing with the lapse of time. Whether this gradually diminishing immunity is dependent upon some substance contained in the body, or, as is more likely, upon an acquired functional capacity of the body-cells, or upon some factor not yet definitely conceived, it is perhaps yet too early wisely to decide.

2. It seems probable that this immunity lasts a longer time in those persons in whom vaccination is followed by a typical scar than in those in whom the scar is less characteristic. Welsh[1] has compiled statistics of three thousand two hundred and seventy-nine post-vaccinal cases of small-pox based on the appearance of the scar, without reference to the period that has elapsed since vaccination, as follows:

	CASES.	DEATHS.	PERCENTAGE OF DEATHS.
Vaccinated in infancy,—good scars	1474	124	8.41
Vaccinated in infancy,—fair scars	701	101	14.4
Vaccinated in infancy,—poor scars	1104	296	26.81
	3279	521	15.88

Welsh thus finds in his cases that the mortality of those having good vaccination scars is only a little more than one-half what it is in those with fair scars, and less than a third what it is in those with poor scars. Approximately the same result is obtained from a study of Marson's[5] statistics, those having good scars giving a mortality of 2.52 per cent., while those with poor scars gave a mortality of 8.82 per cent.

3. Revaccination undoubtedly adds materially to the protection from small-pox, since the protection afforded by vaccination only lasts for a certain limited period.

Marson finds in a study of fifteen thousand cases of small-pox seen by
at the mortality in the vaccinated was much less than in those who
iously suffered from small-pox, and that the greater the number

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writer is indebted to Dr. W. G. Thompson, editor of the American System of
eets of Dr. Welsh's article.

of vaccination scars a small-pox patient had the better were his chances of recovery.

	PER CENT.
Thus, in the unvaccinated the mortality was	33.
In those *said to be* vaccinated, *but with no scar*	23.57
In those who had previously had small-pox	19.
In the vaccinated having one scar	6.80
In the vaccinated having two scars	4.70
In the vaccinated having three scars	1.95
In the vaccinated having four or more scars	.55

Foster[6] in a study of nine hundred and thirty-five cases of small-pox obtains similar statistics:

	PER CENT.
Of 144 cases with 1 scar, there were 17 deaths, or	11.8
Of 279 cases with 2 scars, there were 16 deaths, or	5.6
Of 187 cases with 3 scars, there were 3 deaths, or	1.6

Similar observations were made by Seaton[7] in a study of fifty thousand cases in children.

Revaccination was made compulsory in the Prussian army in 1834, and from that time the mortality from small-pox in Prussia became insignificant.[8]

			PER 100,000.
Thus, during the years	1825–1836	it was	36.4
	1837–1870		1.4
	1873–74–1886–87		0.05

4. A striking example of the effect of vaccination on the mortality from small-pox is furnished by the recorded deaths from small-pox in Madras, India, as shown in a paper by Sir William Moore.[9] The average annual mortality from small-pox at Madras from 1871 to 1884 was fourteen hundred and fifty-seven. In 1883 vaccination was made compulsory in Madras, and during the years 1885–1890 the mortality from small-pox was thirty-five.

The gradual stamping out of small-pox in Bavaria by vaccination and revaccination is well shown by Böhm.[10] Previous to the passage of the vaccination law in 1874 the average mortality from small-pox for sixteen years had been 17.5 per one hundred thousand, while since that time it has been 0.58; but this figure represents simply the average for the eighteen years, the present mortality being much less than this: thus, in 1890, 0.1; in 1891, 0.07; and in 1892, 0.05. It has not reached one per one hundred thousand since 1882.

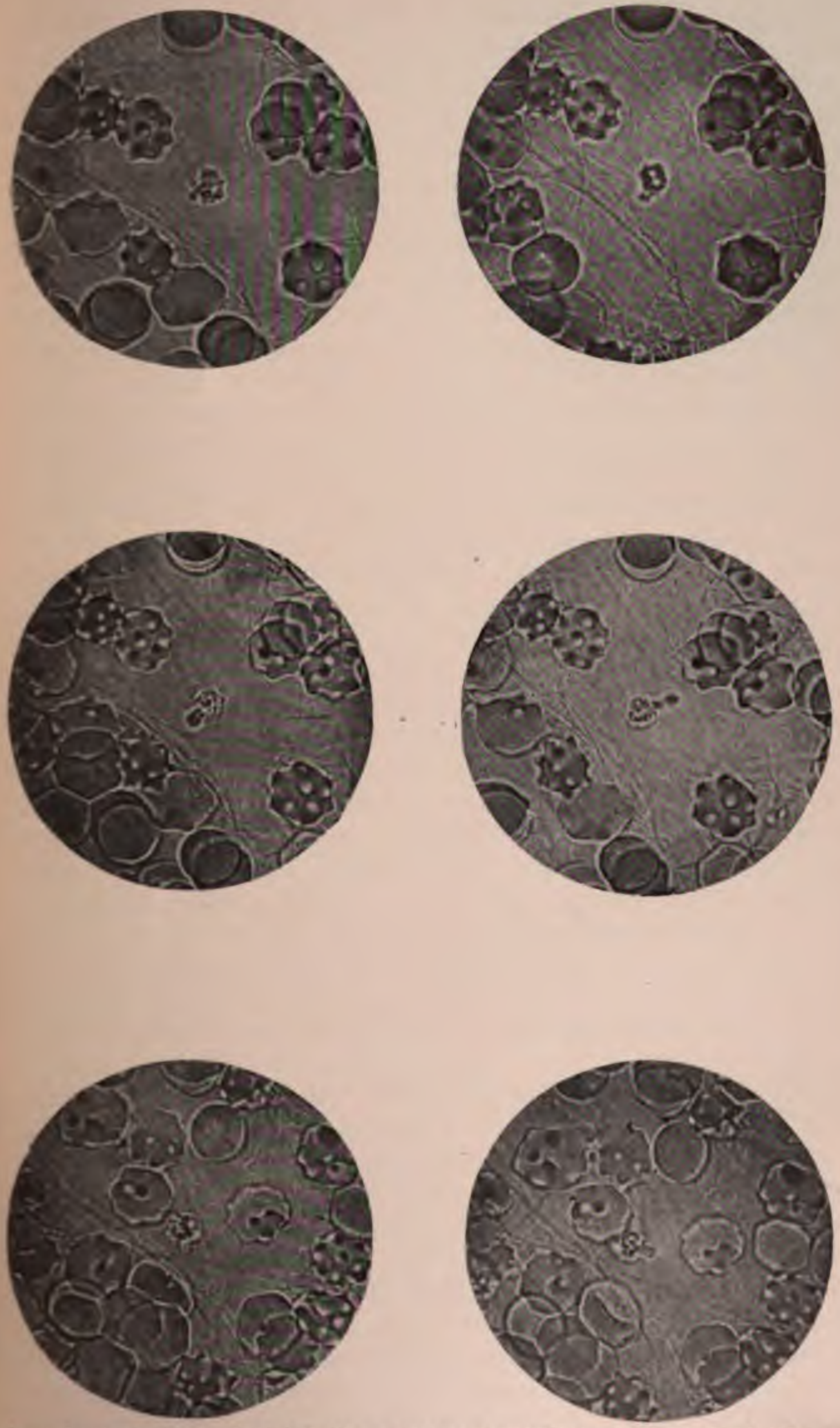
From these statistics it is evident that vaccinia confers a direct immunity from small-pox for a certain period, and that a normal vaccination followed by a good scar produces a more complete immunity than an irregular vaccination followed by a poor scar; also that revaccinations are necessary. At just what period such revaccination should be done has not been exactly determined. For five years after the first vaccination the immunity seems to be fairly complete. A second vaccination five years after the first would seem advisable, a third vaccination being done about ten years later.

THE RELATIONSHIP BETWEEN COW-POX AND SMALL-POX.—Although Jenner believed that cow-pox and small-pox were modifications of the same disease, he never presented further evidence than was afforded by the fact that inoculation of the virus of cow-pox protected against small-pox. Subsequently attempts were made to inoculate cows with small-pox. Gassner[11] in 1801 succeeded in one case in inoculating a cow, and inoculations of children from this cow were successful in producing ordinary vaccinia. Again, in 1836, Theile,[12] of Kasan, inoculated a cow from a small-pox patient. From the cow-pox thus produced three thousand persons were vaccinated, while with that produced in the same way by Ceely[13] twelve thousand were vaccinated, the ordinary symptoms of vaccinia following. More recently, Voigt,[14] of Hamburg, Hime,[15] of Bradford, and Simpson,[16] of India, have produced virus in the same manner. The latter obtained active virus from inoculation of lambs, cows, heifers, fowls, sheep, and calves. There can no longer be any doubt that vaccinia is a modified variola, the modification being effected by passing the virus through a less susceptible animal.

SERUM IMMUNIZATION.—An effort has been made recently to produce in the blood-serum of animals an antitoxin which might be used in the treatment of small-pox. Sternberg[17] found that the potency of vaccine virus was neutralized by subjecting it to contact with the blood of a calf immunized by vaccination. He thus found that such blood-serum did possess some degree of potency. Sternberg and Reed[18] appear to have established in a certain species of monkey an immunity from vaccination lasting thirty days by the injection of the serum of a vaccinated monkey. Considerable work has been done on this subject by Raynaud,[19] Chauveau,[20] Straus, Chambon, and Menard,[21] Copeman,[22] and Kramer and Boyce.[23] At present it is believed that the antitoxin exists in the serum of vaccinated animals in such small quantity that it is impracticable to obtain definite results from its use in man on account of the very large amount it would be necessary to use.

Etiology.—Many investigations have been undertaken in order to discover the micro-organism of variola virus. It was shown nearly thirty years ago by Chauveau[24] and Burton Sanderson[25] that the active agent was in the deposit or filtrate of virus, and not in the clear lymph. At about the same time Keber[26] described micro-organisms in vaccine. An attempt to grow the bacteria of vaccine was made by Quist.[27] His culture-medium consisted of equal parts of blood-serum, glycerin, and water. On examining the growth it was found to contain micrococci, and on injection into animals vaccinia was produced. It is not stated that subcultures from this produced vaccinia.

Buist[28] isolated three different varieties of micrococci from vaccine, which were characterized by the colors of their growth, as white, yellow,



A granular amœboid body, blood of monkey, eighth day of vaccination. Photomicrographs taken at intervals of about thirty seconds. × 1000.

and orange, but these were shown by Pfeiffer[29] to be identical with certain familiar species. Copeman[30] finds that the staphylococcus albus epidermis, the staphylococcus pyogenes aureus, and the staphylococcus cereus flavus are very common in vaccine, and these he believes to be the bacteria isolated by Buist. That being the case, they are undoubtedly contaminations and very undesirable bacteria. Copeman has described a bacillus which he has found in vaccine and variolous lymph and which he believes to be the active principle of vaccine. This bacillus will not grow on ordinary culture-media, but he states that he has succeeded in growing it on a hen's egg, and that vaccinations from it were successful. It seems quite possible that some active principle aside from the bacillus may have been thus transported first to the egg and then from it to the calf, for he does not state that he was able to carry it through several transplantations from one egg to another and then inoculate successfully. It does not seem to have been demonstrated that this bacillus stands in an etiological relation to vaccination. Buttersack,[31] working in the Hygienic Institute of Berlin, discovered in vaccine lymph, as well as in the fluid contents of fresh variola eruption, a filiform structure which he did not find in other clear exudates, such as the blebs following burns. This he considered to be the micro-organism of small-pox and the active agent of vaccination. Landmann[32] and Draer[33] consider that this appearance is not caused by a living organism.

Other observers have described protozoa as existing in the epithelial cells at the seat of vaccine inoculation. A micro-organism of this sort has been described by Pfeiffer,[34] also by Guarnieri[35] and Ruffer.[36] These bodies have not been demonstrated in the lymph.

Reed[37] finds small granular amœboid bodies, like those described by Pfeiffer, in the blood of vaccinated children and calves during the stage of fever, and also the blood of the monkey during the active stage of vaccinia, but he adds that a similar body may occasionally be found in the normal blood of monkeys and children. Reed also describes a pale amœboid body containing a few dark granules as present in the blood in cases of variola and in the blood of variolated monkeys. He found similar bodies in the blood of vaccinated children and monkeys.

Symptomatology.—The ordinary course of vaccination has been described in the previous article (see vol. i. p. 750.) A phenomenon, however, of fairly common occurrence with certain supplies of virus was not mentioned there,—that is, the raspberry excrescence. After some vaccinations no vesicle or areola follows, but in its place there is a small red, elevated nodule, which remains a few days and then disappears, leaving no scar. The significance of this phenomenon is doubtful. By some authors, as Welsh,[38] it is believed to be due to the inoculation of some non-specific material, while others, as Huddleston,[39] hold that it is due to a virus of low grade of activity, and that in some cases it does afford a slight degree of immunity against immediate revaccinations.

Complications.—The complications of vaccination, although compar atively rare, are sufficiently frequent to command attention. The writer has within two years performed autopsies on two children who died from septicæmia following vaccination and due apparently to contamination of the site of vaccination a week or ten days after inoculation. These cases seem to him the more serious since they are probably entirely preventable. The complications of vaccination are due to germ invasion in the wound caused by the vaccinator. The germs may be present in the virus used. Pyogenic bacteria are known to be frequently present in the virus as taken from the vesicles of the calf, and contamination of the dried ivory points still extensively used may occur in handling. Complications, however, do not seem ordinarily to arise from either of these causes, but from subsequent contamination of the unprotected site of vaccination. They usually appear a week or ten days or later after vaccination at about the period when the vaccination invites irritation on account of pain or itching. If then it is unprotected and a dirty finger-nail or a dirty shirt-sleeve is used for friction, the introduction of pathogenic bacteria is easily explainable.

The complications of vaccination have been enumerated by Rahts,[40] as follows:

(*a*) Severe inflammation of the neighboring skin.
(*b*) Swelling and inflammation of the neighboring lymph-nodes.
(*c*) Inflammation and suppuration of connective tissue.
(*d*) Erysipelas.
(*e*) Ulceration and a gangrenous character of the pustule.
(*f*) Blood-poisoning.
(*g*) Syphilis.
(*h*) Acute and chronic skin-diseases.

It is readily seen that most of these would come under the single heading germ-invasion.

Syphilis is practically not seen as a result of vaccination. Jonathan Hutchinson[41] says that, although seven hundred and fifty thousand vaccinations have been made annually for several years, the Local Government Board has not found a single instance of the communication of syphilis by vaccination.

In Saxony nearly two hundred thousand vaccinations were done in 1888.[42] Of complications erysipelas was not rare, and there were some cases of impetigo contagiosa. There were five deaths, four from erysipelas and one from sepsis. Again, in 1890,[43] in two hundred and twenty thousand vaccinations four deaths followed. This report mentions among the complications severe inflammation in the neighborhood of the vaccination, due probably to the irritation of the clothing; also suppuration of the lymph-nodes, pemphigus, and erythema. Of fourteen cases of skin eruption occurring in cases under the supervision of the New York Board of Health,[44] there were nine cases of roseola, three of erythema, and two of impetigo contagiosa.

THE VIRUS.—Although it must be admitted that we do not yet know the micro-organism of vaccine virus, still, scientific advancement in the production of virus has certainly been made in recent years, so that now vaccination may be a much safer operation than formerly. Bovine virus has largely supplanted that of human origin, and this fact has helped the scientific development of the subject, since the methodical production of virus in calves in large amounts can be carried on much better and with less danger of contamination than could be accomplished in the collection of crusts from vaccinations in man.

The bovine virus produced at different establishments has differed materially in the results produced, its efficiency depending largely on the method of obtaining it. Virus may be obtained in calves from the crust, from the underlying pulp or base, or from the serum beneath. One supply of virus formerly largely used was obtained from the serum, and the use of this virus was characterized by many negative results in primary vaccination and by the frequent production of the raspberry excrescence. Other supplies are characterized by an almost uniform, very severe reaction, which causes a suspicion that they may contain a great many pyogenic bacteria.

Tests were made by the experts of the New York Board of Health under Dr. Biggs,[1] the director, of the crusts, pulp, and serum of the vaccine lesion in calves, in order to determine which furnished the most active virus. It was found that the pulp was by far the most efficient; next in order of efficiency came the crust, and last the serum. The pulp was thus shown to be the only portion of the lesion that should be used.

Formerly virus was dispensed on quills. These quills were then superseded by ivory points, which afforded some advantage in convenience for use. At the present time these are being superseded by a glycerin vaccine preparation which is dispensed in closed capillary glass tubes. This last method presents several advantages over the others. One advantage is in the fact that the virus is sealed in capillary tubes in the laboratory and is not opened until it is put in use, while, on the other hand, the ivory points and quills were subject to contamination whenever handled. Another advantage is that the virus which is mixed with forty or fifty per cent. of glycerin seems to improve in quality on being kept a number of weeks or months. At the same time there occurs in the virus a constant diminution in the number of bacteria present, until after about one month the virus is usually, though not always, sterile. This phenomenon, the diminution of contaminating bacteria in the glycerinated virus, has been noted by Copeman[45] in England, Straus,[46] of Paris, Leoni,[47] of Rome, and Lambert,[48] of New York. Glycerinated lymph is just coming into use in this country, but has been in use in England, France, and Germany for some time.

As an example of scientific procedure in the production of vaccine virus the writer quotes the following tests which are used by the New York Board of Health in the production of virus:

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[1] The writer is indebted to Dr. H. M. Biggs for the use of material which is to appear in the New York Board of Health Report for 1897.

1. The animal in which the virus is produced is killed, and a careful autopsy is performed in order to make sure that the animal was healthy.

2. A sample of the virus is sent to a bacteriologist for examination.

3. A sample is sent to a medical tester, and by him is inoculated three times into each of five children who have never previously been vaccinated.

Virus is dispensed only when all these tests are satisfactory, and when every one of the fifteen inoculations is successful.

This virus is now dispensed only in capillary tubes, each tube being accompanied by a needle, a spreader, and a rubber tube for expelling the virus from the capillary glass tube. The results of vaccination with this virus furnish figures which are, so far as is known, unprecedented. In one thousand five hundred primary vaccinations made in three months the public vaccinators have not known of a single failure.

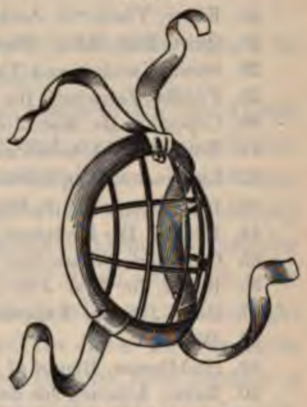
METHOD OF VACCINATING.—Since vaccination is an operation which is occasionally followed by annoying complications, and in very rare instances by death, it is important that it should be performed with as much care as operations of a more serious nature.

A virus should be chosen which is known to be produced in a cleanly and scientific manner and which is tested before it is dispensed. The site of the vaccination should depend on the age of the patient. In primary vaccinations in babies the writer believes that the best location is that part of the leg just below and external to the tubercle of the tibia. It is not, at that age, a dependent portion of the body, and it is much easier to get at than the arm. In adults the arm over the point of insertion of the deltoid is preferable. In those cases in adults where, to avoid disfigurement, vaccination is done on the leg, the case should be watched, and if a severe reaction ensues the patient must be kept in bed.

The seat of operation should be thoroughly cleansed with soap and water and dried. The scarification may be made with a sterile needle, or with several sterile needles mounted together. From four to six scratches through the epithelium should be made parallel to one another and about three-eighths of an inch long; another similar set being made at right angles to these and intersecting them.

These scratches should be so deep that serum exudes, but not deep enough to cause bleeding. Both ends of the capillary tube containing the virus should now be broken off with sterile forceps or cut with sterile scissors, and the rubber tube should be drawn over one end. The virus should be forced out through the uncovered end of the capillary glass tube on to the area of scarification, either by pressure along the rubber tube with the fingers or by blowing through the rubber tube. If the latter method is used, care must be taken that no saliva enters the tube. The virus should be rubbed into the scarified skin with a sterile instrument. The drying of the scarified area may be hastened by blowing air over it with a rubber bulb. The subsequent treatment of this wound differs largely with different physicians. The very large majority of vaccinations receive no more treatment. The clothing is drawn down over the wound, and the patient takes the risk of a chance infection. It is the writer's opinion that all vaccination wounds should be guarded for two or three weeks. Heavy dressings should not be used, as they cause a softening of the scab and a profuse discharge. A protection may be afforded by a piece of felt plaster with a circle one and a half inches in diameter cut out, which thus surrounds the site of vaccination. Over this two thicknesses of gauze bandage may be applied. A better device for protecting the site of vaccination is a wire shield bound over it. Such a device is supplied in the Cowan shield. (Fig. 1.) A vaccination shield should be used only once and then thrown away. Two or three thicknesses of gauze bandage over the shield will hold it firmly and add to the protection without causing softening of the scab. All vaccinations should be inspected on the sixth day and at intervals afterwards, and the shield should not be left off until all irritation from the vaccination has subsided.

FIG. 1.



The Cowan shield.

Should a primary vaccination give a negative result, the operation should be immediately repeated. In those who have been previously vaccinated a negative result does not necessarily indicate the need of immediate revaccination, since the individual may still possess a degree of immunity as the result of his earlier vaccination.

In conclusion, the following important points may be summarized as essentials in vaccination:

(1) An effective and properly prepared virus.

(2) A careful operation.

(3) Attention to secure freedom from chance infection.

(4) Intelligent watching of the local lesion, with renewal of the vaccination if necessary.

Such vaccination should be effective, free from complications, and safe.

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ERYSIPELAS.

BY JOSEPH McFARLAND, M.D.

ERYSIPELAS is a specific, infectious, inflammatory affection, caused by the streptococcus erysipelatis (pyogenes) of Fehleisen.

Ordinarily it attacks the subcutaneous cellular tissue of the face (facial erysipelas), and begins at points where the skin and mucous membranes join, as the alæ nasi and external canthus and external ear. The characteristic lesion is the so-called erysipelas "patch," which forms a sharply circumscribed, distinctly elevated, rose-red, slightly œdematous, tabular swelling. The skin over this patch is burning to the touch; the red color disappears on pressure, to return when the pressure is relieved, and a slight pitting follows the pressure. Upon the surface of the skin the formation of vesicles and blebs is not uncommon. When present they contain a slightly turbid fluid. In malignant cases gangrene of the skin occasionally develops.

The onset of the disease is sudden and the development of the patch rapid, the invasion of the skin taking place almost visibly. The invasion may be progressive, or it may soon limit itself. Sometimes the extension of the disease to new areas is marked by the recovery of the areas first affected. Later the disease may return and reoccupy the original seat.

The local disease is accompanied by profound constitutional disturbances. The onset is generally marked by the occurrence of rigors, headache, and sometimes vomiting. Later there is fever, which may develop into marked hyperpyrexia; delirium is common. Albuminuria nearly always develops, and probably depends upon either bacterial processes in the kidney or the attempt at the elimination of the toxins produced by the streptococci.

The specific bacteria enter the circulation in severe cases, disseminate themselves throughout the organism, and, by lodging in the capillaries of the organs, give origin to abscesses in the lungs, kidneys, and spleen especially. The capillary emboli may also lodge in the liver with miliary abscess formation. Malignant endocarditis is a most serious complication.

In uncomplicated cases the disease gradually subsides, the constitutional [illegible] [illegible], and with their ultimate disappearance the "patch" also disappears, leaving the skin quite normal.

The superficial form known as facial erysipelas may under certain circumstances occur upon other parts of the body. In infants it occurs at the umbilicus and in the neighborhood of the vulva and anus.

In some cases the specific cause of the disease enters wounds deeper than those of the skin and brings about extensive sloughing, sometimes accompanied by an erysipelatous appearance of the edges of the wound and a rapid invasion of the superficial lymphatics. This form—surgical erysipelas—was at one time the most dreaded complication of wounds, and in its most grave form appeared as hospital gangrene. Since the days of antiseptic surgery the disease has become unknown.

Puerperal erysipelas, which is recognizable only by a bacteriological examination of the lochial discharges, is one of the most malignant forms of puerperal sepsis.

Certain pseudo-membranous inflammations of the pharynx and larynx are found to occur entirely without the presence of Klebs-Loeffler bacilli, the cause in these cases being the streptococcus pyogenes, the organism now almost certainly demonstrated to be the cause of erysipelas. Hirsh and Libman have found streptococci in the stomach and stools in infantile enteritis, and late in the course of the affection in the urine and blood of the living child and in the internal organs of children dead of the disease. In such cases it seems as if the streptococci were the cause of the disease.

Cause.—It was Koch who first discovered the presence of streptococci in the serum of the erysipelatous patch, and Fehleisen who first succeeded in cultivating them and with the cultures producing the disease experimentally.

Since these early investigations, the streptococcus pyogenes has been found not only in erysipelas but also in suppurations, phlegmons, sepsis, puerperal fevers, lymphangitis, angina, pneumonia, periostitis, otitis, meningitis, endocarditis, and empyema. It also causes secondary infections in ulcerative phthisis, diphtheria, and scarlatina.

Morphology.—The bacterium of Koch and Fehleisen is a small coccus (.4 μ in diameter), habitually occurring in pairs and chains. The appearance of the chains of cocci is pretty, and highly suggestive of a string of beads. Generally there are from six to ten individuals in a chain, though there may be from ten to twenty, and even as many as a hundred. Von Lingelsheim has attempted to divide the streptococci into species whose chief peculiarity depends upon the length of the chain. His classification is not generally accepted. The cocci are not motile. A curious morphological peculiarity is a rather marked irregularity of size and shape. In the same cover-glass preparation some of the cocci may be seen to be round, some ovoid; some are ovoid with the long diameters corresponding to the length of the chain, others with the short diameter in the length of the chain. In the same chain of cocci some individuals may be observed that are much larger than others. By some bacteriologists these large cocci are looked upon as permanent forms (arthrospores) for the propagation of the species. No spores of the familiar type have been observed.

Staining.—The cocci stain well with the ordinary aqueous solution of the aniline dyes, and are beautifully stained by Gram's method and by Weigert's fibrin method.

Cultivation.—The organisms can be cultivated with ease, though they are somewhat delicate and are easily killed by dilute or feeble germicides and moderate degrees of heat.

When growing upon gelatin plates the colonies of the streptococcus are small and translucent. Under the microscope they are seen to be irregular in outline, from the projecting chains that extend from the body of the colony upon the surrounding gelatin, granular in substance, and slightly yellowish in color. The colonies do not liquefy the gelatin, and do not grow large. In gelatin puncture cultures in tubes the streptococci grow along the path of the wire, forming a slightly opaque granular line, which a lens shows to consist of partly confluent colonies developed in the gelatin.

Upon agar-agar the appearance of the growth is characteristic. The bacteria grow in such a manner as to produce small, generally circumscribed, grayish, translucent colonies, which are numerous in and about the inoculating wire.

Upon coagulated blood-serum the appearance presented by the growing bacteria is much like that upon agar-agar, except that the colonies are whiter in color. No growth seems to occur upon potato.

The streptococcus grows well in milk, which is coagulated and digested by their energy.

In bouillon the development is rather slow, and varies somewhat with the individual or variety. Some streptococci cloud the bouillon evenly, but the more usual form (streptococcus pyogenes conglomeratus) of growth is one in which the bouillon remains clear, and clusters of streptococci are observed precipitated as rounded or irregular flakes and flocculi against the bottom and sides of the tube.

The organism is not sensitive to acids, and can be grown upon acid media. Acids are produced by the organism in its growth.

Virulence.—When cultivated artificially the streptococcus rapidly loses its virulence. Just why this happens it is as yet impossible to decide. It seems to be caused by the failure of the culture medium to supply the same chemical conditions that are found in the tissues of animals. Acting on this principle, Marmorek has recommended the use of a mixture of ascitic or hydrocele fluid and bouillon, in which, in his experiments, the bacteria remained virulent. Petruschky has found that the virulence will be retained if the culture be kept upon ice. Holst, however, found that one of his cultures remained virulent for *nine years* without any special attention. From the observation of Holst it would seem that the maintenance of virulence is more an individuality on the part of the organism than a lack of something in the culture medium. Frosch and Kolle assert that the vitality of the streptococci is retained longer when dry than when growing upon culture media. These experimenters find that the best method of preserving the virulence of the organism is to keep it in gelatin and renew the culture every five days. The cultures are kept on ice.

The virulence of the streptococcus can be increased to a remarkable degree by rapid passage through animals. Marmorek was able to produce a culture so virulent that one-hundred-millionth of a cubic centimetre would kill a rabbit. That increase of virulence was the outcome of persistent infections was well known to the surgeons of a few decades ago, whose frequent observation it was that as an epidemic of hospital erysipelas or gangrene raged it increased in malignancy.

It might also be pointed out that the permanent forms of the bacteria, whatever they may be, are not subject to attenuation of virulence when outside the body in a dry form. Here, again, the observations of the older surgeons are useful to us. They were familiar with certain wards, rooms, and beds in their hospitals where erysipelas always existed and infected every wound exposed there.

Avenues of Entrance.—The streptococci seem to enter the body through larger or smaller wounds. At one time it was supposed that in the idiopathic form no wound was necessary and that the disease originated spontaneously. It is now well recognized that the streptococcus is local in its action, and begins its destructive operations as soon as it enters the tissue; hence we must conclude that in the facial form, without apparent wounds, the avenues of entrance were simply too small to be discovered. The streptococci are probably carried into the deeper layers of the skin by the finger-nail in many cases, having been previously either upon the finger-nail or upon the skin. Surgical erysipelas was probably transmitted from case to case by knives, syringes, and dressings, and quite as frequently by the operator's finger. Puerperal erysipelas probably depends upon lack of cleanliness on the part of the accoucheur, whose finger carries the deadly germs into the uterus during his manipulations.

Contagion.—The disease being infectious and caused by such a well-known germ, it naturally follows that it can be communicated to all coming in contact with it in such a way that proper avenues of entrance are open to its specific cause. Probably the very old and the very *young* are more readily infected than others, but all seem to be susceptible, provided that the cause reaches them.

Experiments on Animals.—Fehleisen was the first to produce the disease in animals by inoculation.

Considering the acute and severe character of the disease, the effect of animal inoculations is often disappointing. A culture of ordinary virulence when introduced subcutaneously into rabbits or guinea-pigs may be followed by no results at all, or may give origin to a local erysipelatous inflammation at the point of injection, or may bring about the formation of a local abscess, or may cause the death of the animal.

When virulent cultures are injected into the ear-vein of a rabbit, the animal dies in a couple of days, partly from toxæmia and partly from general infection. Ordinarily it takes a couple of cubic centimetres of bouillon-culture twenty-four to forty-eight hours old to accomplish this end, though if the virulence be great much less may do it. The extreme virulence of Marmorek's culture, of which one-hundred-millionth of a cubic centimetre is fatal, has already been mentioned.

When the animals die, the cocci can be found in the blood and in the substance of most of the organs.

The injection of filtered cultures into animals is without effect except in large amounts, when fatal toxæmia results.

Morbid Anatomy.—When a section of the skin from an erysipelatous patch is examined under the microscope, stained in such manner as to render the cocci visible, they can be found in large numbers in the lymphatic spaces and vessels of the tissue. The tissue being œdematous, its lymph-spaces become more obvious than usual. They contain very numerous leucocytes, in whose protoplasm many of the cocci may be seen.

Metchnikoff is of the opinion that the leucocytes take up the cocci with intention of killing and devouring them. It has, however, not yet been demonstrated that the cocci were not dead before the leucocytes ingested them.

Frequently infecting emboli containing the streptococci lodge in the capillaries of the organs, producing, as a rule, local suppurative areas. Except for their infectiousness, the emboli are without interest.

The Effect of Erysipelas and its Toxins upon Morbid Growths.—For many years it has been the experience of the profession that the occurrence of erysipelas in chronic ulcers, whether tuberculous, syphilitic, or epitheliomatous, was likely to be followed, in the event of the recovery of the patient, by such complete disorganization of the diseased tissue of the depths and surroundings of the ulcer that spontaneous granulation and healing followed.

It was with the hope of making some practical use of this knowledge that the early experiments of Fehleisen, in which six patients suffering from various surgical affections were inoculated with pure cultures of the streptococcus, were made. The considerable danger attending the inoculation, which was almost as likely to produce the death of the patient as to bring about his convalescence from the morbid growth, caused the method to be abandoned. There are, however, numerous cases upon record to show that, except for its danger, the method was an excellent one.

To Coley belongs the credit of suggesting the use of the erysipelas toxin instead of the live streptococcus, in this way obviating the danger and still accomplishing the good. At first Coley used cultures sterilized by filtration, but found these to be much less active than cultures killed by heat and allowed to retain the dead bodies of the streptococci. Still later it was found that the addition of the harmless bacillus prodigiosus to the cultures rendered them much more efficient. The experiments of Coley seem to show—and, while his views have by no means received universal acceptance, evidence seems to be accumulating to support his conclusions—that in cases of inoperable sarcoma and carcinoma the proper administration of the powerful poison of the mixed erysipelas and prodigiosus cultures will cause the morbid growth to soften, sometimes to slough, and sometimes to be absorbed.

The effect of the toxin upon diseased tissue can by no means be regarded as specific, and the results of the treatment are far from being uniform in consequence. The explanation of the action of the toxin seems to be the low vitality of the neoplastic tissue, which, badly nourished at best and always more or less prone to degenerate, is either killed by the toxin or has its vitality so diminished that recovery from the effects of the toxin is impossible.

The injections are made into the tumor, but are followed by both local and constitutional symptoms. The local effects are swelling, redness, and softening of the tumor; the constitutional symptoms, fever and prostration.

Treatment.—With the development of the "serum-therapy" in the last few years came the impulse to investigate every field into which it might be introduced. Marmorek was the pioneer of this field, and has demonstrated several important facts not before known. In the main, the plan of his work has been on the lines originally laid out by Behring, and consists (1) in the preparation of a highly virulent culture to be used as a toxin, (2) in the immunization of a large animal, like the horse, to immense doses of the toxin, (3) in securing the blood and separating the serum, (4) in establishing a standard test by which the value of the serum can be ascertained.

First of all, Marmorek, by alternate passage through rabbits and cultivation in the mixture of ascitic fluid and bouillon, secured the culture already mentioned, of which one-hundred-millionth of a cubic centimetre was fatal to a rabbit. This culture was injected into horses, beginning with a fraction of a cubic centimetre and ascending until the animal received two hundred cubic centimetres or more, the injections being regulated carefully, so as not to destroy the horse. When it was supposed that sufficient immunity had been acquired, the horse having been under treatment the best part of a year, the blood was tested from time to time to discover its nature. For nearly two months from the last injection the blood remains toxic, but at last the organism reacts strongly, the toxic character of the blood changes, and it is found to contain a protective substance—an antitoxin—capable of saving the lives of rabbits against certainly fatal doses of the virulent streptococci.

This, in brief, is the method pursued by Marmorek, whose experiments have every appearance of thoroughness and whose results with the serum thus obtained have been brilliant. Thus far the method is extremely new, so that it is impossible to predict what fruition it may have.

It has already met with opposition from Petruschky, who, having obtained a culture of streptococcus from Marmorek, together with some of the serum, failed to find the culture as virulent as Marmorek claimed, and also failed to find that the rabbits he injected with the antitoxic serum were protected from the virulence of the cultures.

Erysipelas is a disease that is not susceptible of cure by any known treatment, and, therefore, one for which the use of a potent protective antitoxic serum would be a desideratum in medicine.

RHEUMATISM.

By W. B. CHEADLE, M.D., F.R.C.P.

DEFINITION.

With regard to this, it may be well to point out afresh that the term rheumatism is here used in its strictest sense, as indicating in its different phases that form alone which is distinguished as genuine, true, or acute rheumatism, of which, in the case of adults at least, rheumatic fever is the most extreme and typical expression.

Observations made since the publication of the original article in 1889 tend generally to confirm the view therein put forward and now widely accepted as to the comprehensive character of the disease and the variety of its manifestations, especially during the period of childhood. This is seen most clearly in the case of children, in whom arthritis is not the typical and representative symptom. At this period the joint-tissues are less susceptible than in later life, while the other fibrous tissues, the skin, and the nervous system are more so.

In the rheumatism of childhood the articular affection is at its minimum; endocarditis, pericarditis, subcutaneous nodules, and chorea at their maximum. As life advances this rule is gradually reversed: the joint-affection grows more prominent, constant, and typical, while the other phenomena decline and tend to die out. Endocarditis and pericarditis become less frequent; subcutaneous nodules and chorea, so common in connection with the rheumatism of childhood, almost disappear as maturity is reached. Rheumatism must be regarded not as a mere special inflammation or febrile affection of the joints, but as a disease or morbid condition in which arthritis is one symptom, but one only, among many others.

Etiology and Pathology.—The influence of chill, exercise, climate, the poison of scarlatina, hereditary tendency, age, sex, temperament, and microbic invasion were set forth in the original article. The only points which require further illustration are the influence of the nervous system in its production, the nature of the toxic agent, the effect of climate and season, of family predisposition, the identity of the scarlatinal form, and the influence of micro-organisms in the production of the disease.

CLIMATE AND SEASON.—With regard to this, the researches of Dr. Arthur Newsholme[1] have thrown much additional light. He has shown that acute rheumatism is most prevalent in dry seasons, when the rainfall is exceptionally scanty and the ground water at its lowest. So that a hot, dry season, and, if two follow in succession, the *second*, is the most productive of acute rheumatism, negativing the popular association of the disease with damp and cold. This, however, is not incompatible with the well-established fact that *chill* is a factor of the disease, for it is under just such conditions of dry atmosphere that rapid cooling of the surface of the body by evaporation occurs, especially on free perspiration after exercise.

INHERITED TENDENCY.—With regard to the influence of inherited tendency, further observations have strengthened the evidence of its potency as a factor of the disease. As was pointed out in the original article, statistics upon the point show great discrepancy, varying from twenty to fifty per cent. This discrepancy is due, no doubt, to differences in the thoroughness of the inquiry, the nature of the evidence allowed, and the class of patients from whom the evidence is obtained. Most statistics have been based upon information obtained from hospital patients of the poorer class, who have very imperfect knowledge of any but their immediate relatives or the diseases from which they suffer, and this often for a very limited period of their lives. Parents of the better class know all about their own children, much about their own brothers and sisters and uncles and aunts, although the history of the early years of the older generation may have partly passed out of recollection. Thus, far more complete and reliable family and life histories can be obtained from private patients than from hospital records. In thirty-two consecutive cases out of my private note-books, in twenty-three—that is, in seventy per cent.—there was a definite history of rheumatic fever. These statistics are based upon the occurrence of acute joint-symptoms alone; if other manifestations of rheumatism are taken into account, the proportion with rheumatic family history would, of course, be higher still.[2] In the original article, attention was drawn to the fact that when the inheritance is *double,—i.e.,* when both parents are of rheumatic stock,—the proclivity of the offspring to the disease is greatly intensified, and its severity and persistence also. The following additional case which came under the writer's own observation affords the most striking illustration of this doubled intensity.

The patient, a girl nine years old, the child of a medical man, had chorea in its most severe form; the jactitation was violent in the extreme; speech was entirely lost for eighty-one days, and feeding was very difficult. Repeated attacks of endocarditis, pericarditis, erythema, paresis of limbs, acid sweats, and pains in the joints followed, and successive crops of subcutaneous nodules in such profusion that no less than two hundred were stated to be present at one time. Finally, in spite of all treatment, at the end of nine months of almost continuous illness the child died of cardiac dilatation and failure,—a case of extreme, inveterate, uncontrollable rheumatic affection in all its phases. The family history was charged with rheumatism on both sides. The father had subacute arthritis and muscular rheumatism; his sister died when eight years old of heart-disease after acute rheumatism and chorea; his wife, the patient's mother, had had acute rheumatism, heart-disease, and chorea; her nephew—a cousin of the patient—had rheumatic fever and heart-disease, and a niece, subacute rheumatism.

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[1] Milroy Lectures, Royal College of Physicians, 1895, Lancet.

[2] *Vide* address by the author in introducing the Discussion on Acute Rheumatism at the meeting of the British Medical Association, 1895, Brit. Med. Jour., January 12, 1896.

SCARLATINAL RHEUMATISM.—The view that the arthritis and cardiac inflammations which arise in connection with scarlet fever are truly rheumatic has received fresh support from the discovery of subcutaneous nodules in the scarlatinal form by Dr. Drewitt and Dr. Stewart.[1]

General Pathology.—A survey of the varied phenomena of acute rheumatism points to the presence of some pervading systemic influence acting upon the tissues, and the question as to the nature of the morbid agent is one which has constantly exercised the minds of pathologists. Is the virus a simple chemical product of metabolism, or the product of some micro-organism introduced from without? or, as some have held, is there no virus at all engaged, and is rheumatism merely a reflex nervous phenomenon, due to peripheral irritation by chill, transferred to some hypothetical nervous centre which controls the nutrition of joints?

THE NEUROPATHIC THEORY.

This latter hypothesis, suggested by the joint-disease of Charcot, rests upon slender foundation. Trophic lesions have been traced to injuries of nerve centres and afferent nerves, but, so far as the writer knows, not to reflected irritation. There is no evidence that reflex irritation will give rise to any such lesions, although this theory might explain chorea and possibly a local arthritis. It would not, however, explain endocarditis, or pericarditis, or pleurisy, or subcutaneous nodules.

Although, then, the nervous system may play some part in the phenomena of rheumatism, there is no evidence that it plays, or is capable of playing, a leading part in their production.

TOXÆMIA.

The hypothesis that the toxic agent is a chemical product is favored by the analogy of gout, in which uric acid is a factor, and to which acute rheumatism has many points of close resemblance, in its symptoms, in its varied phenomena, in its irregular course, in its fitful recrudescence, and in the influence of inherited predisposition. Among the most recent theories upon this basis are those of Dr. Latham[1] and Dr. Haig,[2] both founded upon the assumption that uric acid is formed in excess and accumulates, with, according to the former, the addition of lactic acid.

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[1] Garrod, Treatise on Rheumatism, 1890.

There appears, however, to be no satisfactory proof that either uric acid or lactic acid exists in excess in acute rheumatism, either in blood, or tissues, or sweat, or urine. The chemists are not agreed on this. Further, clinical experience is opposed to any direct relation between rheumatism and gout. Yet if both these diseases are due to the presence of this same poison of uric acid, there ought to be a distinct clinical connection between them. But neither gouty people, nor people of gouty race, are subject to acute rheumatism; nor, on the other hand, are rheumatic people, or people of rheumatic race, subject to gout. One disease occurs in a very different class of patients, of different habits and of different environment from the other.

Again, if the *materies morbi* is the same in the two diseases, why are its effects so different in most respects? If uric acid causes no acute serous inflammation of pericardium or pleura in the case of *gout*, why does it set up pericarditis and pleurisy and endocarditis in *rheumatism?* These theories, ingenious enough in themselves, cannot be accepted until uric acid is proved to be generated in excess or to be present in excess in acute rheumatism.

THE MICROBIC THEORY.

The view that the varied phenomena of acute rheumatism are due to the invasion of some micro-organism from without has latterly met with increasing acceptance. It has some analogies in its favor, and has been advocated with great ability by Dr. Newsholme in the Milroy Lectures at the Royal College of Physicians for 1895. Its occasional epidemic prevalence, its variability of type, the incidence on the young, the concurrence of endocarditis, of pericarditis, of pleurisy, of pneumonia, of erythematous eruptions, the rapid anæmia, the tendency to purpuric capillary hemorrhages, the implication of joints, the occasional supervention of hyperpyrexia, the nervous disturbance, and the specific influence of one drug,—viz., salicylic acid,—are all suggestive of an infective disease. In pyæmia, indeed, we have often a similar series of pathological events,—arthritis, endocarditis, pericarditis, pleurisy, pneumonia, erythematous rashes, sweating, and prolonged course. The difference lies chiefly in the absence of the nervous disturbance of chorea, the tendency to suppuration, the continuous progress without periods of decline and of recrudescence, the failure of control by any specific drug, the extreme mortality. There are, again, as Dr. Maclagan has shown, many points of resemblance between acute rheumatism and intermittent fever, more especially in the want of any definite period of incubation, the non-communicability from one person to malaria, the proneness to recur, the long course, the yielding to a specific drug. On the other hand, there is no inflammation of joints and viscera in ague, and, as Dr. Newsholme's observations show, rheumatism is not, like malarious disease, an affection of wet and damp, but of dry subsoil.

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[1] Croonian Lectures, 1886, Lancet.

[2] Wood's Medical and Surgical Monographs, February, 1890.

The fact that rheumatism is directly excited by chill is not incompatible with its infective character, for it has its parallel in the case of pneumonia; while the fact that one attack of rheumatic fever confers no immunity, but rather favors recurrence, although it differentiates acute rheumatism from small-pox, measles, scarlatina, and other eruptive fevers, is only what occurs in certain other infective diseases, as diphtheria, ague, erysipelas, and, above all, influenza. Again, the family proclivity to the disease, which is so marked a feature in the etiology of acute rheumatism, is not conclusive against its infective nature, since a similar hereditary proclivity exists in the case of tuberculosis, and possibly also with regard to scarlet fever, diphtheria, and typhoid. The absence of any proof of transference by infection from person to person is, however, an objection of some importance.

A number of cases have, indeed, been recorded by Mantle,[1] Feissinger,[2] Edlefsen,[3] Friedländer,[4] Thoresen,[5] and others where certain houses have appeared to be especially associated with outbreaks of rheumatism, and a few instances have been observed which suggest the possibility of communication from person to person. The value of the evidence is, however, impaired by the consideration that such occurrences are rare, and that in many instances the patients were members of the same family, living under similar conditions. In view of the powerful influence of heredity and locality, this rare concurrence cannot have great weight. The fact that no specific organism has yet been satisfactorily identified is one of the chief difficulties in the way of the acceptance of the microbic origin of acute rheumatism. Micro-organisms have been found by many observers, as Cornil and Babes,[6] Wilson,[7] Mantle,[8] Petrone,[9] Popoff,[10] Hirschfeld,[11] Bouchard and Charrin,[12] Triboulet,[13] Sacaze,[14] and others.

They have been found in the vegetations of the endocardium, in pericardial exudations, in serum, in synovial fluid, yet they have been diverse,—some pyogenic, some supposed specific forms,—different in different cases as seen by different observers. Each microbe appears as witness against the rest. Moreover, the disease has not been produced in man by inoculation, and the experiments on animals are inconclusive.

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[1] Brit. Med. Jour., June 25, 1887.

[2] Gazette médicale de Paris, No. 14, p. 160, April, 1892.

[3] Zur Statistik und Aetiologie des acute Gelenk-Rheumatismus.

[4] Discussion on above paper.

[5] Thoresen, Om den acute Cedrheumatisme, Norsk Mag. for Lagevidenskalen, 1879, R. 327, 3 Bd. 9.

[6] Les Bactéries, i., 3d ed., 1890.

[7] Edin. Med. Jour., June, 1885.

[8] Brit. Med. Jour., June, 1887.

[9] Gazette des Hôpitaux, 1888, p. 881.

[10] Med. Pubalonia k. Morskinü Stonukü, November, 1887.

[11] Wiesbaden Med. Cong., 1888.

[12] Association française pour l'Avancement des Sciences, 1891.

[13] Revue des Maladies de l'Enfance, 1891.

[14] Archives générales de Médecine, November, 1894.

It would appear, however, that the organisms most usually found are staphylococci,—pus-cocci,—and it has been suggested that the disease is set up by various micro-organisms, as in the case of ulcerative endocarditis.

Sternberg, in his recent work,[1] after reviewing the results obtained by bacteriological research in acute rheumatism, comes to the conclusion that the disease is probably due to infection by pus-cocci, and that the natural immunity to the disease which exists is due to a germicidal substance in the blood, which has its chief origin in the leucocytes, and is possibly soluble only in an alkaline medium. He suggests that in acute rheumatism there is an excess of acid in the system, and that, as a result of this, the natural immunity against infection by these micrococci is neutralized.

It may be asked, however, in this relation, why, if pus-organisms are the active agents of the disease, suppuration in any form should be so conspicuous by its absence.

Possibly, as Buss[2] suggests, they are pyogenic organisms which have lost much of their virulence and their specific pyogenic action.

Buss, Wade,[3] and others have suggested that, in view of the close connection between tonsillitis and acute rheumatism, the tonsils are the channel by which infection enters, as in diphtheria, and probably in scarlet fever.

With regard to the pathological condition of the blood in acute rheumatism, Hayem[4] has called attention to the marked leucocytosis which exists in acute attacks. The number of leucocytes in some cases reaches twenty-five thousand, whereas in mild cases the number rarely rises to more than seven or eight thousand.

ENDOCARDITIS.

The association of acute endocarditis with the rheumatic state is, of course, unquestioned. The closeness of such association has, however, been variously estimated and often greatly underrated. As previously stated, statistics from private cases, when the family and personal history can be traced most completely and accurately, are more reliable than those collected from the poorer class, which are on these points imperfect, and, consequently, misleading.

The private cases in my note-book, in which the family and personal history has been minutely examined into and recorded, have been recently collated by my friend Dr. Poynton, with the following result as to endocarditis. Of ninety-four consecutive cases, in sixty-two—*i.e.*, in sixty-six per cent.—there was a history of acute rheumatism in either the patient or in his immediate blood-relations. If chorea and morbus cordis are accepted as evidence of rheumatic implication, the numbers are seventy-three out of ninety-four, or seventy-seven per cent.

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[1] Text-Book of Bacteriology.

[2] Deutsche Archiv für klinische Medicin, 1894.

[3] Brit. Med. Jour., 1895, vol. i. p. 829.

[4] Du Sang et de ses Altérations anatomiques.

Further, if we except those rare cases in which endocarditis occurs in association with the eruptive fevers or with pyæmia, the only pathological connection of endocarditis is with acute rheumatism and chorea.

Are the cases which occur in association with chorea alone, apart from any other rheumatic manifestation, instances of rheumatic endocarditis?

Antecedent probability is largely in favor of a rheumatic connection. The cardiac affection of chorea is as a rule organic. This was fully established years ago by the careful clinical observations of Dr. Stephen Mackenzie[1] and Dr. Osler.[2] Moreover, endocarditis is the one constant morbid change met with in fatal cases of chorea; and it is allowed to be rheumatic in those cases of chorea where it arises in association with other rheumatic symptoms, such as arthritis or nodules. The most potent argument in favor of the rheumatic origin of the endocarditis of chorea, indeed, is afforded by the pathological associations of the two apart from each other. The especial morbid conditions with which endocarditis is associated are rheumatic,—viz., arthritis, pericarditis, erythema, and, above all, subcutaneous nodules. Chorea is associated in like manner with each of these same rheumatic phases,—sometimes with one or other only, sometimes with several, together or at different periods.

DILATATION OF THE HEART.—The view that in many cases of rheumatism the mitral murmur which so frequently arises is due to simple dilatation of the left ventricle causing mitral leakage, without any organic change in the valve-flaps themselves, and analogous to the well-known pulmonary hæmic murmur, has been recently advanced by Dr. Theodore Fisher.[3] I have seen one case of fatal rheumatic cardiac dropsy in a child in which dilatation of the cavities was the sole lesion found post mortem.

That this simple dilatation alone, without valvular inflammation, is not a common source of mitral murmur is shown by the constancy with which persistent mitral bruit and evidence of organic valvular affection eventually follow.

That dilatation does occur in the early stage of rheumatic fever, as evidenced by increase of cardial dulness, has been shown by Dr. Lees.[4] He finds that this dulness may extend as much as two fingers'-breadths beyond the right edge of the sternum, the same distance beyond the left nipple-line, and upward to the level of the left second costal cartilage.

This is found in cases free from pericarditis. Dr. Lees is inclined to regard the dilatation as a special result of the enfeebling influence of the rheumatic poison. He states that a similar condition may occur in chorea, and this has been confirmed by a series of careful measurements of the cardiac area made by Dr. Poynton at the Hospital for Sick Children, Great Ormond Street, but not yet published. In this relation also it may be useful to refer to some recent observations by Dr. Ewart as to the physical signs which distinguish dilatation from pericardial effusion, although a copious serous exudation into the pericardium is rare in the rheumatism of childhood.

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[1] Trans. Int. Med. Cong., 1881, vol. iv. pp. 100–104.

[2] Amer. Jour. of Med. Sci., vol. xciv., New Series.

[3] Brit. Med. Jour., July 18, 1896.

[4] Lancet, July 25, 1896.

PERICARDITIS.—The tendency of this affection in children to be subacute, chronic, insidious, and recurrent rather than acute was insisted on in the original article, as well as the frequency with which it occurs in the later stages of valvular affection, when the heart has already become dilated and hypertrophied. As was pointed out by Dr. Sturges,[1] in his Lumleian lectures at the College of Physicians in 1894, pericarditis is almost invariably found post mortem in the fatal heart-disease of children.

The characteristic protracted, intermittent, smouldering pericarditis of children leads slowly to thickening and adhesion. After several recurrent attacks the friction, which has returned from time to time, appears no more, rendered impossible by the adhesions and obliterations of the pericardial cavity, and this may lead to the continued existence of pericarditis being overlooked, especially in the late cases, where the heart has become already greatly enlarged. If, however, the child is restless, uncomfortable, vomits occasionally without obvious reason, has pain in the præcordial region, with some tenderness on firm pressure there; if the temperature remains slightly raised, say to 99° F. or 100° F., and the pulse quickened to 120 or 130, without arthritis or other cause to account for it, with increasing feebleness, emaciation, and anæmia, continued pericarditis may be suspected. This quickened pulse-rate, with slight pyrexia insufficient to account for it, is very characteristic of the subacute, recurrent pericarditis of early life. The inference as to its existence would be strengthened by any fresh eruption of subcutaneous nodules.

PERICARDIAL AND MEDIASTINAL FIBROSIS.

The chronic inflammatory process, beginning in the pericardium, in some instances spreads from the pericardium proper to the tissues of the adjacent pleura and mediastinum, so that they become matted together in a dense fibrous mass. This indurative mediastino-pericarditis is indicated clinically by ascending dulness along the middle and upper sternum, and by increasing dyspnœa and cardiac distress. In a few cases the fibrous growth causes pressure upon the great vessels at the base of the heart. Such pressure is most operative upon the veins, and leads to hepatic engorgement, cirrhosis and ascites, imperfect filling of the pulmonary arteries, cyanosis, dyspnœa, and general dropsy. These extreme results of pericardial and mediastinal fibrosis are rare, but several instances, verified by post-mortem examination, have come under the writer's observation, and others have been recorded.

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[1] Brit. Med. Jour., 1896.

Another result of this chronic fibrous pericarditis of childhood has escaped general recognition owing to its extreme rarity,—viz., the effect upon the growing heart itself. The tight, strangling grip of the inelastic fibrous sac not only interferes with the dilatation and contraction of the cardiac chambers, and thus causes grave embarrassment of the mechanism, but when it occurs early and the adhesion is complete it interferes with the progressive development of the organ, so that the heart fails to grow in proportion to the rest of the body, remains of disproportionate smallness, unequal to the work of passing the blood forward, and general dropsy follows. Instances of the kind have been recorded by Watson and Bouilland, and two have come under the writer's personal observation.

TONSILLITIS.

The association of tonsillitis with acute rheumatism has formed the subject of some recent papers by Buss,[1] Wade,[2] and Goedel.[3] As previously noted, it is suggested that it is through the tonsils that the infecting agent finds entrance, and the symptoms of rheumatic fever are the outcome of the absorption of microbic products.

SUBCUTANEOUS TENDINOUS NODULES.

The character of these and their diagnostic value were fully explained in the original article. It may be added that they have been observed not only in connection with tendons and fasciæ, but also in the periosteum in a few instances, and even in the pericardium itself,[4] and that in one instance under the writer's observation as many as two hundred were calculated to have been present at one time. It was previously stated that the connection of these nodules with rheumatism was apparently absolute. Further experience has shown that this term must be expanded to include rheumatoid arthritis. Two cases of typical rheumatoid arthritis have come under the writer's observation; during the more active stage of pain, tenderness, and swelling, a crop of typical nodules was developed. Both these cases were in adults, and, contrary to the rule in children, were distinctly tender. Other examples have been recorded by Duckworth,[5] Howard,[6] Pitt,[7] Bannatyne,[8] and Futcher.[9] Such instances are, however, rare, and most rare in children. The two cases mentioned as in the writer's experience were both in adults. One reason, probably, why these nodules are so seldom met with in rheumatoid arthritis lies in the fact that nodules usually occur in children, in whom rheumatoid arthritis is most uncommon.

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[1] Deutsche Archiv für klinische Medicin, 1894, Bd. liv. S. 1.

[2] Brit. Med. Jour., 1895, vol. i. p. 829.

[3] Deutsche Med. Woch., 1896, Bd. xxii. S. 259.

[4] Money, Lancet, 1886, vol. ii. p. 158.

[5] Clin. Trans., vol. xvi. p. 52.

[6] Pepper's System of Medicine.

[7] Clin. Trans., vol. xxvii. p. 54.

[8] Rheumatoid Arthritis, p. 113.

[9] Johns Hopkins Hospital Bulletins, 1895.

ERYTHEMA NODOSUM.

Some additional evidence to that adduced in the original article in favor of the connection between erythema nodosum and rheumatism is furnished by the statistics of Dr. Stephen Mackenzie. He records one hundred and eight cases; acute or subacute arthritis occurred in sixty-seven, and, what is significant, in two instances endocarditis developed with the eruption, but without any joint-affection. Again, in twenty consecutive cases recorded by Dr. Garrod, eleven had arthritis and six a family history of rheumatism.

CHOREA.

The close connection between chorea and rheumatism has received additional support from evidence which has accumulated during the past few years. As previously set forth, acute rheumatism is the only general disease or fever with which chorea has any association, with the single exception of scarlet fever, and scarlet fever, significantly enough, is the one fever in connection with which acute rheumatism arises.

The scarlet-fever chorea is essentially a rheumatic chorea. Cases of chorea after measles and after diphtheria have been recorded by Henoch, but they are, to say the least, of extreme rarity. The writer has never met with an instance of either, any more than with one of mumps chorea or whooping-cough chorea.

The chorea of pregnancy likewise must be regarded as chiefly, if not solely, a rheumatic chorea, since in the majority of cases it occurs in association with rheumatic fever, or in patients who had previously suffered from rheumatic fever or chorea. In fatal cases, moreover, endocarditis is almost universally found post mortem. In thirty-four cases of chorea gravidarum, collected by Dr. McCann, there was a previous history of rheumatic fever in thirteen, and in eleven more of previous chorea.

Chorea is especially associated with the other rheumatic phenomena as well as with articular rheumatism, with endocarditis, pericarditis, erythema, and the special rheumatic symptom, subcutaneous nodules. Chorea may appear in relation to one or more of these apart from any affection of the joints. The association of chorea with pericarditis alone without any accompanying arthritis is especially significant.

Again, chorea which occurs apart from any other rheumatic manifestation at the time is sometimes followed, instead of being preceded, by arthritis or other rheumatic feature. Moreover, chorea, when unattended by other rheumatic symptoms, is especially liable to occur in members of families in which rheumatism is rife. The statistical evidence previously adduced is supported by later figures taken from patients of the better class, from whom more reliable family histories can be obtained than from the poor.

Out of fifty-five consecutive cases of chorea in private patients compiled from the writer's note-book by Dr. Poynton,—in all of which the family history was minutely investigated,—there was a definite history of acute rheumatism in the patient or near blood-relations in twenty-seven,—*i.e.*, in fifty-two per cent.; in two more there was a family history of chorea; in one, of morbus cordis; in two, of erythema nodosum and chorea. If these latter are accepted as evidence of rheumatic taint, the numbers would be thirty-three out of fifty-five, or sixty per cent. Now out of this total of fifty-five cases of chorea ten are cases of simple grimacing,—*i.e.*, of false or face chorea associated with second dentition. These are, no doubt, usually included in such estimates, and are so included above. If they were excluded, as they should be, the numbers would be twenty-eight or thirty-three out of forty-five, or from sixty-four to seventy-five per cent.

This agrees very closely with Sir Dyce Duckworth's recent estimate of seventy-eight per cent.[1] It is interesting to note in this connection that, according to the observations of Dr. Lewis, of Philadelphia,[2] there is a remarkable correspondence between the annual curve of chorea and that of rheumatism. Again, Dr. Newsholme[3] shows that acute rheumatism is a disease of large towns; chorea is also a disease of large towns.

The chain of evidence is a remarkable one, and the trend of medical opinion is increasingly in favor of regarding chorea as a phase of rheumatic activity. The proof that chorea is invariably an expression of acute rheumatism is not absolute, but the conclusion that it is so in the vast majority of cases seems fairly established.

APPENDICITIS.

During the last few years the occurrence of appendicitis, usually of mild form, in persons of rheumatic history and predisposition has been observed in a number of instances, and in cases recorded by Sir James Grant[4] and Dr. Yeo[5] the attack was accompanied by well-marked affection of the joints. In another instance, related by Dr. Armstrong, the family history of both rheumatism and appendicitis was very remarkable. Many of the cases recorded by others are not so definite,—in some the evidence of appendicitis and in others that of rheumatism (in some of both) being by no means of unmistakable character.

The value of salicylates in the treatment of this form of appendicitis is insisted upon and adduced in support of the rheumatic nature of the affection.

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[1] Rheumatic Nature of Chorea, Lancet, 1894, vol. i. p. 891.

[2] Seasonal Relations of Rheumatism and Chorea, Medical News, Philadelphia, 1886.

[3] Milroy Lecture, Lancet, 1895.

[4] New York Medical Record, November 11, 1893.

[5] Brit. Med. Jour., June 16, 1894.

Dr. Sutherland[1] gives a series of examples, some of which are of doubtful significance, and remarks upon the number of cases of appendicitis which have been recorded in which no adequate cause for its origin could be found. The concretion discovered in some instances he regards as identical with the calculi found in the crypts of the tonsil in chronic tonsillitis, and lays stress on the view taken by some authorities that the appendix is a kind of abdominal tonsil.

The writer has not met with any cases in which appendicitis has been especially associated with the rheumatic state or history, and the elucidation of the question of the pathological relation between the two must depend upon more extended observation and experience.

Diagnosis.—Acute osteomyelitis is an affection which had not attracted much attention when the original article was written. It, however, requires some notice, since in its early stage it presents certain points of resemblance to acute rheumatism. There are pain and tenderness of limb, accompanied by fever, and the disease occurs in children more frequently, perhaps, than in adults. It is distinguished by the seat of the pain and tenderness being not in the joint but in the shaft of the bone—usually one of the long bones, near its head, close to the epiphysis—and by the extreme intensity of the pain and tenderness, by the swelling of the periosteum, and by the great severity of the constitutional disturbance. The pulse is rapid, and the temperature high: out of all proportion to the local lesion.

Treatment.—There is little to be added in the way of the treatment of acute rheumatism and its complications. Numerous drugs have been advocated by various practitioners, but salicylate of sodium, salicin, quinine, and alkalies, in their proper turn, still hold the field. No satisfactory substitutes have been found.

When the heart is greatly enfeebled, strychnine, strophanthus, and caffeine may be added to the list of useful cardiac tonics, of which digitalis is the chief. When the heart is embarrassed by pericardial effusion or adhesions, or if there be extensive aortic incompetency, digitalis is of doubtful safety, and should be replaced by one of the other remedies mentioned above. In cases other than these, where there is great muscular feebleness or dilatation, digitalis is invaluable, and strychnine may often be combined with it with advantage. A really valuable addition to our means of controlling pericarditis has been found in the ice-bag, the use of which has been ably advocated by Dr. Lees,[2] and it is especially useful, according to the writer's experience, in the subacute recurrent pericarditis of children. These young patients usually bear it well, and even like it, and its power in relieving præcordial pain and tenderness and cardiac distress is very marked. It appears also to control the active inflammatory process, as shown by the quick subsidence of friction and cardiac excitement. The ice-bag should not be too large and heavy. It should be applied almost continuously to the cardiac region, being removed from time to time if the effect is felt to be uncomfortably chilling or if the temperature falls below normal. Hot bottles should be applied to the feet and body, lest chilling should occur too readily, the effect of the cold application being carefully watched throughout.

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[1] Lancet, vol. ii., 1895, pp. 457–459, and Edinburgh Hospital Reports, 1895.

[2] Clinical Journal, August, 1895.

The value of opium in the later stages of rheumatic heart-disease may be again enforced. There is no other drug which has the same power to soothe the urgent distress and dyspnœa of dilated heart, or the vomiting and pain of advanced pericardial disease, or the harassing cough which results from congestion of the base of the lungs, often preventing sleep, towards the close of valvular affections.

To be effective it must be given freely,—from one-half a minim or one minim to five or even ten minims of nepenthe or its equivalent every four hours,—the dose being graduated according to the age of the child, the amount of distress, and the effect produced.

Opium oftentimes not only relieves suffering and promotes euthanasia, but occasionally, by the ease and sleep it brings, gives the patient, for a brief period, a further lease of life.

RHEUMATOID ARTHRITIS.

Although this affection is not common in children, being more especially associated with the impaired nutrition and enfeebled health of later life, in persons of gouty or rheumatic families, cases are met with now and again even in very young children. Attention has recently been drawn to chronic joint-affections of this kind in children by Dr. Still, medical registrar and pathologist to the Hospital for Sick Children, in Great Ormond Street,[1] in an interesting paper read before the Royal Medical and Chirurgical Society of London in November, 1896. Dr. Still's treatise is based upon a study of twenty-two cases, and is designed to show that, although a disease identical with the rheumatic arthritis of adults does occur in children, certain cases of joint-disease, usually regarded as rheumatoid arthritis, are in reality not of that nature, but perhaps form a class apart.

The condition is described as a chronic progressive enlargement of joints associated with general enlargement of lymphatic glands and of the spleen. The disease almost always commences before the second dentition, and girls are stated to be more affected than boys. The disease differs clinically in the absence of bony change in the joints and in the presence of enlargement of spleen and glands. The most striking difference in its morbid anatomy is the absence of fibrillation of cartilage even in advanced cases, the absence of bony lipping or grating, or any osteophytic change, and the fusiform enlargement of the joints. The incidence of the joint-affection was adduced as a further point of difference, the knees and wrists being first attacked, instead of the small joints and fingers, as in rheumatoid arthritis. The occurrence of adherent pericardium in three cases, with no changes, or at most a doubtful thickening, of the mitral valve, is put forward as another feature of distinction.

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[1] Med.-Chir. Trans., 1896, communicated by Dr. A. E. Garrod.

It is pointed out that such variations cannot be merely due to difference in age, for a disease exactly corresponding with the rheumatoid arthritis of adults also occurs in quite young children. The condition is further differentiated from the rare form of chronic rheumatism described by Jaccoud as "polyarthrite déformante." In that affection there is evidence of genuine rheumatism, such as nodules, and organic valvular disease, while on the other hand there is absence of gland- and spleen-enlargement. Jaccoud's disease, again, is distinguished from the true rheumatoid arthritis of children by the absence of bony grating and thickening, such thickening as exists being *peri*-articular.

It is suggested, therefore, that under the term rheumatoid arthritis three conditions have, in the case of children, been classed together which are in reality separate diseases, viz.:

1. The form first described, in which there is no bone or cartilage change, but enlargement of glands and spleen.
2. True rheumatoid arthritis, as in adults.
3. A form probably identical with that described by Jaccoud as polyarthrite déformante,—the rarest of all.

In connection with these views it may be remarked that in the typical case adduced in the original article by the writer there was marked enlargement of the lymphatic glands. The same feature has been observed in some instances in adults by Bannatyne, Wohlmann, and Blaxall,[1] and by two French observers, Chauffard and Ramond,[2] who have described occasional enlargement of the lymphatic glands in adults in an acute form of "rheumatoid arthritis," which they distinguish as "infective arthritis."

Treatment.—There is little to be said in this respect, beyond the means set forth in the original article. Dr. Bannatyne[3] regards the creosotes as exerting a more or less specific action on the disease, and especially favors guaiacol carbonate given in doses of from five to ten grains, in powder, pill, or cachet, three times a day. He applies guaiacol and olive oil in equal parts to the joints externally. He claims for this plan increase of appetite, lessening of pain, reduction of temperature, improvement of joint-condition, relief of insomnia, and general improvement in aspect and condition.

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[1] Joint paper in Lancet, April 25, 1896.

[2] Revue de Médecine, May, 1896.

[3] Rheumatoid Arthritis, p. 113, 1896.

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MALARIA.

BY F. F. FORCHHEIMER, M.D., AND GEORGE DOCK, M.D.

Ætiology.—The hæmatozoa of malaria were first accurately described by A. Laveran, in Algiers, in 1880. Since then they have been investigated by numerous observers in all parts of the world. The organisms are usually classified by systematic zoölogists under the sporozoa, in the order hæmosporidia (Danilewsky), although it is not yet settled that they are sporozoa. Those found in man resemble in many respects some found in birds; less so those in frogs and reptiles. The name plasmodium malariæ, given by Marchiafava and Celli, though not well chosen, has become so familiar in medical literature that it can hardly be replaced. The parasites occur in a number of different forms, due partly to the existence of species or varieties, partly to the stages of development in which they are found. At a very early period they appear as round protoplasmic bodies, about one micromillimetre in diameter, with finely granular or hyaline structure ("hyaline bodies"). They stain with nuclear dyes, staining more deeply in small areas which are no doubt nuclear. They may be in the blood-plasma, where they move about, probably by the aid of cilia or flagella, which, however, cannot be seen; but their usual habitat is in the red blood-corpuscles, of which they are true parasites. Here they grow larger, and soon begin to show grains of yellowish to black pigment. During this period the bodies are in constant amœboid motion, throwing out from one to several pseudopods, retracting them, and changing position in the corpuscle. They are now known as "amœboid bodies." At times the amœboid motion ceases, and the parasites assume the "ring" or "seal-ring" form. Finally the bodies reach the maximum size, which varies in the different varieties from one-half or two-thirds the diameter of a red blood-corpuscle to a size larger than an average red cell, the host in the latter case being flattened out and, as it were, expanded. They now contain considerable pigment, either in grains or rods or in irregular masses, and either scattered through the body of the parasite or aggregated in the centre or sometimes in radial lines. Now fine radial lines appear in the protoplasm. These become more and more distinct, finally forming in typical specimens an evenly segmented body, conveniently described as the "segmenting form," the "rosette," "sunflower," or "marguerite." The segments become round or ovoidal. At this time the red blood-corpuscle either is entirely destroyed or but a thin shell of hæmoglobin is left. This becomes broken. The segments, now spoken of as "spores," separate from one another and from the pigment, and are lost in the surrounding blood. The pigment is taken up by fixed or circulating phagocytes, the spores are partly destroyed in the blood, but others infect red cells, and so continue their cycle of existence. The forms as described are those known to be necessary to the continuance of the species in the body and to attacks of ague. Other forms are observed, some of which were at one time supposed to be the most important of all. These are the flagellate forms and the crescents.

"Flagellate bodies" appear under various circumstances. An endoglobular parasite may leave its host at any stage of its development and become a "free body." These are pale, granular, or hyaline, sometimes containing darker, nuclear-looking parts, sometimes vacuoles, and also pigment grains. The latter are often seen in violent motion, resembling that of ebullition, due to the motion of the protoplasm of the parasites. These bodies throw out long protoplasmic processes, sometimes knobbed at the ends, which keep up active motion. The flagella appear to be hollow, and at times pigment grains may be seen moving in them, forming local dilatations as they go. The flagella are from one to four or six times as long as the diameter of a red corpuscle. One to three or four may be seen on a single body. They sometimes get loose and move off in the blood-plasma. Flagella are usually seen only after the parasites have been removed from the body some time, but Dock has described a case in which they were seen within less than a minute after removal, a fact early observed by Laveran.

Other and even more remarkable bodies are the so-called crescents. These have a narrow or broad, sharp-pointed or blunt crescent shape. They are made up of fine granular protoplasm, and contain, usually in the middle, sometimes towards one end, pigment granules or rods in a roundish or irregular mass. The bodies are usually slightly wider across the horns than red blood-corpuscles. Sometimes a thin line or band can be seen extending across the concave part of the crescent. This is supposed to represent the remains of the red blood-corpuscle in which the crescent develops. The crescents, when observed for some time in the ordinary way, become oval, with double contours, then roundish, and ultimately throw out flagella, resembling in appearance the flagellate bodies just described.

Looking on the forms from the spore to the mature segment as the essential representatives of the parasites, the *rôle* of the crescents and flagellates has been the subject of much speculation. The flagellates are believed by most observers to be degenerate forms; by others, bodies capable of living outside the human body. In either case the possibility of a further development is admitted, but not known. The nature and *rôle* of the crescents are quite as obscure. That these develop from small hyaline forms in the red blood-corpuscles is generally admitted, but their function is not known, nor would it be profitable in this place to discuss the various theories at length.[1]

We owe to the careful observation and sharp insight of Golgi the knowledge that there are different species or varieties of parasites, and that these have a distinct relationship with various clinical varieties of malarial disease. Golgi described, especially, forms peculiar to tertian and to quartan fevers, and showed also the relationship of the different stages of their growth with the stages of the disease. Later, Marchiafava and Celli described an important form which causes the common irregular and remittent fevers occurring especially in summer and autumn, the æstivo-autumnal or small form. The chief types have been recognized by various observers in widely different parts of the earth, their occurrence in America having been described by one of us.[2] Some of the Italian investigators also describe a malignant tertian form and a non-pigmented form, but these are not yet sufficiently confirmed. It is possible there are still other varieties.

Regarding the differences of the three chief forms, these consist especially in the length of time necessary for development, the size and appearance of the segmenting body, the number of spores, and the distribution of the pigment. As to the first, the tertian parasite becomes mature in about forty-eight hours, that of quartan fever in about seventy-two hours. The parasite of remittent has a cycle of more variable length than the others, being in some cases less than twenty-four hours, in others more, even thirty-six hours or longer. The tertian parasite reaches the largest size, the mature form being as large as, or larger than, a red blood-corpuscle. In the latter case it has been supposed by some that the parasite is degenerated. The quartan parasite attains about the size of a normal red corpuscle, while that of the æstivo-autumnal fevers is considerably smaller, usually about two-thirds the size of a normal red corpuscle. In the case of the quartan, and even more in that of the remittent parasite, the affected red corpuscles are shrunken and dark,—"brassy," as the Italians say. The tertian parasite forms from fifteen to twenty spores, that of the quartan from eight to ten, the individuals being larger than those of the tertian. The parasite of remittent fever forms from six to twenty or more spores, fifteen to eighteen being very common, and these are much smaller than those of tertian and quartan. The sporulating stage of the remittent form is rarely seen in peripheral blood, but can be found in that of the spleen and some other internal organs. The segmenting forms of the quartan and remittent are fairly symmetrical, but that of tertian shows variations. Often the "rosette form" is not present, the spores being scattered irregularly. In our experience this mode of segmentation is more frequent in the United States than that resulting in the symmetrical form. As regards the pigment, this is rapidly formed in the growing tertian parasite, the motion of which is livelier than that of the others. The grains may remain scattered until the latest stages. In the quartan and remittent forms the pigment tends to be gathered in a roundish clump, and perhaps for that reason seems darker than that of the tertian variety. Golgi supposed the pigment in the quartan form to lie in a protoplasmic shell.

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[1] Investigations by Dr. W. G. MacCollum, of Johns Hopkins University, not yet published in full, indicate a reproductive function of the flagellate bodies.

[2] Medical News, May 30, June 6, 1891; International Medical Magazine, vol. i., 1892.

The several varieties of parasites are associated with the chief types of malarial fever. By combination of two or more generations of one form, or of different forms, various other types are produced. So the ordinary quotidians of temperate climates are caused by the growth of two generations of tertian parasites. But very complex combinations may result, and if the blood be examined at a favorable time may be recognized by an experienced observer. A not infrequent combination in the United States is that of one or more tertian generations and the remittent, in the summer or fall, and in such cases the patients are likely to have pure tertian or quotidian (double tertian) fevers the following spring. Besides these differences, the tertian or quartan parasites may occur in fevers which are remittent, either as the result of some inflammatory complication, or perhaps as the result of a peculiarity of the patient. On the other hand, the parasite of remittent fever may occur in a case having normal temperature between the paroxysms. The latter variety is rarer than the former.

The free pigmented parasites and their flagellate bodies are very common in tertian cases, less so in quartan and remittent.

The crescents belong to the æstivo-autumnal fevers, but are sometimes found with the tertian in chronic and recurring cases when the other forms of the remittent parasite cannot be seen. It may be that in these cases the characteristic forms of the latter do not appear in the peripheral blood. The earlier stages might easily be mistaken for those of the tertian or quartan.

It is not known how or where the parasites exist outside of the body, the supposition being that in some form they grow in plants, or in the earth or water. On account of the fact just stated, we cannot demonstrate how the germs gain entrance to the human body. The wide-spread belief that the parasites enter by the respiratory tract must certainly be true of some cases. An example of the best sort of proof available has recently been given by Richard Newton (*International Medical Magazine*, October, 1895) from Fort Sill, Indian Territory. In the barracks there all the men used water from a common source. It was found, however, that malarial fever occurred in those barracks most exposed to the wind blowing from the stream, the banks of which furnished the combination of frequent overflow and decaying vegetation.

That infection can take place through the alimentary canal, as from water, cannot be denied, and numerous examples have been published to prove this, not all of them, however, free from the possibility of error. Infection by the bites of insects may also be admitted as possible. Numerous experiments in human subjects show that the disease can be in-

[illegible] through insect-bites, this does not occur [illegible]

[illegible] the development of the malarial germs out-[illegible] discussed in a former volume. It is of great [illegible] that we do not accurately know all these factors. [illegible] when the physical conditions seem unfavorable, [illegible] the conditions seem just the reverse. The [illegible] absence of the germ is to observe the result [illegible] beings, especially those of the white race, in a [illegible]

[illegible] Pathological Anatomy.—The relation of the various [illegible] the parasite to the clinical stages of the disease is as [illegible] liberated during the paroxysm. They then develop [illegible] and become mature just before the next paroxysm. [illegible] the chill often occurs at the time of segmentation, but [illegible] are not synchronous, the chill sometimes coming as late [illegible] Even in cases of pure type one can often see occasional para-[illegible] later stages than the majority. In combined infections [illegible] generations may be distinguished, so that one can recognize [illegible] of a double tertian or quartan, or a combination of tertian and [illegible] of these and the smaller form. With practice a very close [illegible] may be made also to the hours when the next paroxysms [illegible]

The process of development of each parasite involves the destruction of a red blood-corpuscle, since even in the case of the smaller parasites the infected red cells are so much altered that they are speedily removed from the circulation. Thus is produced the anæmia of acute malaria, the extent of which depends, other things being equal, on the number of parasites developing. This varies enormously. In some cases not more than one parasite may be found among many thousands of red blood-corpuscles. In others, as in a case reported by one of the writers, one-fifth of the corpuscles in the finger-blood were infected. This explains how various observers have noted a loss of one million red corpuscles per cubic millimetre after a single paroxysm, with a diminution of fifteen per cent. of hæmoglobin. It is sometimes found that the hæmoglobin continues to sink even after regeneration of red corpuscles has begun, which can be explained by the loss of partly destroyed corpuscles. In regeneration, as usual after anæmia from direct loss of blood, the corpuscles increase more rapidly than the hæmoglobin.

The digestion of the hæmoglobin of the corpuscles leads to the formation of the pigment granules already described. This derivation of hæmoglobin, like the latter itself, does not give the microchemic reactions for iron, but Carbone claims to have obtained iron from it. The pigment, in the parasites free in the blood or in phagocytes, causes the so-called melanosis, long recognized as an evidence of malarial disease.

The most careful observations on the leucocytes in malaria are those of Billings,[1] which agree with earlier ones of Kelsch and others. Billings describes a decrease in the number of leucocytes shortly after the chill, the minimum occurring at the end of the paroxysm. The number of leucocytes then increases, though the difference is not great. The polynuclear cells are reduced relatively and absolutely, the mononuclear cells, including the lymphocytes, are increased. In fatal cases an agonal leucocytosis has been observed. In chronic cases there is often a leucocytosis of from thirty thousand to forty thousand cells to the cubic millimetre.

Besides the alteration of the blood-cells, there are in acute malaria alterations in the distribution of the blood-mass. In the stage of congestion there is an excess of blood in the internal organs, especially in the vessels of the abdomen. In fatal cases the brain, lungs, spleen, liver, bone-marrow, or the gastro-intestinal tract may be affected in such a way that not only is there an excess of blood but also an unusual proportion of parasites in those parts. There may be also thrombosis of the smaller arteries. These anomalies often coincide with marked symptoms on the part of the organs affected.

In mild cases of malarial fever there may be but little alteration in the tissues other than the blood. The symptoms of the disease, often more striking in mild than in severe cases, are supposed to be due directly or indirectly to toxic substances caused by the action of the parasites on the hæmoglobin. The substances are not yet known.

In fatal cases there are necroses and degenerations of the liver, kidney, and other organs, usually in microscopic foci. In the liver this condition has been seen to be associated with alterations suggesting the early stage of portal cirrhosis. The relation of malaria, however, to the latter process is not yet entirely clear. The pigmentation of the various organs, especially the brain, liver, spleen, and sometimes the intestinal mucosa, was known long before the origin of the pigmentation was understood.

The chief alterations of organs other than those mentioned may be briefly described. The spleen is almost invariably enlarged, often enormously so. This is due partly to hyperæmia, partly to large numbers of blood-corpuscles and parasites, partly to hyperplasia, and finally to necrosis and œdema.

The acute enlargement of the liver is caused in a similar way.

Chronic malarial infection causes alterations of the blood of various kinds. Bignami and Dionisi describe four principal forms. In some cases the conditions in the blood are like those in secondary anæmia, but with diminished leucocytes. In others the blood resembles that of pernicious anæmia. These are fatal. Another form is progressive on account of lack of regeneration on the part of the bone-marrow. The fourth is associated with the malarial cachexia, the symptoms of which predominate.

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[1] Johns Hopkins Hospital Bulletin, No. 42, p. 89.

oculated [illegible] becomes hard, anæmic, and deeply pig[illegible] natural [illegible]

Th[illegible]—For the recognition of the malarial organ[illegible] of experience is necessary, and on account of [illegible] it seems important that all physicians in Mal[illegible] have such knowledge themselves or obtain an[illegible] have it. With a knowledge of histology, espe[illegible] high-power lenses, as in bacteriology, and instruction [illegible] material, it does not take long to acquire a [illegible] germs. On the other hand, an enthusiastic and [illegible] man, away from competent assistance, may [illegible] without being certain of what he sees, making errors [illegible]

[illegible] diagnosis of the parasites certain precautions are neces[illegible] trustworthy results. The best time to examine in an [illegible] few hours before the paroxysm,—say from one to three [illegible] before the beginning. At that time the large forms with [illegible] pigment are almost always present. The pigment and the [illegible] motion, and, later, the segmenting forms, are comparatively easy [illegible]. The blood should be taken in a small drop, so that when [illegible] is let down the blood will spread out so thin that every corpuscle [illegible]. The preparation should be made as rapidly as possible, to prevent the corpuscles from becoming crenated. Such a preparation is best ex[illegible] at once, and the best results are obtained if the examination is so made. If a preparation proves to be unsatisfactory, it can at once be replaced by another. If an immediate examination cannot be made, the edge of the cover-glass may be smeared with vaseline or olive-oil, when it will keep for as much as twenty-four hours in tolerably fresh condition. If even this cannot be done, or if permanent preparations are wanted, blood is spread on cover-glasses, allowed to dry in the air, and then "fixed." For the fixing various methods are used. For ordinary purposes good results may be obtained by passing the cover, blood up, three times across a flame. It is usually more convenient to use absolute alcohol and ether. These are placed in a wide-mouthed bottle, in the proportion of one to three, the covers put in the bottle and allowed to remain for from fifteen minutes to a day, the latitude in this respect being one of the greatest advantages of this method for physicians.

The fresh (unstained) preparations should be examined in a good light, with lenses of good definition and flat field, and a magnifying power of from six hundred to a thousand. The light should be regulated by a narrow diaphragm. The field should be systematically examined, and the number, sizes, and other peculiarities of the parasites noted. The vacuoles sometimes present in the blood may give the beginner some trouble. Aside from the differences of color, refraction, and shape of these as compared with the parasites, readily learned by a little practice, the formation of pseudopods and the presence of pigment serve as useful guides to the differentiation. Beginners often mistake highly refracting spines on crenated corpuscles for pigment. This is easily avoided by opening wide the diaphragm. True pigment will then be more distinct; the other will disappear.

For staining, various methods may be used. Perhaps the best is a double stain of eosin and methylene blue. The fixed cover-glasses are stained about a minute in a half per cent. solution of eosin in seventy per cent. alcohol. They are washed, dried with bibulous paper, then stained in a watery methylene blue solution, Loeffler's being a very convenient one. These may be examined in water, but still better in balsam. The parasites are stained a clear blue, deeper in some parts. The nuclei of the leucocytes are darker blue. The red corpuscles are bright red, and allow the parasites to be readily distinguished in them.

Besides recognizing the existence of the infection, one should make an effort at a diagnosis of the type. For this purpose it is almost necessary to examine just before a paroxysm in order to get the mature forms. A diagnosis of the type and of the probable time of the paroxysm should be made only from the observation of a number of parasites. It often happens that a few parasites out of the ordinary cycle are present, and if they are used as guides to the type or the time of paroxysms disappointment will follow. The diagnostic value of the recognition of the various species is easily understood by remembering their relation to the disease. The value of the crescents in diagnosis has been exaggerated by some. Although they are usually present in chronic cases, yet they may occur early in an attack which subsides under treatment, though not as quickly as one of the milder and less obstinate types.—G. D.

Since the chapter was written by one of us which appeared in a former volume of this work, our knowledge has been vastly added to. At that time the clinical aspects of the value of the hæmatozoa as a diagnostic criterion were still dubious; while now their presence in the blood of a patient signifies malaria, and, *per contra*, their absence means no malaria. As the result of this view, notwithstanding the statement by Bacelli and by Golgi "that death from malarial infection without the well-known forms of the hæmatozoöns being found in the blood does occur," clinical manifestations have undoubtedly been considered non-malarial that are due to this poison. A safe, conservative estimate of our present position would force us to the admission that our etiological knowledge is not sufficiently complete to warrant its application as a final conclusion to all cases. In order to determine the malarial nature of any given form of manifestation, the existence of the hæmatozoön must be shown. For the individual case, however, this is not necessary, and sometimes impossible. "One can die of a malarial affection positively diagnosticated without the presence of the well-known hæmatozoön in the blood." (Bacelli.)

According to the Italian authors, the mechanism of the production of symptoms can be reduced, first, to a morphological blood dyscrasia; second, to a chemical blood dyscrasia. The first causes progressive disease of the red blood-corpuscles, changes in hæmoglobin in the direction towards melanin, then hypoglobulia and anæmia, and finally cachexia. The second is produced by spores and their fission products, resulting in hæmatoxins which cause fever, and which are supposed finally to injure the nervous system especially. Admitting the classification of the hæmatozoön made by one of us, we can divide the manifestations of malaria into two large classes: first, intermittent fever; secondly, the remittent form. Under the head intermittent form we can take into consideration the quotidian, the tertian, the quartan, the irregular, the continuous, and the masked types (intermittens larvata). Under the remittent form we can make first two subdivisions as to the character of the fever, speaking of the benign and pernicious forms; secondly, both of these forms may take upon themselves either the intermittent, the subcontinuous or continuous, the irregular, or the masked types.

INTERMITTENT FORMS.

Above all, the most common form is that type produced by the ordinary tertian and quartan parasites, as described by Golgi. The quotidian type is produced either by the tertian or by the quartan parasite. The tertian type can be produced only by the tertian parasite itself. The quartan parasite may produce either the characteristic quartan attack, a quartan duplex, or a triple quartan. In the first instance we have a paroxysm every fourth day; in the second there will be two successive days with a paroxysm, and the third day free from an attack; and in the last the paroxysms will be quotidian. There may also be infection with several types of hæmatozoön; then the paroxysms will not be developed as regularly as described. *Continuous fever*, as we see it in the temperate zones, due to the parasites of Golgi, usually develops in one of two ways,—either as a resultant of a long-protracted attack of intermittent paroxysms, or, possibly, more commonly, when the patient changes from a wandering life to that of hospital existence, or when for some other reason his condition of general nutrition is changed materially. The child will come under observation with malarial cachexia; he then develops an attack of continuous fever which, after four to seven or eight days, gradually goes into an attack of intermittent with the characteristic hæmatozoön. The *irregular forms*, in all probability, are due to the intermittent hæmatozoön with long or short intervals, and in these we find the crescents of Laveran. In children the *masked forms* are of great importance. Children seem to suffer more from the manifestation of toxins than do adults, and if there be a toxin in malaria it would, as in all other diseases, attack especially the nervous system. We should find, furthermore, that the mucous membranes would be affected by these toxins as the result of eliminative processes. Strictly speaking, if we take the definition of these masked forms as that of attacks that are afebrile, they are, of necessity, very rare, for the simple reason that, especially in children,—and the younger the child the more this is true,—any deviation from perfect health will be marked by some change in temperature. As a rule, changes produced by toxins are manifested by increase in temperature, but not uncommonly the reverse occurs, so that instead of elevation we may have a depression in temperature. There is no doubt of the existence of a pernicious larvata. This is admitted by all the observers who have studied the hæmatozoa in countries in which this pernicious form of fever exists, the hæmatozoön having been found with great regularity and of the usual kind. There can, furthermore, be no doubt concerning the existence of the masked forms as the result of the other forms—the intermittent—of the hæmatozoa. One of us has found them in one type to be referred to presently. The frequency with which this form of malaria occurs in children must be explained by the peculiar reaction of their nervous systems. These paroxysms present themselves in one of two ways: either with or without high fever, always intermittent, and always rudimentary paroxysms. It must be admitted, on the other hand, that the frequency of these masked forms has heretofore been very much overestimated, and much of the discredit attached to the diagnosis of malaria is due to this fact. Yet for practical purposes it will be sufficient to accept clinical evidence in order to determine the justification in retaining certain forms as masked forms of malaria. Given regular paroxysmal recurrences of these attacks, an epidemic occurring in regions known to be malarially infected, the occurrence of other forms of malaria, enlarged spleens, the general condition of nutrition, and even without the detection of the hæmatozoa the true nature will be revealed. To this class, especially occurring in children, belong undoubtedly the diarrhœic form described by Bohn, Filatow, and Moncorvo; the erythema nodosum of Boïcesco and Moncorvo; the intermittent torticollis described by Henoch, Bohn, and Bierbaum in Germany and by Holt and one of us in this country; and, lastly, the neuralgias, especially of the fifth pair of nerves and of the intercostals. It may be added that in the experience of one of us the ordinary intermittent hæmatozoön of Golgi is just as positively found in the two latter types as in the ordinary form of intermittent fever.

FORMS DUE TO THE REMITTENT HÆMATOZOÖN.

It is supposed that all pernicious forms are due to this form of parasite. The ordinary parasite may produce extremely serious symptoms as the result of paroxysms, but rarely if ever causes death. Cachexia may produce a fatal result, as may its sequelæ, but an acute fatal termination is very rare.

The benign fevers may be intermittent, remittent, or continuous. With us the most common form is either the intermittent or the remittent. It is rare in temperate zones to see pernicious fevers. Indeed, the types of malaria differ not only from place to place in the same country, but from year to year, and according to some Italian observers from season to season, which by some investigations seems to be carried out for this country as well. With us truly pernicious fevers are extremely rare, and in an experience of over twenty years one of us has seen but three pernicious attacks of malaria in children. The intermittent forms may be quotidian, they may be regular, and they may vary as to intensity and time; indeed, typical attacks are rare. The attacks may coalesce, producing varying types, even to malignant, these being usually rapidly fatal. In the tertian form, according to Marchiafava and Bignami, the following states can be noticed: the febrile invasion and period of fever with oscillations in temperature, the pseudo-crisis, the precritical invasion, and finally the crisis. This attack may last from twenty-four to forty hours, and, as the apyrexia may last only a few hours, the headaches, weakness, and gastric and intestinal symptoms continuing, the patient thinks the fever has been continuous. But the regularity of the attack may be absent. This type may be complicated as follows: first, by modifications in the fever curve of paroxysms; second, by modifications in the sequence of paroxysms.

1. (*a*) The absence of a definite beginning of fever curve, so that it arises continuously and progressively. (*b*) Prolongation of paroxysms, usually combined with accentuation of the oscillations in the stationary period. (*c*) Absence of precritical elevation.

2. (*a*) Anticipating paroxysms which occur in mild as well as in severe cases. (*b*) Retarding paroxysms occurring in severe attacks as well as in mild ones. (*c*) Prolongation of paroxysms, so that apyrexia becomes imperfect. (*d*) Slight oscillations of temperature during apyrexia. (*e*) Double tertian.

Lastly, complicated or irregular curves may be produced by the following. (*a*) The existence of two varieties of parasites in the blood. (*b*) As a result of therapeutic measures. Quinine may produce a simpler form.

The remittent form, as we see it with us, or the continuous form, may be produced by either the intermittent hæmatozoön or the remittent hæmatozoön. Most commonly, however, it is produced by the remittent hæmatozoön. If the statement made by Marchiafava and Bignami is correct, the mechanism of the production of prolonged attacks is to be referred either to the presence of several generations of parasites or to the coalescence of individual paroxysms. This form has given rise to much confusion as to diagnosis. The differentiation of typhoid fever is extremely difficult in children; so much so is this the case that even to-day excellent authorities assert that a long-continued fever in children cannot be due to malarial infection. On the other hand, it is a curious phenomenon that we should revert to the opinion held twenty-five years ago, that remittent fever in children is always typhoid fever; that this is a very broad generalization which does not hold good almost goes without saying. Both typhoid fever and true malarial remittent fever occur in children of all ages. Widal's test[1] may help us in the diagnosis of the former affection, the detection of the hæmatozoön will always do so in the latter. The peculiar views held regarding the uselessness of administering quinine in remittent or continued fevers doubtless has much to do with the frequency of this type of fever in the autumn. It is but just to say that one of us has been thoroughly converted to the view that true malarial remittent fever occurs in children, both on account of finding the hæmatozoön in these cases, as well as on account of the efficacy of quinine when given in sufficiently large doses for a sufficiently long time. Relapses are common in this form, as well as in all other forms of malarial intoxication. Nothing need be added, as far as the clinical picture is concerned, to the description given by one of us heretofore.

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[1] See article, Typhoid Fever, page 167, of this volume.

PERNICIOUS FEVERS.

The forms as noticed in children are principally of two types,—the comatose forms and the convulsive and eclamptic forms. (Concetti.) The first is noticed especially in very young infants. After one or two paroxysms, intermittent in nature, there come on coma, very high temperature, 42° to 43° C. (107° to 109° F.), sometimes convulsions, and then death. In the convulsive and eclamptic seizures the history seems to be the same.

Treatment.—As far as prophylaxis is concerned, the previous experience of the value of small doses of quinine for a great length of time has been emphasized by continuous experience. For the sake of prophylaxis, quinine should be given in very minute doses,—0.065, gr. i,—morning and evening. Since the writing of the former article by one of us, very little that is new has been added to the methods of giving quinine to children by the mouth. The best methods still remain those recommended in the previous article. Aufrecht has again called attention to the advantages of quinine tannate in children's practice, but does not lay sufficient stress upon dosage nor upon the comparative uncertainty of its action. In the treatment of malaria it has been shown by Golgi that quinine acts especially upon the youthful forms of the hæmatozoön, that it prevents its development to a certain extent, but not absolutely, and that eventually it will attack all the various forms of the hæmatozoön. In the treatment of intermittent forms it has been shown by Golgi that in the tertian form the best time to give quinine is three, four, or five hours before the paroxysm, this also being a good time for the quartan form. The quinine is to be given in one large dose. It is not as efficacious at the end of the attack, because the parasites are not prevented from entering the red corpuscles. By giving the quinine in the beginning of apyrexia a certain effect can be produced, though not a positive one. In the quartan form the end of the second day of apyrexia is very favorable, because of the endocorpuscular condition of the hæmatozoön. One dose in the majority of cases, given at the right time, destroys the infection, but the most rational method is to continue with the quinine for a few days. Golgi quotes Kerner as saying that "six hours after the quinia is introduced the largest quantity of quinia is shown in the blood." Bacelli states that the amount of quinine must be as 1 to 5000 of the weight, in order that perfect effects may be produced. By means of this rule the dosage for children can be accurately determined; as it also applies to the amount to be used by intravenous injection, it will be seen that for administration by the mouth the dose must be increased considerably. This can be done with impunity, as children tolerate comparatively large doses. Laveran gives the following directions: "The type of fever ought, in my opinion, not to sensibly modify the method of treatment, after we have once been assured by an examination of the blood that the fever is indeed due to malarial infection. As an example, we might prescribe for a male adult, on the first, second, and third days, from 0.80–1.00, gr. xii–xv, of quinine hydrochlorate daily in the course of twenty-four hours. On the fourth, fifth, sixth, and seventh days, no quinine. On the eighth, ninth, and tenth days, 0.60–0.80, gr. ix–xii, of quinine hydrochlorate. On the eleventh to the fourteenth day, no quinine. On the fifteenth and sixteenth days, 0.60–0.80, gr. ix–xii, quinine hydrochlorate. From the seventeenth to the twentieth day, no quinine. The twenty-first and twenty-second days, 0.60–0.80, gr. ix–xii, quinine hydrochlorate. In the case of children four years of age and upwards, 0.30–0.40, gr. v–vi, of quinine in divided doses may be prescribed daily. Children from two to four years of age should receive from 0.20 to 0.30, gr. iii–v; children from one to two years of age, 0.12–0.20, gr. iss–iii. In the case of infants under twelve months of age, 0.05–0.10, gr. ¾–iss, quinine hydrochlorate may be given by rectal injection. (J. Simon.)" It will be seen that much stress is laid upon the prevention of relapses, a matter, according to the experience of one of us, of the highest importance, and referred to *in extenso* in the previous edition. Another method of giving quinine—especially recommended by Thayer for milder forms—is the administration of small doses three times daily: he furthermore recommends the giving of the large dose after the paroxysms. It really seems as if all methods lead to the desired result, the only thing to do being to give quinine, because, sooner or later, all the stages of the hæmatozoön will be affected by it. The methods of giving quinine by the rectum and endermically are the ones most dubious in results.

The treatment of the remittent and continuous forms can be summed up in the continuous and constant use of quinine. It will be seen, by referring to the opinions expressed by writers in all countries, that the effect of quinine is very much less positive in this form than in the intermittent forms. It is possible that this has been overestimated. It is probable that many of the conditions classed as remittent fever are not malarial. It is, furthermore, probable that sufficient patience has not been exhibited in the administration of quinine. The conclusions, therefore, as to the efficacy of this drug in remittent forms are more or less imperfect. The method of giving quinine in this form of the disease is to administer it during apyrexia in one large dose, or to divide this dose into two, giving one late in the evening and the other early in the morning, or to give the quinine in comparatively small doses throughout the day. Whichever method is employed, persistency is absolutely necessary, and the quinine should be continued for some time after the discontinuance of the fever, in order that relapses may be prevented.

For the pernicious types it is necessary to give the quinine hypodermically. For this purpose either the hydrochlorate of quinine, or the muriate of quinine and urea, or any other thoroughly soluble preparation can be used. The method of Bacelli consists in giving intravenous injections of quinine hydrochlorate according to the following formula:

℞ Quiniæ hydrochloratis, 1.00;
Sodii chloridi, 0.75;
Aquæ destillatæ, 10.00.

By those who have dealt very much with pernicious forms it is stated that the hypodermic method enjoys all the advantages of the intravenous use of quinine without its disadvantages. In addition to the use of quinine, the Italian and German physicians speak very highly of inhalations of oxygen and hypodermic injections of ether.

The only substance that has been introduced lately as a substitute for quinine is methyl blue, by Gutman and Ehrlich, who based their conclusions upon the treatment of two cases of intermittent fever, their theoretical basis being the fact that the substance is taken up by live plasmodia and by red blood-corpuscles. "The attacks of fever disappear upon using methyl blue within the first few days, and at the latest within eight days the plasmodia also disappear." Continuous experience has shown that these conclusions are hasty and unwarranted, and that in no sense of the word can methyl blue take the place of quinine. Many other substances have been recommended as substitutes for quinine, the most prominent being phenocoll hydrate, picric acid, helianthus annuus (the sunflower), iodine; but it seems that the old method of reasoning of *post hoc ergo propter hoc* has been too freely used and the self-limited nature of many attacks of intermittent fever too easily forgotten, so that Laveran must be considered as right when he says, "It is safe to say that we are acquainted with no drug that is really deserving the title [*i.e.*, a substitute for quinia], and which in a grave case of malarial fever can inspire the same degree of confidence as quinia."

MALARIAL CACHEXIA.

It has been shown by Golgi that arsenic has no effect upon the malarial plasmodia. Indeed, in the chronic forms, in which the crescents are present, medicinal effects are extremely rare, if they exist at all. Notwithstanding this fact, arsenic still holds the same position towards the chronic conditions that quinine does towards the acute ones. Iron preparations are absolutely necessary; otherwise the treatment must remain as it has been, —symptomatic. For the enlargement of the spleen hydropathic measures have been found of great value. The application of a stream of water to the spleen, in the form of the spout bath, as it is employed in several of our bathing places in this country and abroad, seems to be of great benefit. The use of cold douches over the spleen, massage, and electricity have also been highly recommended. Above all, good food, good air, and, if possible, removal from continued infection are essential.—F. F. F.

INFLUENZA (LA GRIPPE).

By CHARLES GODWIN JENNINGS, M.D.

Definition.—Influenza is an acute infectious fever, caused by the bacillus of Pfeiffer, and characterized by a peculiar nervous prostration and catarrh of the respiratory, gastro-enteric, and other mucous membranes.

History.—Until the great pandemic that began in the fall of 1889 most practitioners of the present generation knew of influenza only in the history of the epidemics of the past.

Vague mention is made by old writers of epidemics that in all probability were of this disease, so that it may be said that extensive epidemics of influenza have prevailed from the earliest times.

Accurate descriptions of the disease are found in the writings of the early part of the sixteenth century. Since that time frequent epidemics that have swept over a part or the whole of the civilized world have been described. Since 1655 repeated outbreaks have occurred in the United States, the last great epidemic in 1847–48. In recent years various visitations have occurred that have been classified as influenza by able clinicians, although from the mild character of the epidemics and their limited extent they have not received conspicuous mention in medical literature. Loomis believes that influenza has prevailed in New York for at least twenty-five years, and Da Costa described the characteristics of an epidemic that prevailed over a wide area in the United States in 1879. Only future bacteriological investigation can determine the identity of such mild visitations with the great and grave pandemics.

Originating in Eastern Asia, where the disease appears to be almost endemic, the course of the later epidemics has been almost uniformly from east to west. Appearing first in Bokhara, in May, 1889, the recent pandemic followed the well-beaten path. St. Petersburg was reached by the end of October, and the epidemic quickly attracted the attention of the civilized world by its gravity and extent. The whole of Europe was in its grasp by the end of December. Its appearance was announced in New York by the middle of December, and in two weeks it had assumed epidemic proportions. From the Atlantic seaboard the whole of North America was invaded during the first few weeks of 1890. With Europe as a centre, the civilized portions of the Southern hemisphere were successively invaded along the routes of commercial communication. With occasional local outbreaks and numerous sporadic cases, influenza slumbered during the summer and fall of 1890. In January, 1891, the second great epidemic began in New Orleans, and from that point began its journey around the world, this time travelling from west to east, reversing the usual direction of its course. The Atlantic seaboard was reached in April, and in the succeeding few months it had revisited the whole of Europe. A milder and less prolonged epidemic prevailed in 1892.

Etiology.—Pfeiffer in 1892 announced the discovery of the bacillus of influenza, and corroborative testimony by other eminent bacteriologists confirms his observations and experiments. Pfeiffer's bacillus is very small,—0.8 of a micromillimetre long and 0.4 of a micromillimetre thick. (Klein.) It occurs singly, in pairs, and in masses. Stained specimens show the protoplasm segregated into bulbous extremities with an unstained central shaft. It thus closely resembles a diplococcus. It has been found in large numbers in the blood and sputum, the bronchial secretion, peribronchial tissue, and pleural exudate of influenza patients. It is said that the number of bacilli found in the sputum has a distinct relation to the severity of the disease. It gives characteristic cultures to the fifteenth generation. Inoculated apes and rabbits develop typical influenza.

Susceptibility to influenza is more general than is the case with any other infection except variola. During an epidemic individuals of all ages and conditions are liable to be attacked. The aged and those who are physically and nervously depressed are especially susceptible. Children enjoy a certain immunity which varies in different epidemics. Young infants more uniformly are immune. In the recent epidemic, while young subjects were much less gravely affected than adults, a large percentage of them contracted the disease. The immunity conferred by one attack is short and uncertain. Relapses and second and even third attacks are frequent in the same epidemic, and many individuals contract the disease at each epidemic recurrence.

The abundant evidence accumulated during the recent prevalence of the disease has made it certain that the disease is propagated by personal contact and by fomites; and when the medical profession can overcome the influence of the old idea that the disease is transmitted through the air or in some other occult way, it will be realized that influenza is intensely contagious. The poison appears to be received by the inspired air. Like measles, it infects actively from the inception of the symptoms. The period of incubation is short,—from one to four or five days, and many cases develop within a few hours.

The foregoing facts in the etiology of influenza are sufficient, we believe, to explain what has been the cause of much dispute and uncertainty of expression,—namely, the extraordinary rapidity of the diffusion of the disease.

With our total ignorance of the influences that make an environment favoring an epidemic development of the organism of any contagious disease, it is useless to speculate on the atmospheric or telluric conditions that may precipitate an epidemic of influenza.

Pathological Anatomy.—There are no morbid changes characteristic of influenza. The bacilli are found in the blood, in the secretions of the respiratory mucous membrane, and in the lung tissue. Very few children with uncomplicated influenza die. If death takes place from the intensity of the infection there may be no gross changes indicating the cause of death. Most cases, however, present the lesions of catarrhal inflammation of the respiratory and digestive tracts. Complicated cases present the lesions of the complicating disease. Lobar pneumonia and bronchopneumonia are the most common. Any organ or tissue may be the seat of a secondary infective inflammation.

Symptomatology.—The symptomatology of influenza presents a great variability which is manifest not only in different epidemics, but also in individual cases of the same epidemic. Between the mild cases that differ but little from an ordinary catarrh of the upper respiratory tract and the severe cases that exhibit all the phenomena of a profound systemic infection are cases of all grades of severity and presenting the most varied complexus of symptoms due to local congestions and inflammations.

According to the prominence of the local symptoms, writers have variously classified the protean forms of the disease. No classification is entirely satisfactory.

As the disease presents itself in children, cases may be grouped for clinical description into,—

1. Influenza without prominent catarrhal symptoms.
2. Influenza with prominent catarrhal symptoms.

(1) *Influenza without Prominent Catarrhal Symptoms.*—A typical case of this group begins abruptly with chilliness, quickly followed by severe headache, pain in the eyeballs, and general muscular aching. The various pains are more distressing in this disease than almost any other acute infection, and are quite characteristic. Vomiting is frequently observed. The temperature quickly rises to 102° F. to 104° F.; the pulse is quickened, although often slower than the temperature would indicate. Prostration comes early, and in all except the mildest cases is pronounced. Mild naso-pharyngeal catarrh and a slight persistent cough are usually present. The fever is of an irregularly remittent type, is highest during the first days of the attack, and disappears by lysis. Often for a week or more after defervescence an afternoon rise of one or two degrees persists. A subnormal temperature is not infrequent during the prostration of convalescence. The attack continues from three or four days to a week, and as the fever subsides the child is left anæmic and prostrated. Convalescence is often protracted, and is frequently interrupted by mild febrile relapses, with symptoms of fleeting engorgements of the respiratory and gastro-enteric mucous membranes.

A severe form of influenza without catarrh is observed in children, characterized by the symptoms of profound systemic intoxication. As Holt says, these cases closely resemble pneumonia in their onset and course, with the pulmonary symptoms and physical signs absent. Vomiting and often convulsions mark the onset. The temperature is high, from 103° F. to 106° F. There are severe headache, delirium and stupor, often photophobia and opisthotonos. The symptoms may closely simulate meningitis. The course of these grave symptoms is short. In two or three days they subside, and the case pursues the usual slow convalescence.

Severe influenza in young infants often assumes peculiar features. The clinical history of these cases is well described by Holt. "Even though the temperature is but little above normal, the prostration is extreme. The eyes are sunken, the face is pale, there is marked apathy, and food is often refused altogether. In other cases there are cyanosis and very rapid respiration, indicating acute congestion of the lungs, although no abnormal signs are present, except very feeble breathing sounds. Nearly always there is a disturbance of digestion, with vomiting and undigested stools. Death may occur in two or three days; sometimes it is postponed for a week, the chief symptoms being gradually increasing prostration, and finally collapse, without the development of any marked local evidence of disease. The system seems in these cases to be overpowered by the intensity of the poison. In other cases pneumonia develops, and from this death occurs."

(2) *Influenza with Prominent Catarrhal Symptoms.*—In a large percentage of cases of influenza local symptoms develop dependent upon local congestions and inflammations that are the result of the action of the influenza poison. These local pathological processes cannot be looked upon as complications. The great complexity that is thus given to the symptoms renders it advisable to group cases into clinical varieties.

In the *catarrhal form* either the upper or the lower respiratory tract, or both, may be involved. In some cases the peculiar specific inflammation invades the lung tissue.

When the upper respiratory tract is chiefly involved there are the usual symptoms of a severe, acute rhino-pharyngitis and laryngo-tracheitis. Serous and sero-purulent discharge from the nose is abundant, and often excoriates the nostrils. The pharynx is red, the tonsils are swollen and often the seat of follicular exudate. Rarely a continuous pseudo-membrane forms. The inflammation may extend to the Eustachian tubes and middle ear or to the mouth. Swelling of the cervical glands is usual. The laryngeal and tracheal involvement is shown by the hoarse voice and the harsh, dry, and obstinate cough.

Involvement of the lower respiratory tract gives rise to short, frequent coughing, rapid and oppressed breathing, chest-pain, and soreness. Fleeting engorgements of areas of lung tissue without the development of pneumonia are not infrequently noted.

In a smaller percentage of cases vomiting, diarrhœa, tenesmus, and abdominal pain are the prominent local symptoms. This is the *gastro-enteric form.* The symptoms of a gastro-enteric catarrh go along with the usual general symptoms of influenza, and then, as a rule, fade away. Influenza being a cold-weather disease, the gastro-enteric inflammation is rarely dangerous, although it may be so in very young infants. In a few cases an erythema or an urticaria has been noted.

Complications.—The influenza bacillus appears to be able to excite local inflammations of sufficient intensity to be called complications. More bacteriological work, however, will be necessary to determine the relative importance of Pfeiffer's bacillus and of other pathogenic organisms in the etiology of the complications of influenza.

Influenza, like measles and scarlatina, renders the tissues exquisitely susceptible to pneumococcus and streptococcus invasion, and the few experiments and observations made up to the present time favor the view that the complications of influenza are chiefly secondary infections.

Pneumonia is the most frequent and serious complication. Bronchopneumonia is the usual form, although lobar pneumonia, running a typical course, is not infrequently seen. Abortive and irregular types, probably mixed infections, are common. They are characterized by a short duration, and a temperature, prostration, and cerebral symptoms out of proportion in their severity to the indications of local disease.

Implication of the pleura is frequent, and not infrequently terminates in empyema. Pulmonary inflammations usually appear during the declining stage of the disease, although the infection often expends its force on the lungs from the beginning.

Cervical adenitis from throat infection is very frequent when severe catarrh of the upper respiratory tract accompanies the disease. Suppuration often follows. Catarrhal and purulent otitis media is exceedingly frequent in these cases. Gastro-enteritis of severe type is common in some epidemics. Nephritis and pyelitis have been observed. The central and peripheral nerve disturbances so common in adults are very rare in children.

A severe and persistent anæmia almost always follows influenza of a grave type, and for weeks a child is very susceptible to relapses of the primary disease and to catarrhal inflammations of the various mucous membranes. Only repeated unpleasant experiences will teach the physician the care these patients require during their long convalescence.

Chronic nasal catarrh, adenoids in the naso-pharynx, and hypertrophied tonsils are frequent sequelæ. Susceptibility to tuberculosis is greatly increased.

Diagnosis.—Mild influenza cannot be distinguished from a simple acute catarrh. The prevalence of an epidemic makes the diagnosis of influenza probable.

Severe cases may simulate any one of several of the acute infections. During an epidemic differentiation may be easy, but sporadic cases are often very puzzling, and only the progress of the case will clear up the diagnosis. The disease is frequently a close counterfeit of pneumonia. Subsidence of the respiratory symptoms in two or three days and the absence of physical signs mark an influenza. The cerebral symptoms may closely resemble those of meningitis. Malaria and typhoid fever are excluded by negative bacteriological tests and the absence of the characteristic symptoms of these diseases. A suspicion of scarlatina or measles may confuse the diagnosis for a day or two. Severe constitutional symptoms without the evidence of local disease are characteristic of influenza.

Prognosis.—The prognosis will depend upon the age of the child and the nature and severity of the complications. In older children mild uncomplicated influenza is an insignificant disease, and even the severe cases with alarming initial symptoms are very rarely fatal. In young infants severe influenza is a more serious matter. The uncomplicated form is sometimes fatal, and grave complications, particularly pneumonia, are often a source of danger. The remote effects of an influenza may be serious on account of the anæmia and the susceptibility to other diseases that long persist.

Treatment.—Rest in bed until convalescence is well under way is essential in the treatment of influenza, and the most careful hygienic supervision should be maintained over a child until the danger of relapse and secondary infection has passed. The course of the primary infection cannot be shortened by medication. The indications are to relieve the distressing symptoms and sustain the strength of the patient. A mild cathartic of calomel and soda should be given at the onset of the disease. Following this, a diaphoretic of spirit of nitrous ether and liquid Dover's powder may be given. The headache, nerve symptoms, and muscular pains are best relieved by phenacetin in doses of one grain to a child two years old, repeated every hour for two or three doses. Given in this way for the first three or four days, and administered at the beginning of the afternoon exacerbation of fever and discomfort, this drug takes much from the distress of the attack. Codeine aids the sedative effect of phenacetin and prevents a possible depression. High fever is best controlled by the tepid bath, given in the manner usual in acute diseases. Quinine in moderately full doses has proved in the recent epidemic to be of much value. It may be given during the whole course of the disease, unless there be marked gastroenteric disturbance.

The distressing cough of the first days of the disease is relieved by codeine in full doses. Later cod-liver oil and creosote act better than the ordinary expectorants.

Local treatment of the nose and throat is essential in all cases attended with severe catarrh of the upper respiratory tract. Thorough irrigation of the nose and throat two or three times a day with normal saline solution, or a mild antiseptic like Seiler's solution, diluted three or four times, is a most efficient prophylactic against secondary streptococcus and pneumococcus infection. Throat treatment should be begun on the second or third day and continued well into convalescence.

Prostration is best met by alcohol or strychnine. Convalescence demands a careful tonic treatment, and is often hastened by a few weeks' sojourn in a mild climate.

THE RELATION OF SCROFULOSIS TO TUBERCULOSIS.

BY HENRY ASHBY, M.D., F.R.C.P.

THE word scrofulosis has suffered greatly at the hands of the present generation of physicians, and is now much less frequently heard than formerly; indeed, it has been entirely abandoned by many writers of repute.[1]

One of the reasons for this is to be found in the fact that it has come to be looked upon by the public as a term of reproach, inasmuch as it is a disease to which "workhouse" and "pauper" children are especially addicted, and is associated in the minds of parents with unsightly swellings and scars in the neck. To say that a child is scrofulous is to suggest something decidedly unpleasant or distasteful to the parents. The profession, I often find, are cautious in the use of the term in speaking to parents. A short time ago a lady took her child to a well-known skin specialist to be cured of a troublesome eczema of the face, and, as usual, asked the cause of the skin-affection. The surgeon noticed a scar in the neck where a caseous gland had been removed some time before, and then told the mother that it was "not an ordinary eczema, but a *glandular* eczema"! No doubt the word scrofula rose to his lips, but he deemed it prudent to use the term "glandular" instead.

But Koch's discovery of the tubercle bacillus, which he showed was present alike in the "gray granulations," which were regarded as pathognomonic of tubercle, and in the caseous gland masses, which were called scrofulous, more than anything else has given the *coup de grace* to the word scrofulosis. Henceforth caseous degenerations were instances of tuberculosis of the chronic variety, and no other term was needed. The presence or absence of the bacillus is now regarded as giving the clue to what is tubercular and what is not, and the much-abused term "scrofulosis" is perishing, or has perished, in contempt.

We must, however, bear in mind that while much to which the term scrofula has been applied is now more correctly and conveniently included in the domain of tuberculosis, yet much that was formerly called scrofulous is not tuberculous in the sense that the specific bacilli are present. The chronic and inveterate catarrhs of the mucous membranes, the enlarged tonsils, the nasal adenoids, the middle-ear catarrhs, the impetigos, etc., are not tubercular processes. They are often precursors of tuberculosis, and occur in children who are especially liable to become tubercular.

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[1] See Dr. Pye-Smith's Lumleian Lectures on Ætiology, delivered before the Royal College of Physicians, 1892.

May we not ask, Is there not a tendency at the present day to overlook this diathesis, and to look too exclusively to the infective side of tuberculosis? Twenty years ago it was the diathesis, hereditary or acquired, that was all-important. To-day there is surely a danger of forgetting this, and of looking upon tuberculosis as we look upon measles, *as an infectious disease*, and to see danger only in the distribution of tubercular bacilli in sputa, milk, meat, or in other ways. Now, it is certainly true that while there can be no tubercular lesion without the bacillus, yet it requires a suitable soil in which to germinate. It seems very doubtful if it can flourish in healthy tissues. "The soil is of more importance than the seed." The seed seems, in spite of our efforts, to be coextensive with our civilization; in all crowded populations it is ever and universally present, waiting, as it were, for a suitable soil to become parasitic. The congested and irritated lymphatic gland in the neck or mediastinum or mesentery, or an inflamed patch of lung or bone, we know are among its favorite soils. But not only does it attach itself to spots in direct communication with the external world, but also in deep-seated parts where there is no direct communication, such as the epiphysis of the hip, the bodies of the vertebræ, or in the cerebellum. Given a suitable soil, created by an injury and chronic inflammation, in an organ however remote from the surface, there the tubercle bacilli find their way and start their ravages. They seem to be as universal as the lactic acid bacillus, for, given exposure to the air and a suitable temperature, milk has not to wait long before it turns sour. The tubercle bacilli are like the vultures of the desert, which appear to come from nowhere the instant a carcass is provided.

Now, I have no wish to depreciate any efforts which are being made to prevent the distribution of tubercular bacilli in milk or by aërial diffusion from phthisical patients, but I certainly think that what was known as the "scrofulous" diathesis, the catarrhs, the chronic inflammations, and the impetigos, are worthy of the closest study in connection with the question of tuberculosis. This question of a suitable soil to which bacilli can cling and grow will cease to be of importance only when mankind has discovered a germless Arcadia, of which for the present he can only dream.

That the diathesis to which the term scrofula has been applied is a very real one all must admit, while they may refuse to use the word scrofulous and decline to invent a name, though the ugly word "lymphatism" has recently been coined to cover part of the ground. That this disposition is hereditary, but may be aggravated by bad sanitary life conditions, is certain. It is generally allowed that it consists in a tendency to chronic inflammations of mucous membranes, skin, and bone, associated with congestion and hyperplasia of the lymphatic tissue and glands.

There are a certain number of our child population who inherit a greater tendency to chronic inflammations and catarrhs than do other children or adults. These inflammations and catarrhs are inveterate, and occur again and again on the slightest provocation. Of this any number of examples could be given, but it is unnecessary in this article to travel over the whole of the ground. Let us take as an instance a child who is brought to the surgeon with a nasal discharge, conjunctivitis, and photophobia which have lasted for months in spite of home treatment. Perhaps the skin of the upper lip or that around the eyes is excoriated and fretted by the discharge. Perhaps there is some neighboring impetigo with a semi-purulent discharge and formation of crusts. Such a case is not tubercular, at least the rhinitis and the conjunctivitis are not tubercular; but we know very well that it is exceedingly likely to become so. The cervical glands are only too apt, sooner or later, to become inflamed or congested and the seat of a tubercular process.

Take another instance. A child has whooping-cough, and this is accompanied by a bronchitis or bronchial catarrh; the attack will very probably be severe in the "scrofulous" child; the bronchial glands become irritated; a suitable soil is provided for the bacilli of tubercle; gray granulations make their appearance; caseation follows, and the neighboring lung tissue is invaded. There may be a chronic pneumonia accompanying the whooping, which becomes tubercular.

In another instance a child suffers from a long-continued attack or repeated attacks of intestinal catarrh. The adenoid tissue of the mucous membrane and the mesenteric glands are irritated, and tubercular bacilli find a suitable soil in their substance. The peritoneum becomes irritated and shares the fate of the organs which it surrounds, a chronic or subacute peritonitis being started.

In these instances we cannot fail to note that it is the lymphatic system which is the special seat of the tubercular process, and this is particularly so in the young. During early life, when active growth is in progress, the lymphatic system is abnormally active, is specially prone to take on inflammation and to harbor and allow to grow various pathogenic organisms. In the great majority of cases during childhood it is the lymphatic glands, especially those which drain mucous membranes, which are the first hosts of the tubercular bacillus. But before the gland can become a suitable culture-ground there must be a chronic irritation or inflammation taking place in it. Whether septic or other cocci prepare the soil for the specific bacilli we can only conjecture.

Another point we cannot fail to note is, that in some cases the original lesion may be a comparatively mild one, while the glandular lesions may be out of all proportion to the original lesion, and *vice versa*. It is interesting also to note how much more liable glands in the cervical region are to take on suppuration than are the bronchial or mesenteric glands. We know that the bronchial glands and mesenteric glands will suppurate at times, but with nothing like the frequency of the more superficially placed ones. Suppuration is presumably due to the activity of pus cocci, and these are far more likely to find their way into the superficial than into the deep glands which drain the bronchial tubes or alimentary tract. Moreover, injury acts as an exciting cause, and naturally the superficial glands are far more likely to be injured than the deep ones. So far as the gland is concerned, suppuration brings the tubercular process to an end.

In another typical instance we have tubercular disease following injury to a joint. The late Professor Syme, of Edinburgh, used to say of the operation for amputation at the ankle-joint which bears his name, "Gentlemen, this operation is for sprained ankle." He evidently wished to impress upon his class that a neglected injury to the ankle might prove the starting-point of "scrofulous" disease.

Chronic spinal disease, hip-joint disease, and disease of the knee and ankle constantly have their origin in injuries followed by an inflammation of a chronic and inveterate kind, the inflamed bone-tissue becoming a suitable soil for the growth and multiplication of the tubercle bacilli.

One other point seems worthy of remark, and that is, while all tubercular lesions resemble one another in histological characters and in the presence of tubercular bacilli, yet clinically they differ enormously.

No doubt it was the wide difference in these clinical characters that led, in part at least, to one set of lesions being called "scrofulous" and another "tuberculous." The former was supposed to be a less fatal form of disease than the latter. Compare the virulence and speedily fatal termination of an acute miliary tuberculosis with lupus or tuberculosis of the skin, which slowly creeps along, infiltrating the skin and lasting for years; or an acute attack of pulmonary tuberculosis which runs a course of a few months only with a so-called fibroid phthisis, which may last the greater part of a lifetime. Then, again, we note that by far the greater number of cases of acute miliary tuberculosis are fatal, while the more chronic forms have a tendency to exhaust the soil or, at any rate, to come to an end. How frequent it is to find healed tubercular lesions on the post-mortem table! Indeed, it is not uncommon to find five or six healed or healing patches of tuberculosis in the same body.

Tubercular glands of external relation belong to the chronic type of tuberculosis, and appear to be but seldom the centres from which the tubercular process spreads to other parts of the body. But what has already been said about the frequency with which patches of tuberculosis heal in the lungs and elsewhere, and the difficulty of diagnosing them for certain when occurring in the deeper parts of the body, detracts to a large extent from the value of the above statement.

In considering this question we must also remember that the importance of a tubercular process depends to a considerable extent upon its anatomical position. Compare the relative danger to life of a caseous or tubercular gland in the neck, or a patch of lupus, with a tubercular growth on the smaller arteries of the brain, or a tubercular bronchial gland embedded in lung tissue. But beyond this matter of location a tubercular process must depend largely upon soil, as I have already pointed out.

With regard to treatment, it cannot be necessary to say much here, and I will confine my remarks to the treatment of chronic enlargement of the cervical glands,—"scrofula" in the older and narrower sense. It is hardly necessary to insist on the careful treatment of the exciting cause of the glandular enlargement, whatever that may be, with a view to its removal. No one will question the importance, in every case where the glandular enlargement remains after the exciting cause has been removed, of losing no time in placing the child under the most favorable climatic conditions, preferably at the sea-side. Whether the iodine in the exhalation from seaweed or sand has any curative effect may be open to question, but at the sea-side, as compared with inland resorts, the air is purer and there is less dampness. On a sandy soil the rainfall is soon absorbed, and the child can be sent out as soon as the rain is over without the exposure to damp that there would be in the country after heavy rain. In Great Britain there is certainly more sunshine by the sea than in most inland places. In the early stages before caseation commences I believe that good may be achieved by hot fomentations applied by sponges wrung out of hot water, followed by the inunction of an ointment consisting of the yellow oxide of mercury, ex ung. lanolini (five to ten per cent.).

How long is such treatment to be persisted in if unsuccessful? We cannot tell with certainty whether an enlarged gland is simply enlarged, or whether it has become the seat of tubercle and has commenced to caseate. It is doubtless within the experience of most that enlarged glands will gradually disappear, and the presumption is that they have not been the seat of tubercle. At operations for the removal of tubercular glands some simply enlarged will be seen, and these, if let alone, will subside. On the other hand, glands which are firm and hard and have been present for several months are almost certainly caseous, and if they atrophy at all it will be only after many years. Possibly they may remain apparently quiescent for a while and then will quickly suppurate, the skin becoming undermined in the course of a few days.

With regard to surgical procedure I can only speak as an onlooker, but the reader is referred to Dr. Fenger's article, page 580 of this volume. The operation of excision should be undertaken only by one skilled in his craft, as, although the excision of a solitary group of glands may be simple enough, still, the surgeon will probably find deep-lying glands which he did not suspect before the operation was begun, and to remove these satisfactorily and with a minimum of risk to his patient demands considerable nerve and skill. Glands caseous and adherent to the sheath of the deep vessels in the triangle of the neck add to the difficulties of the operation, as well as make it tedious.

In those cases in which the glands are softened before they come under the care of the surgeon, scraping and scooping comprise the only possible treatment.

Excision if possible, scraping if necessary, are the recognized surgical procedures of to-day. Galvano-puncture has gone out of use, being a tedious and unsatisfactory process as compared with excision.

TUBERCULOSIS.

By GEORGE BLUMER, M.D.

TUBERCULOSIS.

Definition.—A specific infectious disease, either local or general, caused by the bacillus tuberculosis (Koch), and having as its most typical lesion nodular bodies known as tubercles.

Etiology.—*The Exciting Cause.*—The organism conceded to be the cause of tuberculosis is the bacillus first described by Koch in 1882. As generally seen, it is a bacillus of from one and a half to three and a half microns in length, and two-tenths of a micron in thickness, often somewhat curved, and staining irregularly, so as to present the so-called beaded appearance. In some instances, however, both under natural circumstances and in cultures, it has not the morphology of a bacillus at all, but appears as long threads (Petrone) or as a branched organism (Klein, Fischel, Coppen-Jones). In a recent examination of sputum it was observed as long threads, woven into tangled masses, and apparently showing true branching. All these facts tend to show that the organism should perhaps be classed with the more complex streptothrix group, and, believing this to be the case, Coppen-Jones has suggested the name tuberculomyces instead of tubercle bacillus, adding avium or hominis as necessary. The organism grows best in the presence of oxygen, but can grow in its absence. It is non-motile, and is strictly a parasite, although capable of living outside of the body for some time under suitable conditions. The bacillus possesses certain staining reactions which are common to only a small number of other organisms, these reactions consisting in a great resistance to stains, and after staining a great resistance to decolorizing agents. These staining properties are perhaps partly due to the large quantity of fat present in the organism; this, according to De Schweinitz and Dorset, amounts to thirty-seven per cent. of the weight of the dried bacilli. Hammerschlag, however, states that the substance taking the stain is probably a combination of an albuminoid substance and cellulose. The other organisms which stain by the ordinary methods used for the tubercle bacillus are the leprosy bacillus, the bacilli found in the smegma, and the bacteria found in the cerumen. The leprosy bacillus is distinguished by the ease with which it takes the ordinary Weigert stain. The peculiar stain of the smegma and cerumen organisms is due to their protecting coat of fat, and disappears when this is removed. The tubercle bacillus does not grow on all the ordinary media, but is cultivated best on media containing glycerin, upon blood-serum, or upon potato. Its growth is slow, colonies taking from ten to fourteen days to develop, and the organism requiring a temperature about that of the body. The organism causing tuberculosis in human beings is of a different species, or, at least, variety, from that causing avian tuberculosis, and, according to Theobald Smith, also from that causing bovine tuberculosis. The products of the tubercle bacillus vary according to the media upon which the organism is grown. They consist of albumoses, trytophane, traces of indol and peptone, and certain undetermined toxic substances. The organism increases by fission, and perhaps, also, by sporulation, though the refractive bodies sometimes seen in the bacilli have not been definitely accepted by all authors as spores. Cruikshank states that he has been able to make out definite spores by means of a very high power lens, and that these are not the sharply stained bodies seen after ordinary staining methods, but are smaller refractive bodies. The tubercle bacillus is found, according to Straus, in the nasal cavities of a certain percentage of normal individuals, particularly those in contact with tubercular patients, and those working in public institutions. It was found by him in the nasal cavities of nine out of twenty-nine hospital attendants, one out of nine attendants in public libraries, and one out of seven theatre attendants.

METHODS OF INTRODUCTION.

Direct Hereditary Transmission.—One can imagine transmission of the tubercle bacillus through the sperm, through the ovum, or through the placenta. Tubercle bacilli have not infrequently been found in the semen, and Jäckh has recently reported eight cases in which the inoculation of spermatozoa from tuberculous individuals caused tuberculosis in animals. Nevertheless, their presence in the spermatozoa does not necessarily imply their presence in the spermatozoon, and the chances of their presence in the individual spermatozoon which fertilizes the ovum would seem to be exceedingly small. All experimental evidence is against paternal transmission, and there is no well-authenticated instance in the human being. Sarwey relates a case of a healthy mother who gave birth to a dead child which had in its spinal column a tubercular focus containing bacilli. The father was tuberculous, and Sarwey reports the case as one of paternal transmission, though his evidence is not conclusive. Transmission directly through the ovum is also improbable, although it has been produced experimentally in birds by egg inoculation. Recently, in a large series of observations, Jäckh was unable by inoculation of the ovaries of tuberculous patients to bring about a single definite result. Baumgarten has in one instance been able to observe tubercle bacilli in a Graafian follicle. The most probable route of infection in direct heredity is the placental one, and actual disease of the placenta does not seem to be necessary, although there probably are lesions present in the form of hemorrhages which allow the passage of micro-organisms.

Londe reports four cases in which inoculation of the placenta gave positive results, though there were no observable histological lesions. In some instances tuberculosis of the placenta is present, as in some of the cases reported by Lehmann. The exact proof of the direct passage of the tubercle bacillus through the placenta is furnished by such observations as those of Bar and Rénon, in which the inoculation of placental blood gave rise to tuberculosis in the inoculated animals. In connection with direct transmission cases of congenital tuberculosis may be considered, and although a considerable number have been reported since Jacobi's case, the number is still, comparatively speaking, very small.

In many instances infants with no gross lesions of the organs have been shown by inoculation experiments to have tubercle bacilli in these organs, portions of them on inoculation producing tuberculosis in animals. Londe inoculated in one case the brain and spleen of an apparently healthy child and produced tuberculosis; in another case he produced the same results with the liver and blood.

Bolognesi in two cases got similar results, as did Birch-Hirschfeld and Landouzy. Cases in which tubercular lesions in the organs were found at birth are rare, not more than eight or nine being reported. All the cases are of direct maternal transmission, and probably were transmitted through the placenta. The weight of evidence is against P. Baumgarten's theory, that in most cases of tuberculosis in children the germ is inherited, and lies latent in the tissues sometimes for years.

Transmission by Inhalation.—Inhalation is undoubtedly one of the most frequent sources of transmission. The transmission does not occur from inhalation of the breath of the patient, or from the fresh sputa, as it is impossible to detach the bacilli from moist surfaces. Dried sputum is the commonest source of infection, but in some instances, no doubt, dried urine or fæces might also act as infecting agencies. In some cases of tuberculosis of the genital tract the urine contains large numbers of tubercle bacilli, and the same is true of the fæces in late cases of intestinal tuberculosis. The organism enters the respiratory tract on dust particles, such infected dust being particularly apt to occur in rooms which have been occupied by consumptives, and in public buildings, such as hospitals, theatres, jails, and libraries. The researches of Straus have shown that the bacilli lodge in the nasal cavities of attendants in such institutions with a moderate degree of frequency, and the work of Cornet and others has demonstrated the presence of tubercle bacilli in the dust of houses, hospitals, and even of the street. The inhaled bacilli are probably not carried directly to the more remote parts of the lung, as the tidal air does not reach these. It would seem, from the frequency of tuberculosis of the bronchial glands, particularly in children, that they were in many instances carried into the neighboring lymphatics, presumably by phagocytes; in other instances they may reach the deeper portions of the lung by direct growth from above downward along the mucous membrane.

The experimental researches of Koch, Weichselbaum, Tappeiner, and others have shown that inhalation is one of the easiest methods of producing tuberculosis in animals. Several conclusive human epidemics go to show that the same is true of human beings.

Transmission by Food.—Transmission by food is generally confined to uncooked food, as cooking destroys the bacilli. Milk is the commonest infecting agent, and the bacillus of tuberculosis is, no doubt, in a fair percentage of the milk on the market. Obermüller found by inoculation that ten per cent. of the guinea-pigs inoculated with ordinary market milk became tuberculous, and thirty-eight per cent. of those inoculated with market cream. Bay by centrifugalization found tubercle bacilli in two per cent. of mixed milk and in fourteen per cent. of unmixed milk. In many instances the cows show no perceptible disease of the udder. There are one or two cases in which tubercle bacilli have been detected in human milk, but such are rare. Niepece has reported a case where a child born of sound parents developed tubercular meningitis after being nursed by a woman whose milk contained tubercle bacilli.

These bacilli have also been found in butter and cheese. Galtier found that cheese was infective in some instances eleven months after the introduction of tubercle bacilli, and Heim found that rancid butter forty days old was capable of producing tuberculosis in guinea-pigs. The infective properties of food are usually destroyed by cooking. The infection of food by flies must always be thought of, as they can carry infected material from one point to another, and the bacilli can pass through their intestinal canal unchanged. That the gastric juice exerts any harmful influence on the tubercle bacillus under natural circumstances is extremely doubtful, as the organism can resist it for from five to six hours outside of the body. Under natural circumstances the bacilli would have a still better chance of escaping unharmed, the gastric juice being diluted, and the bacilli often being coated with food, and more or less protected in this way.

Transmission by Inoculation.—Authentic cases of the inoculation of tuberculosis are not very rare, many of them having occurred in pathological anatomists and assistants in pathological laboratories. J. C. White has reported an interesting case in which local tuberculosis followed the piercing of the ears for ear-rings, and another in which the disease resulted from washing handkerchiefs infected with tubercular sputum. It is stated that in the Riviera the washing of soiled linen is a frequent source of infection. Most of the lesions so produced run an exceedingly chronic course, but in some instances general infection may follow.

PREDISPOSING CAUSES.

Age.—The incidence of tuberculosis varies considerably at various periods between the ages of one and fifteen years. The following are the statistics of Schwer, covering over thirteen hundred cases :

	PER CENT.
One day to four weeks	0.00
Five weeks to nine weeks	0.08
Nine weeks to five months	10.4
Six months to one year	17.5
Two years	26.0
Three years	45.2
Four years	32.9
Five years	37.7
Six years to ten years	35.7
Eleven years to fifteen years	31.5

It is seen that practically no cases occur before the ninth week of life; after that the percentage gradually rises until the third year, and then falls progressively until the fifteenth.

Lannelongue's statistics of external tuberculosis in children show, as is seen, a very small percentage in the first year of life, a large number of cases in the second, third, and fourth years, and after that a gradual diminution up to fifteen. Out of ten hundred and five cases there were from

	CASES.
One to four weeks	4
Five to nine weeks	6
Nine weeks to five months	17
Six months to twelve months	60
One year to two years	144
Third year	107
Fourth year	108
Fifth year	108
Sixth year	95
Seventh year	73

and from then on a gradual decrease up to fifteen. The maximum of frequency, according to Schwer, is at three years, according to Lannelongue, between two and three years.

Sex.—Sex apparently is not a predisposing factor in tuberculosis of children. In Parrot's statistics the males and females are almost evenly divided.

Race.—With regard to race, Billings's statistics, showing that the negro and Irish races are particularly apt to contract the disease, and that the Jews are relatively immune, are probably as correct for children as for adults. It would seem as though tuberculosis must also be very common among the Egyptians, for Milton, of Cairo, publishes statistics of one thousand operations for tuberculous glands alone.

Environment.—Tuberculosis is much more common in cities than in the country. It is particularly rife in the tenement-house portions of cities, where people are crowded together and the streets are narrow and sheltered from the sun and wind. We have under these conditions bad air, generally associated with bad food and bad hygiene, and, further, on account of the lack of sunlight, one of the most powerful agents in the natural destruction of the tubercle bacilli, the number of these in a virulent condition is, no doubt, larger than in the open streets of the city or in the country. Institutional environment also predisposes to tuberculosis, the number of children in institutions dying of tuberculosis, particularly after epidemics of acute disease, being very large.

Individual Predisposition.—It is now generally accepted that the heredity of tuberculosis exists in the vast majority of instances as a predisposition rather than as a direct transmission of the germ. In just what this predisposition consists we do not know, but we recognize certain types of children more prone to the disease than others. The main types are two in number, the tuberculous, with bright eyes, oval face, thin skin, and long bones, and the scrofulous, with heavy, thick lips and hands, opaque skin, and large, thick bones.

Trauma.—The history of falls or blows is so frequent in some forms of tuberculosis, particularly in bone and joint tuberculosis, that we must regard these accidents as causes. Gibney, from an analysis of a large number of cases, states that he believes that a slight injury often develops or acts as an exciting cause of tubercular joint-disease. In this connection may be mentioned the cases of general tuberculosis following operative interference. The question has been thoroughly discussed by Depage and Gallet in a paper read before the Belgian Surgical Society. The conclusions to be drawn from the discussion are that generalization is so rare that it need not be taken into account as a contra-indication to operation, and that it is best avoided by thorough operation, with as little handling of the tissues as possible, and the use of sharp rather than blunt instruments, the curette being particularly dangerous.

Previous Disease.—Of the greatest importance as predisposing factors of tuberculosis are the acute infectious diseases, particularly measles, whooping-cough, and influenza. They probably act both by lowering the resistance and by causing changes in the air-passages which render them more susceptible to the bacillus of tuberculosis. Small-pox, scarlet fever, syphilis, and typhoid also predispose, probably by the lowering of the resistance alone. In many of these instances it is probable that a latent or partly healed tuberculosis already existed, and flamed up again on the resistance being lowered by the acute attack.

Verneuil has reported instances of this sort after malaria and the eruptive fevers.

Canellis has reported over sixty cases of tuberculosis which followed the grippe epidemic of 1890, all of these being apparently directly due to this cause, and Baivy has reported two cases in which latent tuberculosis became active after influenza. Stenosis of the pulmonary artery is usually followed by pulmonary tuberculosis, which is often the cause of death in these cases. On the other hand, Otto in forty-eight cases of disease of the left heart in men found tuberculosis only once, but in one hundred and eighty-five cases of pulmonary tuberculosis he found twenty-nine cases of chronic and four of acute endocarditis.

Chlorosis is also a disease in which tuberculosis is liable to occur. Chronic intestinal catarrh may be a predisposing factor by altering the mucosa so as to allow the entrance of the tubercle bacilli.

PATHOLOGY AND PATHOLOGICAL ANATOMY.

The Gross Appearance of the Tubercle.—The gray miliary tubercle, the most typical lesion of the disease, appears, as its name indicates, in the form of a nodule about the size of a millet-seed, and of a gray, translucent appearance. It has generally been pointed out as the smallest tubercle which can be seen with the naked eye, but, as Virchow has shown, smaller tubercles, which he calls submiliary tubercles, are just on the point of visibility. After undergoing degeneration the miliary tubercle is no longer gray and translucent, but appears as a small nodule, of a grayish-white color when the cells are fatty, and when caseation has taken place as a nodule with a yellow centre and a gray or grayish-white periphery. The fusion of several miliary tubercles causes the conglomerate tubercle, which varies in size from a pea to a pigeon's egg, or even larger. It is distinguished from the miliary tubercle merely by its size and irregular margins, as the general appearance is usually about the same as that of the small caseous tubercle,—*i.e.*, a yellow centre and a gray or grayish-white translucent periphery. Often, even to the naked eye, it can be seen to be made up of two or three single tubercle nodules, parts of whose outlines can still be seen at its periphery. Occasionally large solitary tubercles are found, particularly in the cerebellum and the liver, these resembling, to the naked eye, the large conglomerate tubercle, and being quite often surrounded by a ring of smaller satellites. The tuberculous exudates observed on the serous surfaces are sometimes indistinguishable macroscopically from exudates due to other causes. In most instances, however, either the presence of actual tubercles or the caseous character of the exudate would lead one to suspect a tuberculous origin. Tuberculous granulation-tissue can be absolutely differentiated from ordinary granulation-tissue, in the gross, only by the actual presence of tubercle nodules therein. Tubercular abscesses may contain tubercle nodules in their walls. The pus is usually thicker than in ordinary abscesses, and has a granular appearance.

The Microscopical Appearances of the Tubercle.—It is necessary to state, before entering into a description of these appearances, that they are not pathognomonic of tubercle; other of the infective granulomata, as syphilis, leprosy, actinomycosis, and so on, may produce similar appearances. They may also be produced by other bacilli, by certain of the blastomyces, by foreign bodies, and by the products of the tubercle bacillus itself.

The Nodular Tubercle.—The changes which occur after the introduction of the tubercle bacilli are essentially the following. Immediately after the introduction of the tubercle bacilli these begin to increase in number, at the same time extending from the original seat of inoculation, partly by direct growth and partly by the help of the lymph stream, which takes them up and carries them away from the original seat of deposit. This process continues from the time of introduction to the fifth day, at which period many of the bacilli can be seen in the tissue-cells. At this time the fixed cells of the part begin to divide by the indirect method, the process involving the connective-tissue cells, the endothelium of the vessels, and the cells of the parenchyma of whatever organ is attacked. From this subdivision result cells resembling epithelial cells, but more irregular in shape, which are called epithelioid cells.

Closely following the formation of epithelioid cells there appear in the tubercle two other forms of cells, one the ordinary polynuclear leucocyte, the other a small round cell resembling the lymphocyte of the blood. From their morphology, and from the fact that they appear first at the periphery of the tubercle (this having no blood-supply of its own), it is presumed that they come from the blood, and this view is further substantiated by the fact that these cells do not undergo subdivision, but only regressive changes. The polynuclear leucocytes, which tend to wander into the centre of the tubercle, are much more susceptible to the harmful agents produced by the bacillus than the small round cells; they usually degenerate quite early, and are often seen in the tubercle in very small numbers. The lymphocytes persist longer, and, though some are found in the centre of the tubercle, they are most numerous at the periphery. Later, when degeneration has taken place, polynuclear leucocytes may again appear, invading the necrotic tissue. On the tenth or eleventh day a net-work of fibres can often be made out in the tubercle between the cells. Some of these fibres can usually be seen to connect directly with the surrounding connective tissue, and all are probably the remains of the pre-existing connective tissue of the part. The fibres vary considerably in different tubercles, and in different stages of the same tubercle. Sometimes they form a well-marked reticular net-work, while in other instances they cannot clearly be made out. According to Falk, fibrils of true fibrin are also found in the early stages of tubercle formation, mostly in the periphery of the nodule. Most commonly in old nodules, but also in many instances in fresh ones, there is found still another cell, the giant cell. This cell is usually found in fresh tubercles in the centre of the nodule; in older ones quite often in the growing periphery. It is a large, irregularly round or oval cell, varying in diameter from two to three microns, and often having distinct branching processes. It contains from ten to fifty or sixty nuclei. These cells originate from the epithelioid cells, and are probably due to the injury of their protoplasm by the products of the tubercle bacillus, the injury being powerful enough to inhibit the subdivision of the protoplasm, but not of the nucleus, the latter, therefore, continuing to develop, whilst the former does not. Perhaps the giant cells may also be due to the fusion of several epithelioid cells, and in some instances they seem to be similar to the blood-vessel giant cells occasionally seen in granulation-tissue. The giant cell, like the tubercle itself, generally undergoes central degeneration, so that the nuclei are often found arranged either at the periphery, in the case of the round cell, or at the poles, in the case of the oval cell. The tubercle bacilli are found in the nodules from the early to the late stages, first in the connective-tissue cells, then in the epithelioid cells, and finally, and in chronic tuberculosis quite commonly, in the giant cells. The bacilli are not equally abundant at all times in the same tubercle, nor are they present in equal numbers in tubercles of different organs. In the early stages of tubercle formation the bacilli are not usually very abundant, but become more so after degeneration takes place; they are perhaps seen in largest number in the caseous walls of lung-cavities. As Councilman has pointed out, tubercle bacilli are very hard to find in tubercles of the liver, and the same may be said to be true of tubercles in lymphatic glands. The conglomerate tubercle presents the same histological appearance as the miliary; it contains the same types of cells and undergoes the same changes, differing only in extent. Often in the early stages the exact number of miliary tubercles making up a conglomerate one can be made out, but after extensive degeneration the boundary lines are obliterated, and we see only the extensive caseous centre surrounded by tubercular tissue.

In the walls of sinuses leading from tuberculous joints and bone-disease we find what is known as tubercular granulation-tissue. It is in reality but a diffuse tuberculosis, and contains the same elements as the nodular tubercle. It shows also the same tendency to necrotic changes, and with appropriate staining the tubercle bacillus may be demonstrated. Occasionally tubercle nodules can be seen in it. The walls of tuberculous abscesses present the same appearance as does tubercular granulation-tissue. The contents are not the same as true pus from a histological point of view, but consist of granular and fatty débris, with an occasional polynuclear leucocyte. It is generally held at present, however, that the tubercle bacillus is capable, in rare instances, of producing suppuration, with the formation of true pus. With regard to tuberculous exudates, when not actually caseous, tubercle nodules can generally be found, or, if these are not present, the elements described as composing tuberculous granulation-tissue generally are. In either the granulation-tissue or the exudate early degeneration in some form is almost sure to follow.

Degeneration of Tubercles.—All tubercles ultimately undergo degeneration, this being mainly due to certain of the products of the tubercle bacillus, and only in a slight degree to lack of nutrition, since much larger areas than tubercles may be nourished without direct blood-supply. The common forms of degeneration are caseation, calcification, and sclerosis. Caseation or caseous degeneration is a form of coagulative necrosis, the cells undergoing fatty change subsequent to the coagulative process. This process is the most common degenerative change seen in tuberculosis, and may occur at any period, attacking the smallest and the largest tubercles.

[illegible] the central portion of the tubercle, the peripheral portion representing the growing edge. The caseous material is to the naked eye homogeneous and has a yellowish-white color, and under the microscope is finely granular, containing fat droplets and particles of nuclei. With the ordinary hæmatoxylin and eosin stain the cheesy material stains with eosin, though it often has a bluish tinge, on account of the large number of nuclear fragments which it contains. This caseous material may undergo softening and break down, forming a tuberculous abscess. This process is at times associated with secondary infection of tuberculous areas with the pus organisms, but not necessarily so. At times the fluid parts of the caseous area are absorbed, and nothing remains but a dry, crumbly material, which often becomes entirely surrounded by a dense fibrous capsule.

Calcification usually occurs in caseous areas, and is the result of the deposit of lime salts. It occurs quite frequently in tuberculous glands, particularly those of the bronchial and mesenteric group, and renders those affected of a stony hardness. Small areas of calcification are often seen in other tubercular foci, the lime salts taking a very characteristic, reactive blue stain with the hæmatoxylin. Sclerosis or fibrosis is essentially a reparative process, and is most commonly seen in lung tuberculosis, especially in the so-called fibroid phthisis. It is one of the processes by which the healing of lung tuberculosis takes place,—a not infrequent occurrence, as is shown by observation on the autopsy table. As it occurs in the tubercle, it is a process consisting of a gradual transformation of the tubercle cell into a fibrous tissue cell. In healed tuberculosis the process is not confined to changes in the tubercles themselves, but is often largely aided by the interstitial pneumonia which occurs in the surrounding lung-tissue and shuts off the affected area.

Distribution of Tubercles.—The lesions of tuberculosis may be found in any tissue or organ of the body, though it may be generally stated that the seats of election vary according to age, and that one or two tissues of the body are relatively exempt. In children the seats of election are the lung, the lymphatic glands, the bones and joints, and perhaps we should add the meninges. The statement frequently made that the pancreas and the thyroid and thymus glands are relatively exempt has been disproved by the work of Chiari and of Jacobi. Probably the two tissues most exempt are the voluntary muscles and the large blood-vessels. The former are not infrequently attacked in connection with joint-disease, though often strikingly exempt in tuberculosis of the parietal pleura, which generally stops short at the intercostal muscle. The large vessels are probably more frequently attacked than is generally supposed, for, though only seven or eight cases of tuberculosis of the aorta are on record, the writer has personally seen two cases within the past year. The œsophagus and stomach and the ovary are also relatively free from attacks of tuberculosis. In general tuberculosis, where there is every reason to believe that the blood-current has been infected, the tubercles do not attack all organs equally. This is probably partly due to mechanical reasons, but perhaps also to certain peculiarities of the organs themselves. In general infections with other organisms the same irregularity of distribution has been observed, and it has been supposed by some that perhaps certain organs possess secretions antagonistic to bacterial development.

Symptomatology.—General tuberculosis in children may occur as an acute, a subacute, or a chronic infection. In nearly all instances the origin is an old focus of some sort, most frequently a broken-down lymph gland or an old pulmonary lesion. Generalization may also result from an infection from an external tuberculosis or from bone or joint tuberculosis, and in rare instances is directly due to operative interference in local tuberculosis. The mechanism of generalization is the same in all these cases, the essential cause being the accession of tuberculous material into the blood-stream, generally from a broken-down cheesy mass.

As Weigert has shown, the intima of the small veins, particularly in pulmonary tuberculosis, is very apt to show miliary tubercles, and Koch has pointed out that the small arteries are also at times invaded by the tuberculous process. Hektoen in his recent article shows that in tuberculous meningitis the vessels are almost invariably affected. Ponfick has demonstrated the presence of tubercles in the inner lining of the thoracic duct in several cases, and cases are on record where an adjacent lymph gland has been found perforating directly into the duct. The dissemination of the bacilli in the blood is followed by the formation of emboli in the small vessels of the various organs. These emboli do not consist, as Kockel has shown, of the bacilli alone, but are composed of bacilli surrounded by polynuclear leucocytes. The miliary tubercle resulting develops first in the blood-vessels, the endothelial cells being those first affected.

In the several recorded cases of general tuberculosis in children without apparent pre-existing lesions, it is probable that these were overlooked, and it is easy to see how this could be done when it is borne in mind that tuberculosis in children often attacks parts not easily accessible to examination, as the bones and joints. Of the different forms of acute miliary tuberculosis, that with the predominating symptoms of general infection would seem to be most appropriately described here, the symptoms of the pulmonary and meningeal forms being so intimately related to these organs that they are better discussed with them. Pure acute miliary tuberculosis without localizing symptoms may occur either in an acute or in a chronic form. The acute form is generally spoken of as the typhoid form, the symptoms closely resembling those of that disease. The onset is usually insidious, the patients, if old enough, complaining of a feeling of general malaise, with headache, for from ten days to two weeks before the marked symptoms appear. During this time the patient often has a delicate appearance, and there is well-marked anæmia without apparent cause, associated with progressive loss of weight and appetite. In a few instances the disease appears quite suddenly with sudden rise of temperature. It is probable that not only the onset but the course of the disease is influenced both by the rapidity with which the bacilli enter the circulation and by their virulence in different instances. After the disease is well under way the symptoms are much like those of typhoid,—headache, fever, stupidity, and as physical signs enlargement of the spleen and abdominal tenderness. In some cases diarrhœa is present. Rose spots are not present, but in some cases there is an erythematous rash which may closely resemble them. As the disease advances there is always emaciation and generally a peculiar pallor which may be accompanied by cyanosis. In older children the face often has an anxious expression. The respirations are frequently accelerated out of all proportion to the fever or to the signs of lung-involvement. The pulse is usually accelerated in proportion to the temperature, which ranges from 103° to 105° F. Death may be due to asthenia or may result from the occurrence of an acute meningitis, or more commonly to an acute lung-complication at the termination of the disease. The more chronic form may last from eight to ten weeks, the symptoms being the same as in the acute form, but less marked, the temperature ranging lower and sometimes showing distinct intermissions, and the general condition not being so materially affected. In infants the disease may run a course corresponding clinically to ordinary cases of marasmus. These cases present simply emaciation and pallor with no apparent cause. At first no fever is present, and when this does appear at the end of two or three weeks it is usually not high, ranging between 100° and 102° F. These cases are often accompanied by diarrhœa, anorexia, and other gastro-intestinal symptoms, which are not, as a general rule, due to intestinal tuberculosis, but are caused by the general condition. In the last few days of life symptoms of broncho-pneumonia may appear which at autopsy prove to be due to tuberculous lesions. In connection with acute tuberculosis must be mentioned certain classes of cases first described by Landouzy and the French school. One group of the cases is analogous to cases of streptococcus toxæmia from a local infection, or to cases of tetanus. The tubercular lesions are slight, the symptoms being apparently due to a toxæmia rather than to a septicæmia. In children this type of the disease comes on gradually, often beginning with vomiting; the temperature ranges high, and mental symptoms, usually in the form of depression, are well marked. The abdomen is distended, and both liver and spleen may be enlarged. The disease may last two or three weeks, but in some instances proves fatal in a few days. At autopsy the tuberculous lesions present are out of all proportion to the symptoms observed during life, and may consist of only a slight lung-lesion or a few enlarged glands. Cases have rarely been observed in this country, though Osler has described a typical one occurring in an adult from tuberculous glands of the neck.

Another type of acute tuberculosis has been particularly described by Aviragnet and Jeannel under the name of typho-tuberculosis, or acute infectious tuberculous fever. The disease is stated by Jeannel to begin with a sharp rise of temperature, in which it differs from typhoid fever, the temperature being further characterized by great irregularity, both rising and falling very abruptly, the falls being accompanied by profuse sweating. The temperature chart may resemble that of certain forms of malaria. There is no marked stupor, the intelligence is retained, the appetite may be good, and there are generally alternating diarrhœa and constipation. As a final point, the author lays stress on the rapid disappearance of the fever under antipyrin. As Landouzy points out, in this form, as in the preceding, the post-mortem findings may be slight, and out of all proportion to the clinical symptoms. This disease differs from the preceding in its closer resemblance to typhoid, and in the fact that it is due to a true septicæmia, and not to a toxæmia. Some cases apparently get well, according to the descriptions of Landouzy and Aviragnet.

Diagnosis.—The diagnosis of acute miliary tuberculosis is in most instances difficult, and can be made only by taking into account every detail which can possibly be of assistance. There is no one sign which by itself will lead to a definite diagnosis. Almost the only disease with which acute miliary tuberculosis can be confounded is typhoid fever. In a few instances, with intermitting fever, malaria might be thought of, but the examination of the blood would, of course, quickly eliminate this disease. In making a diagnosis, the general surroundings, the symptoms, the physical signs, and the microscopical and bacteriological examination of the secretions must all be considered. The family history of the patient should always be carefully inquired into, and the possibility of infection from a house or room previously occupied by a tuberculous individual should be thought of. The history of a recent infective disease would be a suspicious circumstance, and a pre-existing glandular or external tuberculosis would make the case a clear one in many instances. With regard to the symptoms, cases of acute miliary tuberculosis differ from typhoid fever chiefly in that the fever is apt to be much more irregular, and that the rose spots are absent. The spleen is apt to be less enlarged than in typhoid, and the pulse relatively quicker as compared with the fever. Certain writers have laid great stress on a peculiar pallor in general tuberculosis, and others have laid emphasis upon the fact that in this disease the respirations are hurried out of all proportion to the fever and the lung-involvement. In the form resembling marasmus in infants the diagnosis would rest mainly on the fact that whilst in the case of tuberculosis there are anæmia and wasting without any appreciable cause, in marasmus a history of some digestive derangement can generally be elicited. Then, again, a low form of fever is often present in the tuberculous process, and in the final stages some localizing symptom, as a lung-lesion, generally shows itself. The ophthalmoscope should be used, when possible, with the hope of discovering tubercles of the choroid. Grasset and Wedel have proposed the use of tuberculin in small doses for purposes of diagnosis. They use it in a very dilute solution of carbolic acid made at the time of the inoculation. Doses of from two-tenths to five-tenths of a milligramme were given subcutaneously, and in no case did unpleasant effects result. The writers claim that by taking the temperature carefully for two or three days before the inoculation, and comparing it with the record subsequent to this, the result of the procedure often materially aids the diagnosis.

The microscopical diagnosis in cases of acute miliary tuberculosis should include the examination of the blood, the sputum, if present, the urine, and the fæces. The blood may be examined both from a histological and from a bacteriological stand-point.

The researches of Warthin, cited by Cabot, show that in acute miliary tuberculosis the number of leucocytes is generally decreased, whilst the differential count shows a relative increase in the polynuclear elements and a moderate decrease in the small mononuclear elements. Mayer's work on the blood in typhoid shows that, while the leucocytes in this disease also are generally decreased, there is a relative increase in both the small and the large mononuclear elements. A case, then, in which the small mononuclears were relatively decreased, with an accompanying general decrease in leucocytes, would lead one to suspect acute miliary tuberculosis, whilst, on the other hand, the relative increase of the small mononuclears would be in favor of typhoid fever.[1] In some cases of both diseases the presence of an inflammatory complication might impair the value of a differential blood-count by producing a leucocytosis. The value of a bacteriological examination of the blood lies in the fact that in certain cases of acute miliary tuberculosis Weichselbaum and other observers have been able to detect the tubercle bacillus in the circulating blood. The detection is very difficult, but possibly might be rendered more easy by the centrifugalization of the blood, as is done with Daland's hæmatokrit. Inoculation of animals would be a reliable method, but, unfortunately, the results are too long in developing in most cases. The sputum should, of course, be examined in older children, though in many instances no tubercle bacilli are found. In younger children, who generally swallow the sputum, the vomitus, if possible, should be obtained and examined. Not enough importance has been placed upon the examination of the fæces in children with pulmonary tuberculosis. In a fair percentage of such cases, if the purulent particles be picked out, they will be found to contain tubercle bacilli, and this in the absence of intestinal tuberculosis. In all cases the urine must be examined for tubercle bacilli, as some cases of generalized tuberculosis originate from genito-urinary tuberculosis. The use of the centrifugal machine is of great aid in many of these examinations, particularly those of the urine and sputum. In connection with the presence of tubercle bacilli in the urine a warning must be given that this organism be not confounded with the smegma bacilli. In order to avoid the confusion, the urine should, particularly in females, be a catheterized specimen.

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[1] Widal's reaction is also of value, p. 167.

Sawyer has stated that in many instances, when no bacilli can be found in the sputum, they can be found in the rectal mucus. If by such means bacilli having the staining reaction of tubercle bacilli be discovered, they should be submitted to the tests for distinguishing the tubercle from the smegma bacilli, for at least one case has come under our observation in which in all probability the smegma bacilli were found in the rectal mucus. Finally, in some instances it might be advisable to resort to lumbar puncture as a means of diagnosis, Kohn having recently reported a case in which tubercle bacilli were found in the cerebro-spinal fluid some time before active meningeal symptoms showed themselves. The most rapid and reliable method of staining bacilli in all these cases is the following. In the case of the sputum or the fæces, the small, purulent particles should be chosen for examination, care being taken in the case of the sputum not to mistake particles of food for these. These purulent particles can best be picked out by spreading the suspected material on some dark surface, preferably on a glass plate painted black on its under side. If necessary, a hand-lens may be used to distinguish the particles. The particle chosen is crushed on a cover-glass and smeared out into an even layer. It is then allowed to dry in the air, and finally passed three times through the flame of the Bunsen to fix it. It is then ready for the stain. The following solutions are used:

1. Ziehl's solution of fuchsin—
    - Distilled water, gm. c;
    - Carbolic acid crystal, gm. v;
    - Alcohol, gm. x;
    - Fuchsin, gm. i.
2. Gabbet's solution—
    - Methyl blue, gm. i–ii;
    - Twenty-five per cent. sulphuric acid, 100 c.c.

The cover-glass is held in the forceps and a few drops of solution 1 are placed on its upper surface; the under side is then heated until bubbles begin to form, when the fuchsin solution is washed off with water and a few drops of the Gabbet solution are placed on the glass. This solution is allowed to remain there from forty-five seconds to one minute, when it is washed off with water and the slip mounted in water or balsam, according as to whether a temporary or a permanent preparation is desired. The whole process takes but two or three minutes, the tubercle bacilli being stained red, the nuclei of the pus-cells and bacteria, other than the tubercle bacilli, being stained blue.

Prognosis.—In the great majority of cases of acute miliary tuberculosis the prognosis is unfavorable. A few of the more chronic cases seem to recover temporarily. If we accept the typho-tuberculosis of certain French writers, we must recognize a class of cases which recover with relative frequency.

Treatment.—The treatment of tuberculosis should be prophylactic as well as curative. As Dr. Jacobi has pointed out in his article in this Cyclopædia, the contagion is mostly caused by the dried sputum; hence means to prevent this method of contamination are most required. Laws against spitting in public buildings and conveyances, except into proper receptacles, should be strictly enforced. All sputum should be either burned or placed under such conditions that disintegration takes place before drying. Patients with lung tuberculosis should use at their homes proper receptacles; elsewhere they should expectorate into rags, which can be subsequently burned.

The hygienic treatment of the disease is very important, and consists of fresh air, good food, and warm clothing. Change of climate may be beneficial in children as in adults, the climates most recommended in this country being the Adirondacks and the pine regions of Carolina, or, in the West, Southern California, Colorado, New Mexico, and Arizona. As general supportive measures, cod-liver oil, the hypophosphites, arsenic, or strychnine may be given.

So far as drug-treatment directed towards the disease process itself is concerned, creosote or its active principle, guaiacol, seems to be the most satisfactory remedy. It should be given in as large doses as can be borne by the patient.

It is necessary here to speak of the various serum treatments which have been tried within the past two years. The most important of these remedies are Maragliano's anti-tubercular serum, Paquin's anti-tubercular serum, and Klebs's antiphthisin. In Maragliano's first four hundred and twelve cases there was apparent cure in sixty-seven, notable improvement in one hundred and sixty-four, no improvement in one hundred and fifty-four, and death in forty-seven cases. Paquin claims cure in every case in the first stages of the disease, and several cases are reported by other observers in the more advanced stages which were markedly benefited by his serum. Klebs's antiphthisin has been claimed to be of benefit by Dennison and others.

It may be said in general that of all the serum treatments at present existing none have the confidence of the majority of the medical profession, and it would seem that they are perhaps only the forerunners of more powerful and more satisfactory remedies based on the same principles.

INFANTILE SCURVY.

By THOMAS BARLOW, M.D., F.R.C.P.

INTRODUCTORY NOTE.

In the second volume of this Cyclopædia the present writer gave a detailed record of illustrative cases intended to show the transition between typical scurvy as it occurs in adult life and childhood, and the more obscure and mixed forms of what he believed to be essentially the same disease in infancy. The results of the post-mortem examinations then available on the infant form of the disease were described, and the general conclusions as to the etiology and treatment of the malady were formulated in accordance with lines originally laid down by Cheadle in his papers in 1878, 1882, and 1883, and by the present writer in 1883.

Since that date recorded experience on this subject has greatly increased. This is partly accounted for by the fact that observation has been awakened, but there is reason to suspect, also, that the disease is on the increase.

A large number of cases have been reported in America and Germany as well as in Great Britain. The writer's personal records include more than one hundred cases, the results of which were embodied in the Bradshawe Lecture in 1894.

The editor of the present supplemental volume has considered the subject worthy of another article, discussing the problems of the disease in a more systematized method and embodying the later experience.

The reader is, however, referred to the detailed reports of the cases in vol. ii. as a basis for the present review, and it is necessary to examine the drawings there given in order to understand the section on morbid anatomy in the present article.

Definition.—Infantile scurvy is a disease which is characterized by marked anæmia and severe pains connected with the bones. Its chief anatomical feature is the extravasation of blood between the periosteum and the shaft of the bones of the lower limbs; during the predentition period the subperiosteal regions may be the exclusive site of hemorrhage, but after the eruption of the teeth extravasation in the gums occurs, as in the scurvy of adults and children, though, as a rule, with less severity. That which distinguishes infantile scurvy from other anæmic hemorrhagic diseases is its

well as curative. [illegible] manifestations, by the administration of
pædia, the conta[illegible] vegetables and fruits.
prevent this [illegible] of infantile scurvy is generally abrupt.
spitting in pub[illegible] regarded by parent and nurse as pre-
should be [illegible] considerable covering of subcutaneous fat,
under such [illegible] of rickets afforded by the beaded ribs and
tients with [illegible] junctions less striking than they otherwise
elsewh[illegible] is observed to become paler and less mobile than
burne[illegible] winces and cries when one of the lower limbs is
Th[illegible] when it is lifted out of the bath. Within a day
of [illegible] limb likewise becomes tender and both limbs droop.
be[illegible] has attempted to stand or walk, he now no longer
[illegible] attempts. At this stage the hip-joints are often kept semi-
[illegible] legs hang down in a condition of pseudo-paralysis. A care-
[illegible] have already observed that the maximum tenderness is near
[illegible] ankles. There now appears a little swelling of one or both
[illegible] This swelling is symmetrical on the two sides, and yet not
[illegible] The lower third of one leg and the upper third of the other
[illegible] affected, and the lower third of one thigh and the upper third of the other. There is no alteration in the color of the skin over the swelling, nor is there any local heat over it. The veins about the ankles and on the dorsum of the foot are sometimes a little distended. Pitting, if present, is slight, but the legs and feet look shiny and stretched. In a day or two the swelling extends. Then, if not previously affected, one or both thighs may suffer. Tenderness and local swelling become manifest above the knees and below the hips for a varying extent, and give the impression of some deep-seated effusion which forms a partial or general sheath round the shaft of each femur. At this stage the limbs are generally extended and their bulk is definitely increased and they feel heavier than normal; the powerlessness is very striking; the nurse, and often the doctor, believe that the child is paralyzed. By this time the pallor of the child has become greatly accentuated. He lies on his back, and, although he turns his head with an anxious look on the face whenever a stranger approaches, he seems to have lost the power to raise himself. As before, the lower limbs are exceedingly sensitive. There are piercing screams which, to a casual observer, might suggest meningitis, but they are predominantly related to movements or threatened movements of the lower limbs. There is, however, some tenderness in other parts of the body. The upper limbs, the head, and the trunk may be tender. Some swelling may occur just above the lower forearm epiphyses or below the shoulders. Also a thickening round the shaft of the humerus starting from the upper or lower epiphysial junction may appear. Pseudo-paralysis of one or other upper limb may be present. *But the upper-limb affection is in every respect less marked than the affection of the lower limbs, and in many cases it is not demonstrable clinically.*

On the back of the trunk rounded swellings may occur over the infraspinous fossa of one or both scapulæ. Similar swellings may occur over the iliac portions of the innominate near the crest of the bone. Deep thickening has been found for a varying distance in the groove on each side of the dorsal and lumbar spines. The changes observed in the thorax are very striking. The sternum with the costal cartilages present an appearance as though they had been thrust backward *en bloc*, so that they are on a plane posterior to their normal situation. This alteration is brought about, as we shall learn in the section on morbid anatomy, by a series of partial fractures in the anterior extremities of the ribs. Slight crepitus may be obtained external to the junction of the costal cartilages with the ribs.

In more advanced stages of the disease, soft crepitus is sometimes obtainable near the epiphysial junctions of the upper and lower ends of the femora and near the upper epiphysial junctions of the tibiæ, and in rare cases below the upper epiphysial junctions of the humeri. Such fractures when they occur are generally symmetrical.

Fracture rarely occurs in the middle of the femur, and it may be produced by the minimum of violence, such as a clumsy movement by the nurse; but the fractures near the epiphyses appear without evidence of any violence whatever.

Deep-seated swelling may occur on the lateral aspects of the cranium and even on the facial bones, and the associated tenderness is sometimes excessive.

One or both orbits often present remarkable changes. Within a few days of the onset, suddenly, and without obvious cause, there occurs a moderate proptosis downward and forward of one or both eyeballs. This arrives at its maximum within twenty-four hours, and then a deep-seated ecchymosis and some thickening of the upper eyelid are found, and it is obvious that an effusion of some kind must have occurred in the back and upper part of the orbit. Both orbits are generally affected, but they are often affected on successive days and to an unequal extent. The exophthalmos is not extreme, tension of the eyeball is not increased, and the ophthalmoscope yields no evidence of hemorrhage or inflammation in the deeper structures of the eye. The child obviously suffers distress from these eye-troubles, and often cries, especially at their onset. There is no local heat or other suggestion of inflammatory reaction. Sometimes a little concomitant ecchymosis of the ocular conjunctiva occurs, but, as a rule, it is only the deeper part of the upper lid in which extravasation is manifest, and the color of the lid is a very deep brown and not that of the ordinary superficial "black eye." The appearance presented is, indeed, very like that which is sometimes found in fracture of the anterior part of the basis cranii, followed by extravasation in the posterior and upper part of the orbit.

As the foregoing symptoms are related to the osseous system and are the most significant in the disease under discussion, it will be convenient to recapitulate the general features which they have in common.

(*a*) There is generally symmetry in these lesions, but there is often difference in severity of the lesion on the two sides of the body.

(*b*) When both sides are affected there is generally a slight interval of time between the incidence on the two sides.

(*c*) Although there are swelling and tenderness, there is no local heat over these lesions, and, as a rule, although there may be concomitant ecchymoses of the skin, there is no extravasation in the skin directly over the site of the lesion.

(*d*) With every successive part affected the general anæmia becomes more profound.

THE SKIN.

Besides the marked and increasing pallor, there becomes manifest during the advance of the disease a striking muddy or sallow tint of the skin. Bruise-like hemorrhagic areas may be present in severe cases. They are probably induced by slight pressure. Small purpuric spots are decidedly uncommon. In view of the severe progressive anæmia, it is indeed remarkable how meagre on the whole is the amount of hemorrhage which occurs in the superficial skin structures. If the patient be rickety, the head-sweating which is characteristic of rickets is often increased.

THE GUMS.

In infantile scurvy, if no teeth have appeared, there is nothing characteristic in the gums. If, on the other hand, teeth have emerged, the existence of hemorrhagic granulations encircling the teeth which are above the gum can generally be observed. These granulations appear either contemporaneously with the limb-affection or shortly afterwards. Those portions of the gums through which no teeth have emerged are absolutely free from granulations. But careful inspection will often reveal the existence of minute islands of ecchymosis on the surface of the gum or just below it, corresponding with the sites of the teeth which are about to emerge.

These points are illustrated in the drawing on page 274 in vol. ii. of this Cyclopædia.

In cases of moderate severity the gum-affection often does not advance beyond the above stage. It is generally more marked in the upper than in the lower gum. If several teeth have emerged, the gum-affection then becomes more pronounced; the granulations may enlarge so much as to project from the mouth; they bleed readily, interfere with the feeding of the child, and cause much fetor. It is thus one of the most important facts about the disease that the sponginess of the gums is in direct proportion to the number of teeth which have appeared; and the absence of gum-affection in toothless infants affected with scurvy is one of the reasons which delayed the identification of the real nature of the disease.

ALIMENTARY TRACT.

There is no digestive symptom definitely characteristic, beyond the special form of stomatitis described above and the fetor and difficulty of feeding which occur in the severe examples of it. The child sometimes rejects its food, but vomiting, if present, is rarely severe. A few cases of melæna have occurred.

RESPIRATORY SYSTEM.

The breathing becomes shallow and increased in frequency when the thoracic deformity before described is very marked. This is often the only respiratory change, but in the profoundly anæmic cases death from pulmonary complication is to be feared. When the disease is fatal, areas of broncho-pneumonia and of pulmonary apoplexy and small hemorrhagic effusions into the pleuræ may be found post mortem, although their existence during life escaped detection.

CIRCULATORY SYSTEM.

The symptoms are those of profound anæmia. The blood shows concomitant impoverishment, both in the number of the red corpuscles and in the amount of hæmoglobin, but there is capacity for rapid recovery under proper dietetic treatment, unlike that of any of the other forms of grave anæmia.

THE URINE.

Considerable excess of uric-acid deposit is not uncommon. Diffused blood in small quantity in the urine is very common, and albumen commensurate with the amount of blood is present. Blood-corpuscles and a few small blood-casts may be found microscopically. It is obvious that small renal apoplexies occur, but there is no evidence of nephritis.

NERVOUS SYSTEM.

If the infant be rickety, laryngismus stridulus often becomes accentuated during the course of the malady which we are considering. The writer has also seen tetany present in the course of infantile scurvy. Such symptoms have been cited in support of the old view that the disease is to be considered as acute rickets, which view we shall discuss in the section on etiology.

We have already referred to the extreme tenderness of the head, associated with swellings of the lateral portions of the cranium and of the face-bones.

In rare cases other head-symptoms of great gravity may occur,—viz., convulsions and a state of semi-coma supervening on extreme exhaustion and anæmia, and lasting for a few days, but not necessarily fatal.

The symptoms connected with the eyeballs have already been described.

There has been no proof given hitherto of affection of the spinal cord, of the spinal membranes, or of the peripheral nerves, although the pseudo-paralysis of the lower limbs has repeatedly given rise to the erroneous diagnosis of paraplegia. With proper care the knee-jerks can be obtained even in very severe cases.

THE BODY TEMPERATURE.

Undue stress has been laid by some writers on the pyrexia in this disease, and it is true that sometimes there is a rise of temperature. It is often raised for one or two days, when successive lesions appear, especially if there be much tension, but it is rarely higher than 100° F. or 101° F. It is at other times normal or even subnormal.

PROGRESS AND DURATION.

There are many degrees of infantile scurvy, and the duration varies within considerable limits.

The mild cases, and even the moderately severe ones, generally recover within two or three months. The severe cases may last six months, and if suitable and adequate food be withheld, there may be recrudescence. The very severe cases are by no means always fatal.

A moderate case, if treated properly with antiscorbutic remedies, shows almost immediate improvement, and recovers in a month, so far as gum-affection, tenderness of limbs, pseudo-paralysis, and anæmia are concerned. During the recovery of a typical case the subsidence of tenderness and general swelling of the limbs by degrees permits of the identification of a sheath-like investment of imperfect bone which has formed round that part of the shaft which was primarily affected. In the later stages of recovery this sheath-like investment undergoes slow involution and after a time becomes unrecognizable, although the ordinary signs of rickets are still manifest. Union takes place in the fractures near the epiphyses with little or no displacement. The deformity of the chest recovers completely, and the orbital lesions leave no permanent change.

In the severe cases, when anæmia and exhaustion are extreme, and the proper food is withheld, death may ensue from some intercurrent ailment,—*e.g.*, pneumonia, diarrhœa, or one of the exanthemata.

There is no infantile disease, not even congenital syphilis, in which there is more striking modification of symptoms under early suitable treatment than in the one under discussion.

MORBID ANATOMY.

Osseous System.—Subperiosteal hemorrhage is the typical feature. The long bones of the lower extremities are first affected, and of these the femur and the tibia predominantly.

The fibula is only slightly affected, as a rule, and the external surface of the ilium near the crest is the part of the pelvis which suffers most. The commencing zone of extravasation in the long bones is near the junction of the shaft with the epiphysis, and in slight and moderate cases it may not extend beyond this region; in severe cases the whole shaft becomes invested with a layer of coagulum. The blood is manifestly derived from the periosteal vessels; the periosteum is raised up like a sheath and on its inner surface distended ramiform vessels can be seen in great abundance; also bridges of coagulum extend between the upraised periosteum and the coagulum which invests the shaft. The upraised periosteum shows, often, evidences of its osteogenic function by thin deposits of new granular bone, which ultimately give rise to the imperfect bony sheath described in the clinical section as occurring in the severe cases. When the shaft is stripped of coagulum it is found to be smooth and white, but there is no evidence of periostitis. It is remarkable that necrosis does not occur. Fractures when present occur most commonly just abovĕ the lower and below the upper epiphysis, and during the autopsy the shaft becomes readily separated from its upper and lower attachments, almost like a sequestrum.

There is little or no displacement until the periosteal sheath has been incised. The anatomical characters above described are illustrated in the drawing given on page 275 in vol. ii. of this Cyclopædia. Much less commonly there is fracture across the shaft. In the active stage here described there is no indication of callus. A vertical section of the shaft in a severe case shows also extensive endosteal hemorrhage, and in the most severe cases there is rarefaction of the bony structure, so that when the medulla with the blood-clot has been removed the shaft may be represented only by a mere shell of bone. On microscopic examination the periosteum shows much vascularity, but no cellular infiltration. Extensive hemorrhage is shown in the deeper portions. Considerable absorption of trabecular structure is indicated in the shaft, and enlarged spaces with eroded margins are well marked. Rickety changes in the ossifying zone occur in various stages.

The joints are usually normal. (In one case only the writer found a very small hemorrhage in the synovial membrane of one hip-joint, but this was insignificant and exceptional.)

The deeper layers of the muscles in contact with the vascular periosteum show in severe cases extensive blood extravasation. The superficial layers in such cases are thinned out and paler than normal in consequence of the infiltration with blood-serum, but coagulum is not found in these layers.

In the light of post-mortem investigation it is easy to understand the group of symptoms which have been pointed out in the clinical section as specially present in connection with the lower limbs,—viz., the weight which is brought about by the considerable outpour of blood into the periosteal sac and the deep muscles, the tenseness, the absence of local heat or of ecchymosis at the surface, the tenderness, and the pseudo-paralysis.

In the upper limbs occur lesions similar to those which have been described in the lower limbs, but notably less severe. Symmetrical fractures have been found below the upper epiphyses of the humeri.

The changes in connection with the scapulæ were in one case found to be more severe than those around the bones of the upper limb. Subperiosteal hemorrhage had occurred in the venter and in the supraspinous and infraspinous fossæ, and the amount had been so great in the last situations as to give rise to marked prominences obvious during life. Osseous material was found deposited in thin plates over the blood-clot by the upraised periosteum.

The changes found in the ribs are strictly analogous to those affecting the limb bones. The rib may be bare and separated for a considerable extent from its periosteum by blood extravasation. The depression or falling back of the sternum with the costal cartilages already described in the symptomatology is seen post mortem to be due to a series of fractures and displacements of the anterior extremity of the ribs just external to the junction with the costal cartilage. In the severe cases, on section of a rib there is found considerable rarefaction of the centre, so that the bony structure is only a shell full of broken-down blood-clot. On the outer surface of the cranium and the facial bones subperiosteal extravasation has likewise been found, and Möller has recorded one case, probably of this nature, in which hemorrhage had occurred on the inner surface of the frontal and had extended into the orbits separating the periosteum from the orbital plates, causing proptosis such as we have described in the symptomatology. Hemorrhagic pachymeningitis has been recorded in infantile scurvy, and it is probable that the hemorrhage has been derived from the inner surface of the cranial bones, as in Möller's case.

VISCERA.

Hemorrhagic extravasations have been found in the lungs with or without pneumonia.

Accompanying the lung-lesions, or independently of them, hemorrhagic effusions in the pleural cavity and petechiæ on the parietal pleura occur.

In the same or different cases small hemorrhagic foci have been found in the spleen, the kidneys, the lymphatic and mesenteric glands, and the solitary glands and Peyer's patches of the intestines. Dr. Cheadle and Dr. Sutherland have each recorded a case in which gum extravasations and visceral hemorrhage occurred, but in which, strange to say, the bones and muscles were free.

PATHOGENESIS AND ETIOLOGY.

Let us now consider what is the essential nature of this disease, and what is the cause of it. The conditions under which it arises may be first discussed.

Age.—Before the age of five months this group of symptoms is very rare, and, taking the range of the first two years of life, the most common incidence is between the seventh and the eighteenth month.

There is no difference to be made out with regard to the sexes.

As to season, it would appear that the cases which occur during the colder months are more protracted and more grave than those which occur during the summer.

As to social status, some of the fatal cases have been poor children, but the disease has been recognized in all ranks, and many more examples of moderate severity have been found among the children of the rich than among the impoverished. Thus the disease is more frequently found in the course of private practice than in hospital clinics.

With regard to congenital syphilis, Steiner has stated that out of ten cases of so-called acute rickets observed by him two supervened on congenital syphilis at the age of four months, and that these two were fatal. No record of autopsy is given in either of these cases. It may, in the absence of anatomical verification, fairly be asked whether the bone-affection from which these children suffered was not, after all, the special syphilitic affection of the extremities of the shafts first described by Wegner and Parrot. There are many clinical resemblances between the so-called acute rickets and the early syphilitic bone-affection which can be discriminated only by post-mortem investigation. Symmetrical pseudo-paralysis occurs in both, and in both some displacement of epiphyses from shaft may ensue. In both some thickening may be felt for a little distance up and round the shaft. But in the syphilitic bone-disease the pain and tenderness are trivial, while in the disease we are now considering they are extreme. Also in the syphilitic affection the general swelling of the limb is slight instead of being tense and shiny. In the syphilitic affection concomitant joint-effusion and even suppuration are not rare, but suppuration never occurs in the so-called acute rickets, and joint-effusion is practically unknown. The age of the patient gives some assistance in diagnosis. The syphilitic affection may appear at three months, or, indeed, any time after six weeks, while infantile scurvy is rare before five months. Post-mortem examination gives conclusive distinctions. The primary change in the syphilitic affection is an endosteal one, and consists of what M. Parrot calls a gelatiniform alteration in the osseous material at the extremity of the shaft. This may extend for a varying distance up the centre of the shaft. In the majority of cases there is a concomitant perichondritis and periostitis, and in a few a little suppuration at the extremities of the epiphysial line, and sometimes (as can be detected clinically) an effusion in the neighboring joint, but there is no hemorrhagic extravasation between the shaft and the periosteum such as we have described in cases of infantile scurvy. It seems to the writer a tenable view that Steiner's two fatal cases were syphilitic throughout, and that they ought not to have been classed with the group now under review.

In many cases of congenital syphilis it is quite true that by the sixth month all *outward* manifestations have passed away, and in any given case of infantile scurvy it is, no doubt, difficult to exclude absolutely the possibility of previous syphilis. Nevertheless, as a rule, the histories of the cases which now concern us do not yield any support to the hypothesis of a syphilitic origin, and, as we have seen, the autopsies which are available show no lesion characteristic of congenital syphilis. To sum up, we may conclude that there is no evidence that syphilis plays any part in the causation of the symptoms.

A more important question is the relation between the symptoms and lesions of what we have called infantile scurvy and those of typical rickets. For years past these cases have been described by the German authorities under the designation of *acute* rickets. But anatomical verification was singularly meagre in the early records, and the designation given was considered unsatisfactory by the very writers who employed it. It is undoubted that in the great majority of cases some amount of rickets is present, as evidenced by beading of the ribs, enlargement of the epiphyses, and delayed teething. Moreover, head-sweating and laryngismus stridulus, if previously present, are often increased during the development of the disease now under discussion. It is, however, to be observed that after the disappearance of the special group of limb-symptoms which we have described, in a large number of cases there remain persistent signs of ordinary rickets, and that these signs pass through the typical stages of involution which belong to that rather chronic disease. The amount of rickety change in the shape of spongioid ossification at the epiphysial junction region found post mortem has been very variable, and in some of the milder cases which recovered the evidence of rickets was insignificant; in some it was impossible on clinical grounds to say that there was any.

When the question is approached from another side, it is found that in post-mortem examination of typical indubitable rickets hemorrhagic subperiosteal extravasations are conspicuously absent. Even in very aggravated instances in which the bony deformity is excessive and either multiple fractures occur or extreme bone softening is present, these special hemorrhagic lesions do not occur, and this in spite of grave cachexia. Some of the German writers have frankly acknowledged this difficulty, and have tried to draw the distinction between *acute* rickets, or "rickets beginning acutely," and *severe* rickets.

It is obvious that the phenomena of the disease under consideration and those of typical rickets do not present what logicians describe as "concomitant variations." In regard to any possible relation to hæmophilia, it may be remarked (1) that none of the recorded cases seem to belong to families of "bleeders," and (2) that the general absence of joint-affection is quite unlike hæmophilia, in which the occurrence of special joint-attacks is characteristic. The protracted course, the typical form of limb-affection, and the rarity of small skin extravasations distinguish this disease from purpura hæmorrhagica. The spongy gums also, when present, separate the disease alike from hæmophilia and from purpura hæmorrhagica.

The striking response to diet-change presently to be described distinguishes the disease from other forms of hemorrhagic malady.

With what known disease, then, has the group of symptoms described and lesions found the most affinity? We maintain that it presents most affinity with scurvy.

The bone-lesions which we have described at length are of the same character as those which have been described in undoubted scurvy of adults and adolescents. Poupart, the great French surgeon, in 1699 made some dissections of patients who had died of scurvy in the Hôpital St. Louis at Paris. He describes a slight grating of limb bones when they were moved, and he found on post-mortem examination that in these cases the epiphyses were separated from their shafts. He points out that young people under eighteen years of age were specially liable to have their epiphyses separated in scurvy. He also mentions that in "some we perceived a small low noise when they breathed, and that in them the cartilages with the sternum were found separated from the ribs." This condition is exactly that which has been described in our infantile cases, and in the records of scurvy there are many other instances to be found, as, for example, Godechen's cases of fractures of the anterior extremities of the ribs.

Lind and Budd both record subperiosteal blood extravasations round the bones of the lower extremities and in connection with the jaw. Several necropsies were made during the last siege of Paris on young scorbutic subjects, and subperiosteal blood extravasation was found round the tibia. A marked example of this kind was communicated to the writer by M. Vidal.

So also endosteal blood extravasations, both in the long bones and in the ribs, and fractures resulting from rarefying osteitis, have been found in undoubted scurvy, and these lesions are precisely those which we have found in the infantile cases under consideration. The lower-limb affection is curiously predominant both in adult scurvy and in the infantile cases.

If we pass to other tissues, the similarities are quite as striking. Thus the superficial muscular masses show serous exudations and the deeper muscular masses (nearer the periosteum) show blood-clot alike in both, and in both there is often a certain amount of muscular wasting. The visceral lesions in the pleuræ, lungs, etc., are alike. The profound anæmia and the pallor accompanied by the muddy tint which is probably due to reabsorption of altered hæmoglobin occur in both groups. With respect to the crucial question of the gums, we have seen that the infantile cases may be divided into those having limb-symptoms with spongy gums, and those having limb-symptoms without spongy gums, but that the limb-symptoms are the same in both. We have further seen that the sponginess is conditioned by the presence of teeth. When several teeth have emerged the sponginess is very pronounced, and in severe cases the bleeding, fetor, and projecting granulations of the gums have been undistinguishable from that which is found in adult scurvy. When no teeth are present there is no sponginess, though careful inspection may reveal minute ecchymoses over the sites of the teeth which are shortly to appear. The sponginess of the gums is generally regarded as pathognomonic of adult scurvy, but there are some important collateral observations bearing on this point which deserve consideration. In large groups of men subject to identical scurvy-producing conditions it has been established that a toothless person may present weakness of lower limbs, anæmia, and cachexia, and yet be entirely exempt from sponginess of gums, while his com-patients have typical spongy gums. It is also well known that in mild cases of land-scurvy sponginess is limited strictly to the neighborhood of those teeth which remain; if there be large spaces where the teeth have dropped out, no sponginess appears in these spaces. In the original article in vol. ii. of this Cyclopaedia a series of five cases are given of children ranging between the ages of two years and ten years. In these cases the scurvy was undoubted and rapidly yielded to dietetic treatment. The gum-affection was much more severe than in the infantile cases; the limb-affection, though giving great distress and affecting the lower limbs predominantly, was distinctly less severe than in the infantile group. Thus we have in the cases occurring in childhood a middle form between the infantile and the adult scurvy. The inverse relation as to severity of gum-affection versus limb-affection is reasonable when we remember the physiological differences which are present. Before the emergence of the teeth the aggregate of blood-vessels and the blood-supply are smaller, and the liability to attrition is less than at a subsequent period, when every tooth means the existence of a large leash of blood-vessels. But the physiological activity of the growing bones of an infant is profound from the very outset. Changes in general nutrition modify this activity in a very rapid and subtle way. Suppose there has been induced a condition of rickets with its typical alteration along the epiphysial junction zone and in the periosteum, there is then a prepared soil where hemorrhagic lesions can develop if an adequate blood-change is present. Let us look at it from another side. Rickets pure and simple, so far as experience teaches, does not give rise to these hemorrhagic lesions. If it were so, hemorrhagic cases would be much more common, because rickets, in all grades of severity, is so common a disease. Scurvy, on the other hand, we know from adult experience is efficient in the production of such lesions. It seems reasonable, then, that if in our infantile cases the scorbutic blood-change comes into play, the rickety alteration already present may act as a physiological determinant of the regions where the scurvy becomes most manifest.

The question now arises, Is there anything in the antecedent conditions of these infants at all parallel with those under which adult scurvy appears? What are the antecedent conditions under which adult scurvy appears? There are many things which predispose to it,—lack of sunshine, cold, exhaustion, etc.,—but there is conclusive proof that prolonged deprivation of fresh vegetables, or of *their equivalents*, is the most constant antecedent.

The qualifications are important. To produce the disease, the deprivation must be prolonged, for it is clear that the organism is capable of drawing on its reserves for varying periods, so as to neutralize the deprivation of a complete aliment. Again, on the question of equivalents, we now know that fresh raw meat and blood and fresh milk are antiscorbutic as well as fresh vegetables and fresh fruit juices, though probably not in so rapid a fashion.

Considered quantitatively, it seems that much larger amounts of fresh, uncooked meat and fresh milk are requisite as antiscorbutics than of fresh vegetables and fresh fruit juices.

The chemistry of scurvy is still an incompletely solved problem, but the researches of Garrod, Ralfe, and others point to a diminished alkalinity of the blood, and almost certainly to a defect in the presentment of the saline constituents of the food, and in the readiness with which they give up their bases. It is probable that there is a biological as well as a chemical side to the problem. The further we get from a living food, the less appears to be its antiscorbutic efficacy. Fresh vegetables are more rapid antiscorbutics than cooked or preserved vegetables, and the mere saline constituents of vegetables are notably inefficient. Raw meat and blood are more antiscorbutic than cooked meat, and raw meat juice than beef tea. It will, I believe, be proved by experience that raw, uncooked milk is more antiscorbutic than cooked milk.

To bring home the parallel between the antecedents of our infantile group and those of adult scurvy, we see that it does not consist in faulty hygiene. A very large number of the infantile cases have been nurtured in healthy homes and with good surroundings.

But in the matter of food, we find that at the *time* of *onset* of symptoms in no single undoubted case has the child been breast-fed. In a considerable majority, when full histories have been obtained, it is ascertained that these infants have been nourished on "preserved foods." First come those fed on proprietary foods, prepared for use by the addition of water to certain powders. After these come cases brought up on the different forms of condensed milk and the proprietary foods made with condensed milk. There is a large group in which, although milk has been given, it has been very dilute and accompanied by proprietary food, and the dilution has been continued through the later months of infancy. There has thus been either a deprivation or a marked diminution of fresh living food.

Let it be granted that such a diet is a scorbutic diet. The reasonableness of this hypothesis will be tested in a given case by altering the diet in the antiscorbutic direction, making no other change, and noting the evolution of the symptoms. Thus, for condensed milk let fresh cow's milk be substituted, and for extremely dilute cow's milk let undiluted fresh cow's milk be substituted. For example, to a child six months old, suffering from the symptoms described, let a full pint be given. Replace the proprietary food by some sieved potato, to be mixed with the milk. Add also a tablespoonful of fresh gravy. During the day, in divided doses, give one tablespoonful of juice of orange or grapes mixed with water, as required.

It will be found, as a rule, that the food thus changed is taken greedily and without disturbance of digestion. The sponginess of the gums is the first symptom to recede, and this disappears within three days. The tenderness of the limbs rapidly lessens; no new limb-swelling appears, though it takes some time for the swelling already present to subside. The screaming and irritability very soon pass away, and the child begins to sit up in bed, and subsequently to move the limbs voluntarily. The anæmia improves, and if there has been hæmaturia it is arrested almost immediately. The progress of the disease is obviously controlled.

Fresh air and sunshine, though they will not prevent the advent of the disease, probably aid its recovery when the proper change has been made in the diet.

It is noteworthy that in not a few cases after the marasmus has subsided, and the principal symptoms have been brought under control, the greediness for the altered food becomes lessened and the infant seems to be unable to digest the full amounts of vegetable and undiluted cow's milk which at first were so readily assimilated.

This is exactly parallel with what has been found in the practical treatment of adult scurvy.

BORDER-LAND CASES.

We have already seen that there are many degrees of infantile scurvy, and that there are marked variations in the severity of different lesions according to age. It seems possible that, as there is a vanishing-point of rickets, there is also a vanishing-point of scurvy. In Cases B and C, p. 267, vol. ii., there was great tenderness of the lower limbs, with inability to stand, although *no swelling could be detected.*

When in a slight case of rickets the irritability and tenderness are *out of all proportion* to the obvious bone-changes, the modification of the food in an antiscorbutic sense will often prove immediately adequate to remove the above symptoms, and will suggest retrospectively a scorbutic factor.

We have considered orbital hemorrhage in connection with the typical cases. Mr. Holmes Spicer suggests that occasionally it may be a solitary sign of scurvy.

In like manner hæmaturia, which is a not uncommon accompaniment of the severe type of the disease, may occur without limb-symptoms, and respond immediately to the addition of living food to the dietary.

In the severe cases marasmus and profound anæmia with a muddy complexion are so marked that we are apt to regard them as essential. But in mild cases at the outset there is no marasmus, and the anæmia may be but slight.

Treatment.—The observations made on this disease accentuate all that has been hitherto written on the vital importance of breast-feeding in all ranks of society.

In the detailed reports of cases and in the notes of the writer there is no instance of an infant suffering from these symptoms who was at the time receiving milk from the human breast.

At the discussion on infantile scurvy at the Berlin International Medical Congress, Dr. Pott stated that he had seen one example of twin-children suffering from the disease, although breast-fed. But no other symptom beyond stomatitis is mentioned, and it is stated that suckling was impossible on account of the state of the mouth. Considering that, as we have shown, the true scorbutic gum-affection does not appear until after the teeth have appeared, it seems an open question whether these children were really suffering from scurvy. The case hardly invalidates our original contention.

In the *Deutsches Archiv für Klinische Medicin* for 1880, Dr. Kuhn gives a record of an epidemic of scurvy at Moringen. Thirteen cases of infants are recorded, several of them quite young and being suckled by mothers who at the time were scorbutic. With respect to the children's ailments, severe catarrh of the mouth, bronchitis, and some skin-affections (pemphigus, erythema, and in a few cases petechiæ) are mentioned. Several died, but there is no account of any autopsy.

The details given of the cases are too meagre to afford us any assistance.

With respect to the general directions as to breast-feeding, if the mother be herself scorbutic, or if her breast-milk be deficient in quality and amount, it is doubtless desirable that it should be supplemented.

But determined efforts should be made to improve the mother's nutrition, so that, if possible, the breast-feeding may be maintained as a partial mode of alimentation. When breast-feeding, either by a mother or by a wet-nurse, is unattainable, what form of artificial alimentation is to be recommended?

We have indicated in the foregoing section the general lines on which scurvy is to be met when it appears.

The administration of fresh, undiluted milk in adequate amount, in place of diluted, condensed, or peptonized milk, and the addition of sieved potato or other vegetable to the milk, are the most rapid and efficient methods. The separate use of small quantities of the juice of fresh fruit is an important adjunct, and less valuable is the addition of freshly expressed meat juice. It is important to remember that fresh, undiluted milk, if given in adequate amount, will meet the scorbutic need without any other addition. The late Dr. Patrick Black proved the antiscorbutic value of an abundant supply of fresh milk in the treatment of cases of adult sea-scurvy at the Dreadnought Hospital, and his observations never received the attention they deserved. But the sieved potato or other vegetable and the juice of fresh fruit will make the recovery more rapid, especially as in infant-feeding we are continually thwarted by the difficulty in the digestion of casein, and are therefore limited in the amount of milk which we may give.

Again, in the town supply of milk we have to encounter the grave risks of pathogenic organisms, and we are forced by this consideration to adopt some form of sterilization as a routine practice.

Experience seems to show, however, that prolonged sterilization and sterilization at high temperatures lessen the antiscorbutic power of milk. Sterilization or pasteurization at the lowest temperature and for the shortest duration consistent with the destruction of pathogenic organisms would seem to be desirable.

The use of sterilized milk which has been stored for a long period (and which has presumably been long sterilized at a high temperature) is certainly fraught with the risk of inducing scurvy. Peptonized milk, and humanized milk especially, when the latter has been sterilized and stored, are now among the commonest antecedents of scurvy in the children of well-to-do English people. Experience shows that infantile scurvy rarely begins very early. About the eighth month we should be on the alert for its appearance, especially if at that time there are evidences of rickets and anæmia, upon which scurvy is so readily grafted.

If at this period we see no chance of safely employing fresh, unboiled milk, we ought to begin to add a little unsieved potato, instead of having recourse to the proprietary food, and we ought to give some fresh fruit juice as a prophylactic. The fear of non-assimilation of starch during the second half of the infant's first year of life has acted as a needless deterrent in the employment of fresh vegetable material in the child's dietary.

LOCAL TREATMENT OF INFANTILE SCURVY.

The Gums.—Caustic applications, like nitrate of silver, are distinctly injurious.

The heroic measure of snipping off the granulations and then applying fuming nitric acid, which the writer has heard seriously proposed in a severe case, was based on a lack of appreciation of the true nature of the gum-affection, with incredulity as to the possible occurrence of scurvy in infancy. Under the dietetic treatment above described it is remarkable how soon the swelling and bleeding of the gums subside. If any local application is required, diluted orange juice, to which glycerin has been added, is the most suitable.

LOCAL TREATMENT OF LIMBS.

In Mr. Page's case and in a few others an exploratory incision was made down to the bone and a mass of blood-clot was removed from the periosteal sac. From the diagnostic side these explorations were invaluable, and when the nature of the case was ambiguous they were justifiable, but such a measure is unnecessary, and may be attended with some risk from hemorrhage. In one of the early recorded German cases (under the title of acute rickets) leeches were applied to the swollen limbs. The anæmia became aggravated, and new lesions appeared elsewhere. Thus, one of the few therapeutic suggestions concerning this disease in the German memoirs was that "antiphlogistic remedies were unsuitable." We have found it sometimes useful to apply wet compresses (wrung out nearly dry) firmly round the lower limbs. These can subsequently be surrounded by dry compresses or layers of cotton wadding. Splints are rarely needed, but small sand-bags placed alongside the limbs may be employed. Movement should be limited. During the active phase of the disease shampooing and friction should be strictly forbidden. Baths and douches should not be given; gentle sponging, for purposes of cleanliness only, should be applied in the horizontal posture. The clothing should be loose and simple, so that when the child is changed the least possible disturbance takes place. The child ought to be allowed to micturate and defecate in the horizontal posture.

A deep wooden tray comfortably padded is a very convenient apparatus. The child can lie quite flat on this and be moved to an invalid carriage from his bed without disturbance of limbs or body.

The nurse should be warned that spontaneous fractures may occur near the ends of the long bones and external to the junctions of the ribs with the costal cartilages. She must be made to realize this danger, and to understand likewise that the back-muscles are weak, and that in such a condition of extreme anæmia there is fear of syncope. She will then at length see the necessity of care about raising the child, and will avoid doing so except when imperatively necessary. There are few diseases in which knowledge of the anatomical conditions is so useful in dictating the line of treatment.

There is great benefit to be obtained by getting the child sunlight and fresh air, and the padded wooden tray is useful in that respect. When recovery has begun, the child begins to raise his back and afterwards to move his lower limbs.

There is very little risk in what the child does voluntarily in this way, but no attempt should be made to hasten matters by letting him stand or bear weight on his limbs. Fracture may occur during the convalescent stage, though not so commonly as in the active phase.

We have already referred to the striking fact that during the early treatment the child will take the antiscorbutic diet with avidity and digest it well. Subsequently, when the scorbutic need has been met, there may be inability to digest so large an amount of fresh milk or vegetables as at the beginning of treatment.

Nothing has been said about the use of drugs in the early, active stages of the disease. Iron, arsenic, phosphorus, etc., are useless if the proper diet is not ordered, and if the diet is placed on a right basis there is little need for drugs. It is strongly urged that at first exclusive attention should be given to securing immobility and to watching the effect of modifications of diet. During convalescence cod-liver oil and iron preparations may be given, and salt-water sponging may be carefully employed.

HISTORICAL RETROSPECT.

Under the title of acute rickets many cases were published in German literature from 1859 onward which presented the clinical type described in the foregoing pages. The most notable of these were given by Möller, in the *Königsberger Medicinische Jahrbuch*, 1859, Band i., Heft 3, S. 377, and in the same journal for 1862, Band iii., Heft 2, S. 135. Bohn, Förster, Hirschsprung, and Fürst recorded other cases, chiefly in the *Jahrbuch für Kinderheilkunde*. Stiebel, Senator, Steiner, and Baginsky in their several [illegible] this group of cases under the designation of acute febrile [illegible] of rickets. The possibility of scurvy was not [illegible] by the German writers. Some of them put it aside be[illegible] of sponginess of the gums. This we have discussed [illegible] sections.

[illegible] cases, in which sponginess of gums appeared and [illegible] him, he puts aside the scorbutic theory, because the [illegible] of antiscorbutics (of which he gives no detail) was not fol[illegible] immediate recovery. But his case was one of extensive multi[illegible] which had existed for a considerable time, and the cachexia [illegible] severe. None of these observers appear to have adopted the [illegible] during the early stage of the disorder. It is, however, very [illegible] read in the literature above mentioned the evidence given of [illegible] recovery when the spring returned and the sunshine came, quite [illegible] what we find in the old narratives of recovery of long-standing [illegible] and land-scurvy in adults. Moreover, the likelihood of the [illegible] of fresh milk and fresh vegetables to the diet of these cases inde[illegible] of the doctor's knowledge and prescription is not considered.

At length, in 1873, a Danish physician, Dr. Ingerslev, described one of [illegible] cases as infantile scurvy. In *Virchow's Jahresbericht*, 1873, S. 697, there is a brief summary of the case. The illness lasted six months, with "painful joint-affection" and spongy gums with carrion-like odor. "It terminated in spring, when the child received abundance of fresh garden cress." There was complete recovery.

Before 1882 the English medical literature bearing on the so-called acute rickets was very limited. There is a brief reference in Jenner's lectures to rickets commencing acutely, and West describes the condition of the gums associated with "odontitis infantum," which is obviously scurvy, though he does not recognize it as such.

In 1881 Gee narrated five cases under the title of osteal or periosteal cachexia. He maintained that the obscure symptoms referable to the bones and the cachexia could not be explained by rickets on the one hand or by congenital syphilis on the other. Marked attention was given to the curious deformity of the front of the chest, which has been described in the foregoing pages. Gee made no reference to the state of the gums. But in 1878 Cheadle had described three cases with definite sponginess of the gums, and also obscure symptoms referred to the lower limbs.

Cheadle claimed these cases on clinical grounds to be true scurvy, although in the absence of pathological evidence he was unable to state what the real nature of the limb-affection was. In 1879 and in 1882 Cheadle published further examples, and suggested that scurvy might readily be grafted on a rickety stock. These valuable papers laid down the lines of the true interpretation of the disease. The present writer believes that they are the foundation of the clinical knowledge of the subject, and that their doctrine remains unassailable.

The pathological evidence on this disease had been singularly meagre, with the exception of an imperfect and atypical case of Möller's, in which hemorrhagic pachymeningitis and orbital hemorrhage occurred.

As the outcome of three autopsies on typical severe cases the present writer was enabled to enunciate the anatomical substratum which seemed to explain the various symptoms observed during life. These results were published in the *Medico-Chirurgical Transactions* of London in 1883, vol. lxvi. p. 100. Besides the account of the autopsies, an analysis was given of thirty-one cases, of which eleven had been under the writer's care and twenty had been published elsewhere. The following propositions were enunciated:

1. The characteristic symptoms of the so-called acute rickets—viz., the special limb-affection and the cachexia, with or without sponginess of the gums—are not due to rickets, but are truly scorbutic.

2. The anatomical basis of the limb-affection is subperiosteal hemorrhage, and this hemorrhage probably accounts for some of the anæmia.

3. The disease may occur in rickety children, and perhaps in them more readily than in non-rickety children, but the amount of rickets present may be almost *nil*.

4. Although the disease tends spontaneously in many cases towards a slow but complete recovery, marked improvement often follows a vigorous and especially an early antiscorbutic treatment.

5. The treatment recommended is locally, during the acute stage, wet compresses and avoidance of movement; *internally*, the use of raw meat juice, fresh undiluted milk, and orange juice, or of some fresh vegetable, and from the first the access of as much fresh air as possible.

6. The use of the term acute rickets should be abolished for these cases, and that of infantile scurvy substituted, the special note of which, as distinguished from a dull scurvy, is the greater incidence of the disease on the bones.

7. In regard to the artificial feeding of infants, it seems probable that the so-called proprietary infant foods cannot be trusted as sole aliment for any lengthened period, however useful they may be as temporary adjuncts.

Since 1883 much confirmatory evidence has been recorded. Typical autopsies have been reported by Stephen Mackenzie, Colcott Fox, Cheadle, Sutherland, Wallis Ord, Northrup, Rehn, and others. In addition to the characteristic affections of the limbs and viscera, hemorrhagic effusions into the arachnoid cavity have been found in three cases by Sutherland, Wallis Ord, and the present writer. Important series of cases have been published with analyses by Rehn and Heubner.

Some of the most interesting material has been furnished from the United States of America. The first case observed there was in 1889, and was recorded by Northrup, but by February, 1894, no less than one hundred and six cases were reported to the New York Academy of Medicine by Starr, Rotch, Holt, and others. It seems probable that in England a

America the disease has been decidedly more common during the last two decades than previously, and that this corresponds with the greatly extended use of proprietary infant foods. It has till of late been more common in England among the children of the well-to-do than among the children of the poor, and Cheadle has pointed out that the children of the poor often receive at an earlier period some vegetable food—*e.g.*, potatoes, etc.—than do those of the well-to-do.

The anatomical substratum of subperiosteal hemorrhage has been generally accepted. The scorbutic factor has been widely, though not universally, accepted. Some writers—*e.g.*, Fürst and Ashby—consider that these cases are examples of rickets plus the hemorrhagic diathesis, and they prefer to designate the disease as hemorrhagic rickets. The present writer maintains in reply:

1. That rickets, which is usually present, may be extremely slight in amount, and, indeed, in some cases be incapable of clinical demonstration.

2. That the hemorrhage, anæmia, and cachexia respond to living food when administered early in this disease in a manner exactly parallel to what we find in adult scurvy.

3. That in no hemorrhagic disease other than scurvy is such rapid response to living food observed as we find in these cases.

The conclusions appended to the American Pediatric Society's Collective Investigation on Infantile Scurvy in North America (tenth annual meeting, June 2, 1898) are as follows:

"1. That the development of the disease follows in each case the prolonged employment of some diet unsuitable to the individual child, and that often a change of diet which at first thought would seem to be unsuitable may be followed by prompt recovery.

"2. That in spite of this fact regarding individual cases, the combined report of collected cases makes it probable that in these there were certain forms of diet which were particularly prone to be followed by the development of scurvy. First in point of numbers here are to be mentioned the various proprietary foods.

"3. In fine, that in general the cases reported seem to indicate that the farther a food is removed in character from the natural food of a child the more likely its use is to be followed by the development of scurvy."

A minority report was presented by Augustus Caillé in these words:

"1. From a study of this report and from due consideration of other known facts, scurvy appears to be a chronic ptomaine poisoning due to the absorption of toxins.

"2. It follows that the prolonged use of improper food and abnormal intestinal fermentation is a predisposing factor.

"3. Sterilizing, pasteurizing, or cooking of milk food is not *per se* responsible for the scurvy condition.

"4. A change of food and the administration of fruit juice and treatment of any underlying cause is the rational therapeutic procedure in scurvy."

SPORADIC CRETINISM (INFANTILE AND JUVENILE MYXŒDEMA).

By WILLIAM OSLER, M.D.

In vol. ii. Dr. Bury has discussed the whole subject of cretinism, and has dwelt with particular fulness on the morbid anatomy and general symptomatology. In the eight years which have elapsed since the appearance of his article three points of great interest have been added: (*a*) a knowledge of the frequency of sporadic cretinism; (*b*) its relationship to the thyroid gland; and (*c*), most important of all, a method of successful treatment.

I do not propose to deal with the question of endemic cretinism except in the discussion of its relations to the sporadic form. The recent literature to date is given in Ewald's article in *Nothnagel's Handbuch*, Band xxii. I shall take up only the incidence of the disease in America, the pathology, the relations to endemic cretinism, and the treatment.

I. INCIDENCE OF THE DISEASE IN AMERICA.

In 1893 I made a collective investigation on the subject of sporadic cretinism, and was able to find only eleven cases. Since that time the profession has learned to recognize the condition, and I have collected sixty cases, including those already referred to. Of these twenty-seven cases have been recorded; for the others I am indebted to various physicians throughout the country who have kindly responded to my inquiries, and in many cases sent photographs.

The clinical summary of the cases is as follows:

Sex.—Males, 24; females, 36.

Age.—Under two years, 6; from two to five years, 12; five to ten years, 12; ten to fifteen years, 10; fifteen to twenty years, 7; twenty to thirty years, 3; thirty to forty years, 4; over forty years, 4.

Nationality.—American, white, 12; colored, 1; Polish, 2; French, 1; German, 5; Swede, 1; Hebrew, 1; Norwegian, 1; Irish, 7; English, 1; Swiss, 2; Bohemian, 1; nationality not given, 23.

Locality.—There is no region in the country in which the disease is endemic, nor does it appear to be more prevalent in those districts, as in Michigan and parts of Ontario, where goitre is common.

Condition of the Thyroid Gland.—Goitre was present in 7; gland stated to be normal in 12; gland small in 2; gland not to be felt in 16; no note in 20.

II. THE PATHOLOGY OF SPORADIC CRETINISM.

There are three groups of cases, as noted by Dr. W. Rushton Parker:

(*a*) *With Absence of the Gland.*—The gland has not developed in fœtal life, or becomes completely wasted, so that at autopsy no trace of it is found. The child may be born a cretin, which is excessively rare. In a considerable number of the reports on sporadic cretinism the gland is stated to be absent, but it is almost impossible to judge by palpation if the gland is very small. In one of Hilton Fagge's cases the gland was thought to be absent, but post mortem there was a thyroid gland of some size, with a tumor.

Curling[1] first described the absence of the thyroid in cretinism in the two cases which he reported in 1850.

Bramwell,[2] in a review of the literature in 1892, found ten autopsies, in which in nine cases (in which the condition was noted) the gland was absent. Fletcher Beach states that of one hundred and sixteen cases collected from the literature there were sixteen autopsies, in fourteen of which the gland was absent, while in two goitre existed. In only one case, Dr. Friend's, in the series which I have collected, was absence of the gland noted post mortem.

(*b*) *With Atrophy of the Gland.*—This is a very important group, to which Fagge appears to have been the first to call attention. He gives the case of "a girl, who was stated by her relations to have been perfectly healthy until she was eight years old, when she fell ill with what was supposed to be a second attack of measles, and kept her bed for a fortnight. After her recovery her physical development underwent a remarkable change. Her features were previously well formed; they now acquired the cretinous configuration. Her hair, once black and abundant, became light-colored, dry, crisp, and very scanty. She ceased to grow; at the age of sixteen and three-quarters years she was only four feet one inch in height."[3] He suggests that the febrile illness led to atrophy of the organ, and that this was the cause of the supervention of the cretinous state. In Case II. in my first series the condition seemed to follow an attack of enteritis. Ashby and Wright[4] give the history of a case said to have been well until an attack of enteric fever at seven years of age.

It is interesting to compare these cases with the remarkable instance of operative myxœdema reported by Bruns to the Myxœdema Committee of the Clinical Society.[5] The patient at the time of operation was ten years old. Eighteen years after he had become "a dwarfy cretin." He had not grown since the removal of the thyroid. The expression was that of an idiot; there was characteristic myxœdema, with a mental apathy amounting almost to imbecility.

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[1] Transactions of the Royal Medical and Chirurgical Society, vol. xxxiii., 1850.

[2] Atlas of Clinical Medicine, vol. i.

[3] Fagge, Practice of Medicine, vol. i. p. 772.

[4] Diseases of Children, second edition, p. 479.

[5] Clinical Society Transactions, Supplement to vol. xxi.

The determination of atrophy of the gland during life is very uncertain, as pointed out by Fagge. No trace of the gland may be felt, and yet post mortem a well-marked, perhaps somewhat wasted, organ is found. In the recorded autopsies in sporadic cretinism atrophy has not, so far as I can gather, been found.

Through the kindness of the officers of the Indiana School for Feeble-Minded Children at Fort Wayne, I am enabled to report upon a fatal case in which there was extreme atrophy of the gland. The patient, Louise S. (Case VI. of the series), aged fourteen years, born in America, parents not related, no goitre in the family; nationality, German; height, 110.5 centimetres; circumference of head, 56 centimetres; from occiput to root of nose, 33 centimetres; from external meatus to external meatus, 26.7 centimetres; circumference of neck, 28 centimetres. The skin is loose and flabby, elastic and soft, very abundant. She is a deaf-mute, but appears intelligent. There is no curvature. The thorax is 57.3 centimetres; abdomen, 68.6 centimetres. The limbs seem a little enlarged about the epiphyses. There is no goitre. Dr. Delia Howe has sent a subsequent note that she had no treatment until September, 1895. She was then given the thyroid extract, three grains three times a day. She improved in many respects. The protruding abdomen disappeared, and she became very much brighter mentally. She became ill in March, and died of acute tuberculosis in November, 1896. At the time of her death she was seventeen years old, and her height was 127 centimetres. The thyroid gland weighed 4 grammes. The normal weight of the organ is from 15 to 20 grammes. I am indebted to Dr. Barker, the associate professor of anatomy in Johns Hopkins University, for a careful description of the condition of the gland, which is of special value, since, so far as I can learn, there has been no histological description of the thyroid in sporadic cretinism. The following is an abstract of his report. To the naked eye there was a very marked increase in the connective tissue separating the lobules, and with low power the acini were seen to be separated from one another. The individual acini were almost solid, except that here and there there were single cyst-like dilatations filled with colloid. High powers showed the most marked atrophy of the gland and certain remarkable transformations in the epithelium. A majority of the acini in the individual lobules possess very narrow lumina, which are often encroached upon by papillary projections, or partially filled with proliferated and desquamated cells. Cells lining the acini are so much altered that, not knowing the specimen, one would not recognize them as of the thyroid. The cell bodies vary a great deal in size, from small cells equal in dimension to those of the normal thyroid to large, flat structures, actual giant cells. The nuclei of the cells show marked alterations, many of them being huge, bladder-like nuclei. In some nuclei the chromatin is disposed peripherally.

The lumina of the acini vary much in size; some of the alveoli possess no lumina at all. In others only a very small central opening is to be seen. Extremely few contain any colloid. A majority are either entirely empty or show inside them only desquamated cells from the alveolar walls. A few cysts from three to six times the size of the normal alveoli are present, lined by flattened thyroid epithelium. Some of them have evidently been formed by the fusion of several acini. The colloid varies in its behavior to van Gieson's stain. In some of the few cysts present it is of a bright-yellow tint; in others it stains of a reddish brown, and is then more refractive. In the connective tissue one or two spaces filled with refractive colloid were found. These probably represent lymph-vessels, but there is no great amount of colloid inside lymph-spaces, nor is any colloid to be seen within the blood-vessels. Dr. Barker remarks that "the findings in this case are in the main confirmatory of those which have previously been made in endemic cretins. The condition is very similar to that which has been reported by de Coulon in endemic cretinism."[1]

(*c*) *Sporadic Cretinism with Goitre.*—Of the sixty cases collected in this country, seven had goitre. Herein lies a very striking difference between the sporadic and the endemic cretinism. In the latter the percentage of goitre in some statistics has been as high as sixty (Knapp). In Fletcher Beach's collected statistics of one hundred and sixteen sporadic cretins in different countries, the thyroid is noted as "not felt" in seventy-three, "felt" in eleven, and enlarged in seven cases. He states that of sixteen post-mortems of which he has been able to find an account, the thyroid gland was absent in fourteen cases, and there was bronchocele in two.

Figs. 1 and 2 show two cases of goitrous cretins, for the notes of which I am indebted to the description of Dr. Darey, of Northwood, Iowa, and of Dr. Kessel, of Cresco, Iowa. It is interesting to note that the father's sister had goitre, and a brother of these patients died after an operation for goitre.

III. THE RELATIONS OF SPORADIC TO ENDEMIC CRETINISM.

When the "cretinoid state," to use Gull's expression, whether developing spontaneously or following thyroidectomy, became recognized as a direct result of the loss of the function of the thyroid gland, it was a simple matter to suggest that true cretinism, both sporadic and endemic, had the same origin. The generic term cretinism may, indeed, be used to cover these four allied states, endemic, sporadic, idiopathic of adults (myxœdema), and operative, following total excision of the gland. One and the same pathological basis exists in the entire group,—viz., loss or perversion of the function of the thyroid; the anatomical basis is varied,—total absence, atrophy, or goitre. Kocher[1] in his masterly presentation of the subject takes this wide ground. Without exception, so far as I know, writers have assumed this position, but quite recently this relationship has been questioned by Bircher, a well-known student of endemic cretinism, in an excellent section on the thyroid gland in vol. i. of Lubarsch and Ostertag's "Ergebnisse," etc., 1896. He concludes "that the cretinoid degeneration is in no way connected with disturbance in the function of the thyroid gland." He bases this opinion upon the persistence of the thyroid, cystic and degenerated, it is true, in a large proportion of cases. In twenty cases fifteen had goitre, four had normal thyroid glands, and in one only it could not be felt. In three cases in which he had extirpated the thyroid in cretins and in the non-degenerated parts he had found normal thyroid tissue. Moreover, a cretin from whom he removed the cystic goitre became myxœdematous and was temporarily cured by the implanting of a gland; but, as Hanau and Ewald both remark, this is no evidence that the thyroid has nothing to do with cretinism. It would be very much the same as the development of uræmia in a case of chronic nephritis after bilateral nephrotomy.

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[1] Virchow's Archiv, February, 1897.

Both Curling and Fagge believed the conditions which they described to be identical with endemic cretinism. They further have the great merit of recognizing the loss of function of the thyroid as the probable essential factor in the disease. Curling's title is in itself suggestive, "two cases of absence of the thyroid body, and symmetrical swellings of the fat tissue of the neck, connected with defective cerebral development." In seeking an explanation he speaks of "the absence of those changes which result from the action of the thyroid, or on some imperfection in the assimilating processes consequent on the want of this gland; and the facts here detailed may not be without significance in directing the researches of future inquirers into the use of this body." Fagge held that the presence of the thyroid gland was "protective against the occurrence of cretinism." A similar opinion had been expressed about endemic cretinism in 1830 by Troxler (Kocher).

We may ask, in the first place, are there any essential differences between the sporadic and the endemic form of cretinism? A single definition covers both conditions,—*a chronic affection characterized by disturbance of the growth of the skeleton and soft parts, a remarkable retardation of development, an extraordinary disproportion between the different parts of the body, and a retention of the infantile state, with a corresponding lack of mental progress.* One has only to compare the picture given of cretins in Switzerland with those of the sporadic form both in England and in this country, to see that the two states, so far as external characters go, are identical. The differences between the two forms are as follows:

(1) The endemic variety develops under local conditions as yet unknown,

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[1] Zur Verhütung des Cretinismus und cretinoid Zustände nach neuen Forschungen, Zeitschrift für Chirurgie, Band xxxiv., 1892.

associated with a peculiar poison of doubtful nature. Bircher states tha "the cretinoid degeneration is a chronic infectious disease, the organic miasm of which is associated with certain marine deposits of the earth's surface and which gains access to the body through the drinking-water." It i only of late years that any light has been thrown upon the intimate relation of goitre and cretinism. I have already referred to the opinions of Troxler, Curling, and Fagge, and almost all writers on the endemic form agree with the statement of Morel, that goitre is the first step on the roa leading to cretinism. It is possible, however, that changes other than thos which lead to goitre may be effective; any condition associated with los of function of the gland.

(2) The differences in the changes in the bony skeleton. In the endemi cretin a premature ossification of the spheno-basilar bone has been described and the fontanelles close early. How far these are constant character remains to be demonstrated. In the sporadic form the fontanelles ofte remain open for a long period, even until after the tenth year, and there i a greater retardation in the development of the long bones.

(3) The endemic cretin is said not to show the same myxœdematou characters as the sporadic cretin, but the accounts vary in different authors and the description of the cutaneous condition given by Kocher (whose experience with cretinism has been very large) fits that of the sporadic cretin exactly. Other minor differences are mentioned, such as the shorter life of th sporadic cretin. Ewald,[1] indeed, states that he knows of no instance in whic life has been prolonged beyond the thirtieth year, but of the cases which w have collected there were seven beyond the thirtieth year. Another poin is the less frequent presence of goitre. The percentage of goitre in endemi cretins has been noted as high as sixty. In the Sardinian Commissio there were three thousand nine hundred and twelve instances of goitre i five thousand nine hundred and twenty-three cases. In the collected serie of sixty cases there were only seven with goitre. The two cases wit goitre (see Figs. 1 and 2) are good examples of goitrous cretins occurrin in a family in which goitre was prevalent.

(4) Bircher states that the thyroid extract has no influence upon th endemic cretin, and this he claims illustrates the independence of loss of thyroid function; but I cannot gather that he, or, indeed, anybody else, ha systematically tried the thyroid treatment upon young cases. There is som positive evidence. Kraepelin[2] (who holds that in endemic cretinism diseas of the thyroid is the first link in the chain, and that the changes in th skin, retardation of growth, and dementia result directly from loss of function of the thyroid) speaks most hopefully of arresting the disease by thyroid feeding if begun early. He adds that even in long-standing case it has been possible by the thyreodin to reduce wholly the swelling of th

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[1] Nothnagel's Specielle Pathologie und Therapie, Bd. xxii.,—the best recent accoun of cretinism and allied states.

[2] Psychiatrie, fifth edition, 1896.

skin, and he has seen the menstrual function restored. The psychical features were not much improved.

Professor Gaule, of Zurich, writes me that he is informed by Kocher that all the cases that are brought to the hospital are treated with the thyroid extract.

Professor Sahli, of Berne, writes, "So far as I know, the observations on operative myxœdema have been confirmed by the thyroid treatment of young cretins." The question is one deserving of the most careful study in the goitre and cretin districts of Europe.

The minor differences between endemic and sporadic cretinism, many of which are still doubtful, cannot for a moment be set against the similarity of the two conditions in almost all points save the local (telluric) influences in the causation of the former, and there remains only the question of the state of the thyroid gland. It is remarkable that in endemic cretinism the observations should be so scanty. Hanau[1] reports three cases, in all of which the thyroid glands showed alterations, being smaller, and in one there was a cyst. The connective tissue was abundant, the alveoli smaller, and in only a few was there the normal colloid. Langhans[2] in one case found the gland enlarged and made up of large alveoli, many of which were empty and only a few filled with refractive colloid. De Coulon,[3] working in Berne, has examined the thyroid gland in four cretins, and in one cretin of bodily aspect but of good mental development, and, after a most thorough histological study, concludes "that the thyroid had not completely degenerated, but the tissue showed changes which indicated that its function was impaired, or, at least, reduced to a very low 'ebb.' The smallness of the alveoli, their disappearance in consequence of the relative increase in the connective tissue, the absence of colloid in the majority of the alveoli and in almost all the lymph vessels, so also the condition of the epithelium and the nuclei, . . . speak in favor of this view." Also the condition of the existing colloid and its reactions suggested, he thought, important chemical changes of a degenerative character.

In the atrophied gland of Case VI. the lesions were identical with those found in one of de Coulon's cases, which still further strengthens the position of those who hold the essential unity of the process. In neither form is the state of the thyroid gland always the same; loss or serious perversion of function is the important factor, and this may follow absence, atrophy, or hypertrophy. There is no fixed stamp or type of cretin; the range is from a mere mass of humanity, without a ray of intelligence, to the high-grade cretin, with but slight bodily and still slighter mental changes; and it is only reasonable to believe that to these grades are correlated varying degrees of thyroid degeneration. Bircher lays great stress upon the presence of normal-looking tissue in the glands which he excised from cretins, but gives no statement of any histological study; nor is the development of myxœdema in one of these cases and of tetany in another inconsistent with the view that the thyroid is the seat of the essential lesion, since the activity of the gland may have been sufficient only to maintain the organism at a certain plane of cretinoid existence.

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[1] Transactions of the Berlin International Congress.

[2] Virchow's Archiv, Bd. cxxviii.

[3] Ibid., Bd. cxlvii., 1, January, 1897.

If the experimental work on the importance of the para-thyroids should be confirmed, the whole question will have to be reviewed from this standpoint.

There are, of course, gaps in our knowledge, but the evidence at present available warrants, I believe, the conclusion that the changes characteristic of cretinism, endemic as well as sporadic, result from *loss of function of the thyroid gland.*

IV. DIAGNOSIS.

The number of cases which I have been able to collect indicates that in this country the profession has rapidly learned to recognize sporadic cretinism. It is also evident from statements and from photographs which I have received that there are a number of conditions which are apt to be mistaken for it, and that even men in institution life have not very clear ideas upon the subject. There are several points to which I may refer.

(*a*) *The recognition of early slight cases.* After the third year the condition is, as a rule, diagnosed at sight, and advanced types offer not the slightest difficulty. The earlier the recognition is made the greater is the prospect of complete recovery. About the eighteenth month the subject may present the aspect of myxœdema rather than of cretinism, and the swollen, waxy skin even suggests Bright's disease. Case I. of the series was supposed to have chronic nephritis. The failure of development, the inability to talk or to walk, and the retarded dentition begin to attract attention as the child reaches this period. The absence of expression, the open mouth, the large tongue, and the drooling suggest that something is wrong. The development may be so slow that a child of three years looks not older than fifteen months. There are possibly cases due to thyroid insufficiency in young infants, which correspond to the *myxœdème fruste* of the French, which may be readily overlooked. To this several writers have recently called attention. Dr. Bury,[1] in the discussion last year at the British Medical Association, states that he saw a baby a year old which had ceased to "get on." It became flabby, fat, lost its vivacity, and began to show a protuberant abdomen, a lax skin, and other cretinoid appearances. Treatment with small doses of the thyroid, one-fourth tabloid daily, speedily picked him up; he grew, became lively, and at the end of six months treatment was discontinued without retrogression. Such cases are probably due to transient, perhaps functional, disturbance of the thyroid. Herrick[2] also in his recent paper refers to the ease with which these milder cases may at first be overlooked.

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[1] British Medical Journal, 1896, ii. p. 621.

[2] Archives of Pediatrics, April, 1897.

(*b*) *The diagnosis from other types of idiocy.* Naturally enough, it has been suggested that diminished or perverted function of the thyroid gland might be responsible for the mental and bodily defects in ordinary idiocy, and more particularly in dwarfs. The question is one deserving of careful study, particularly by those who have opportunities for clinical and post-mortem observation in the large institutions.

The Mongol type of idiot resembles the cretin more closely than any other. Telford-Smith, in speaking of this form, says, "Idiots belonging to the so-called Mongol type are those who most nearly resemble the cretin, both in physical aspect and in mental character. In idiots of this type we get the stunted growth, the dull, heavy expression, with open mouth and thick lips; the slow, deliberate movement, and hoarse, guttural, and monosyllabic speech; the mental apathy and lack of spontaneity; the sluggish circulation and sensitiveness to cold. A thickened condition of subcutaneous tissue is often found, with dulled cutaneous sensibility. The skin is coarse and dry, the hair short and thin. First and second dentition are delayed. As far as palpation enables one to judge, the thyroid gland is subnormal in size. Pseudo-lipomata I have not found." He has tried the effect of thyroid extract with some benefit, but there is not the same remarkable change as in the cretins. I cannot altogether concur with Dr. Telford-Smith's statement as to the slow, deliberate movements and mental apathy of Mongolian idiots. It was a form in which Dr. Kerlin, of Elwyn, was particularly interested, and with him I had many opportunities of seeing cases. They rather impressed me as vivacious, often very sprightly and mischievous. In no instance was there any condition of the subcutaneous tissue suggestive of myxœdema.

Deaf-mutism is a not infrequent accompaniment of sporadic cretinism. In the endemic form it is still more common.

The various forms of idiocy dependent upon faulty development of the hemispheres in fœtal life, the hydrocephalic and the microcephalic idiots, and the forms of imbecility associated with the cerebral palsies of children are readily distinguished.

(*c*) *The condition known as fœtal rickets*—achondroplasia, or the chondrodystrophia fœtalis—is liable to be mistaken for cretinism.

The relation of this remarkable condition to cretinism is very carefully discussed by Kaufmann,[1] and more recently by Bircher,[2] to whose papers the reader is referred. John Thomson, in the *Edinburgh Medical Journal* for 1893, gives excellent illustrations of the adult form.[3] The thyroid is not usually involved, though it has been found absent in a fœtus which presented this condition (Bowlby).[4] The intelligence is not specially disturbed, the facial and cranial characters are not those of cretinism, and myxœdema is not present. The most characteristic feature is the dwarfing, with remarkable shortness of the limbs (micromelia), owing to disturbance of the growth of the shafts of the long bones, and with, in most cases, enormous enlargement of the articulations due to a hyperplasia of the cartilaginous ends of the bones. Bircher concludes that the condition is quite independent of the state of the thyroid gland. He is in error, however, when he states that the cases of sporadic cretinism described by Curling and Fagge belong to this group.

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[1] Untersuchungen ueber die sogenannte Fœtale Rachitis (Chondrodystrophia Fœtalis), Berlin, 1892.

[2] Lubarsch und Ostertag, Ergebnisse, Abt. i., 1896.

[3] They are reproduced in Gould and Pyle's Anomalies, etc.

[4] Pathological Society Transactions, 1884.

(*d*) And, lastly, the condition of infantilism may be briefly spoken of as in some instances dependent upon disturbed function of the thyroid, and there may be a possibility of confounding the cases with slight grades of cretinism. Infantilism is "a morphological syndrome characterized by the preservation in the adult of the exterior form of infancy with the non-appearance of the secondary sexual characters."

Occasionally the subjects of infantilism display opposite sexual characteristics,—feminism not only in bodily conformations, but in mental attributes. Apart from hereditary syphilis, in which the condition is not uncommon,[1] infantilism seems rare in this country. It is occasionally seen combined with great obesity. More frequently it is an accompaniment of mental defects in imbeciles and idiots. The onset of puberty, with the development of the secondary sexual characters, is delayed for years after the normal age. The sporadic cretin often presents the characters of infantilism even when above thirty (see photograph of Dr. Sinkler's case), but there are rare instances of infantilism, properly so called, complicated with myxœdematous features, due to loss of function of the thyroid, and such cases might be relieved by appropriate treatment.

V. THE RESULTS OF THYROID TREATMENT.

No type of human transformation is more distressing to look at than an aggravated case of cretinism. It recalls Milton's description of the Shape at the Gates,—

> "If shape it might be called that shape had none
> Distinguishable in member, joint, or limb,"

or the hideous transformations of the fairy prince into some hideous monster. The stunted stature, the semi-bestial aspect, the blubber lips, the retroussé nose, sunken at the root, the wide-open mouth, the lolling tongue, the small eyes, half closed, with swollen lids, the stolid, expressionless face, the squat figure, the muddy, dry skin, combine to make the picture of what has been well termed the "pariah of nature."

Not the magic wand of Prospero nor the brave kiss of the daughter of Hippocrates ever effected such a change as that which we are now enabled

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[1] See Fournier's excellent description in Les Affections Parasyphilitiques, 1894.

Fig. 1*a*.

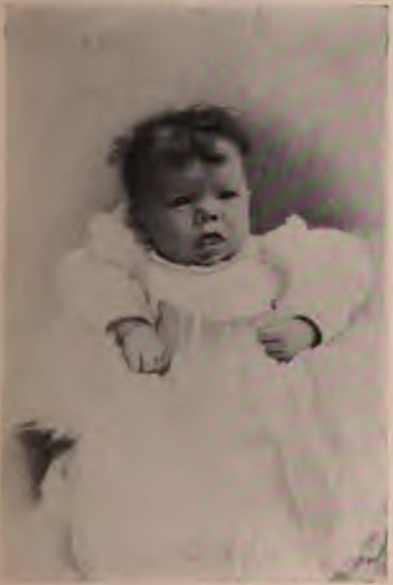
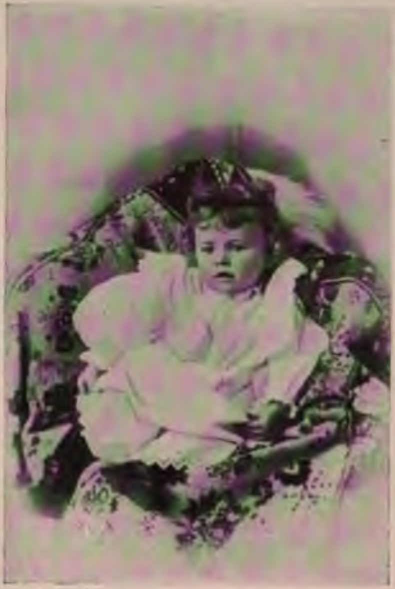


Fig. 1*b*.



Female, aged fifteen months. (Dr. Elsner.)

Fig 2*a*.

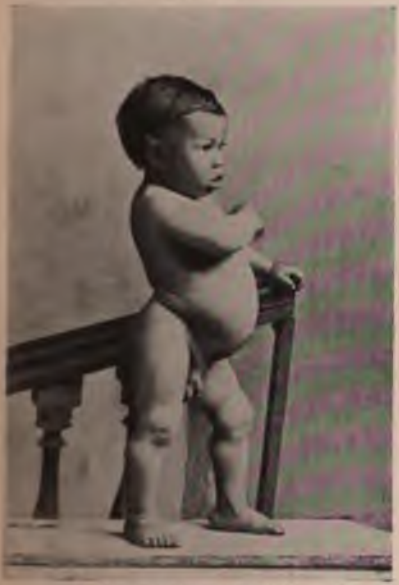
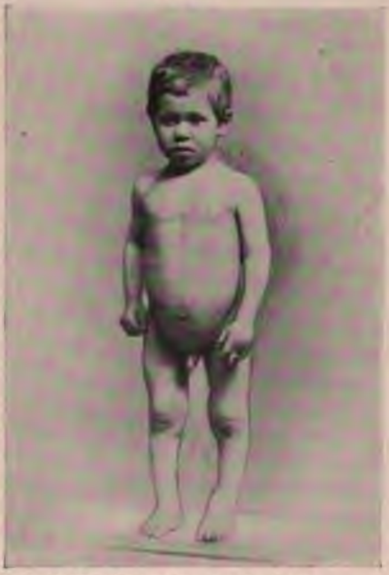


Fig. 2*b*.



Male, aged six. (Dr. Vinke.) Fig 2*b* shows the remarkable change five months later.

Fig. 3*a*.

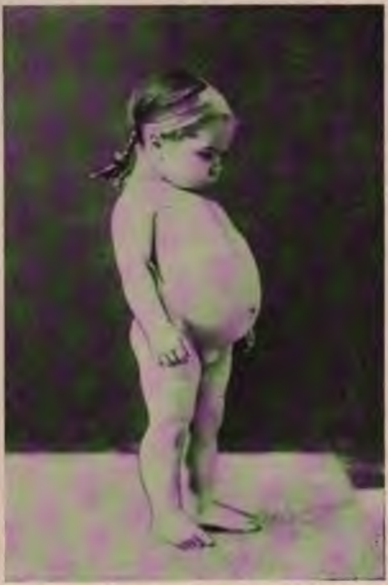
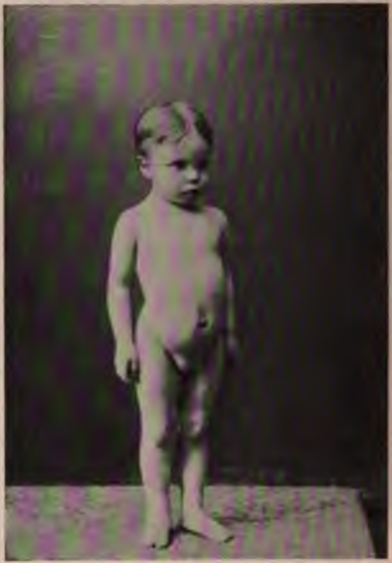


Fig. 3*b*.



Female, aged nine. (Dr. Dickson L. Moore.) Fig. 3*b* shows the remarkable change after seven months' treatment.

Fig. 4*a*.

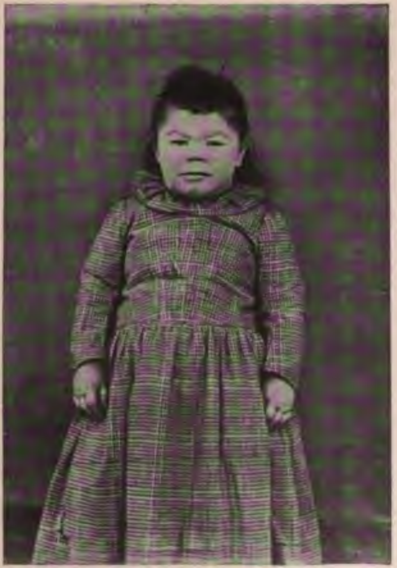
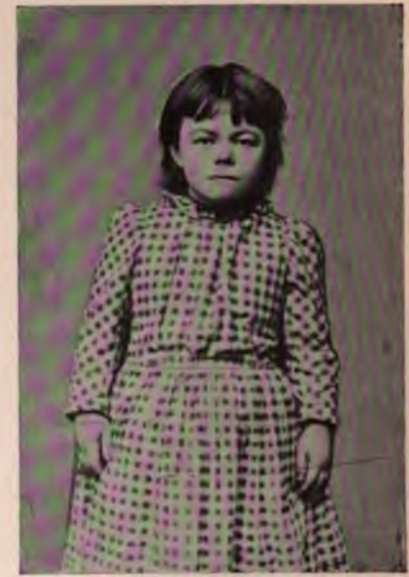


Fig. 4*b*.



Martha L. Y., aged sixteen. (Dr. J. C. Carson.) Fig. 4*a* shows the patient one year before and Fig. 4*b* one year after the administration of thyroid extract.

Fig. 5*a*.

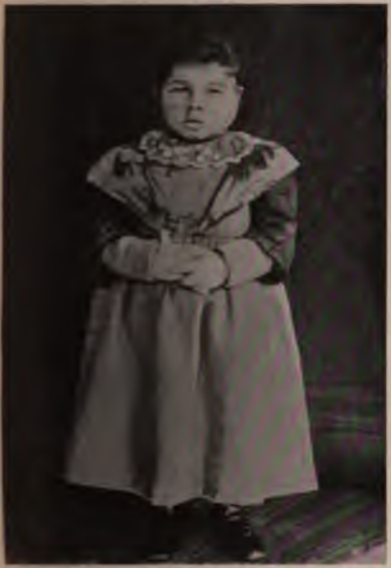
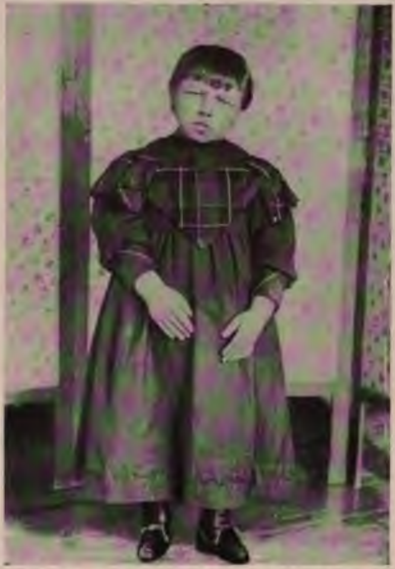


Fig. 5*b*.



Female, aged thirty. (Dr. Wharton Sinkler.) Fig. 5*b* shows the change in appearance at the end of two years' treatment.

Fig. 6*a*.

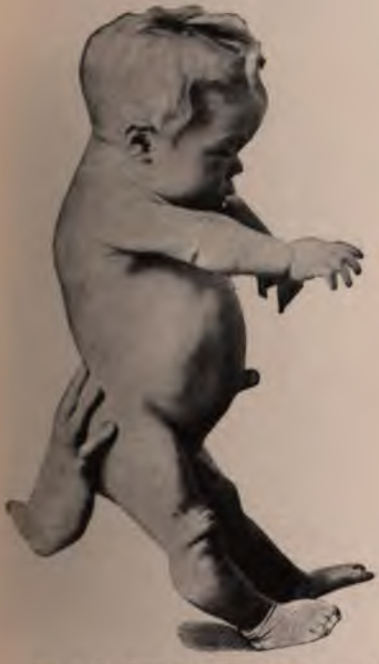
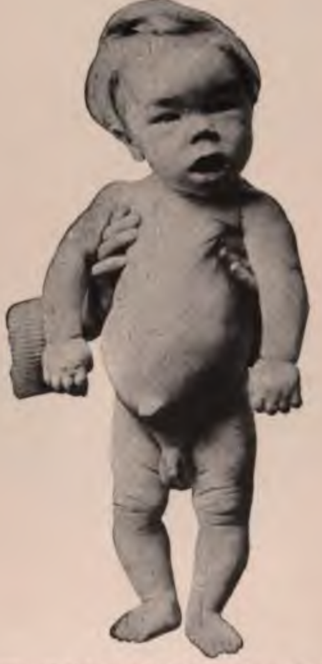


Fig. 6*b*.



(Dr. J. W. Coyner.) Figs. 6*a* to 6*f* show a perfectly phenomenal change which took place in this child in eleven months' treatment. I know of no set of photographs, among all that have been published on the subject in late years, which shows such an extraordinary transformation in so short a time.

FIG. 6c.

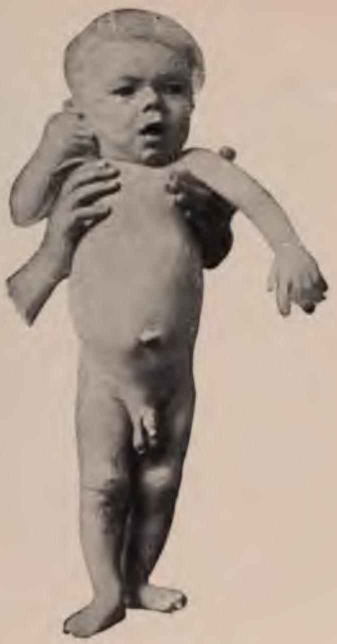


FIG. 6d.

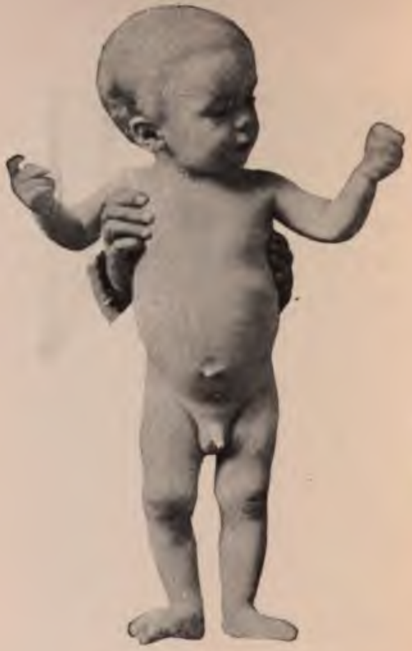


FIG. 6f.

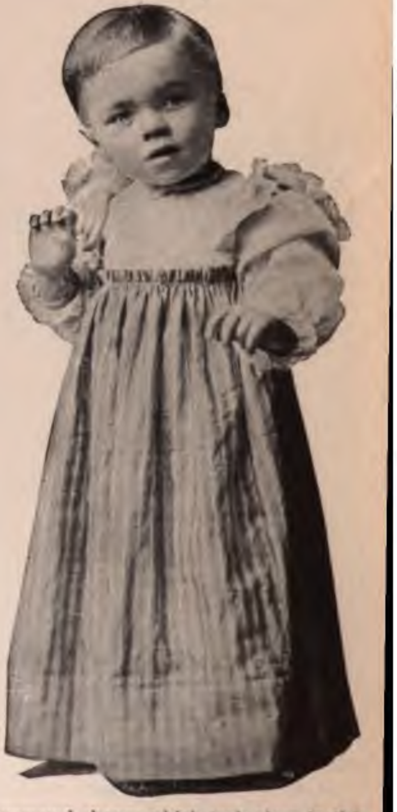
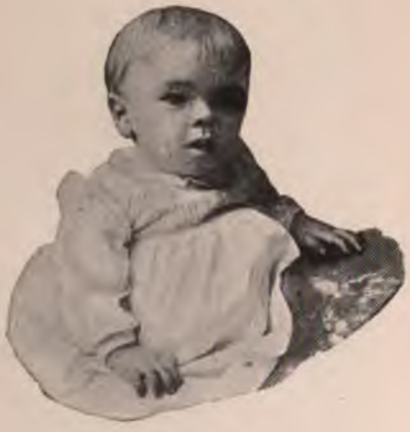


FIG. 6e.



(Dr. J. W. Coyner.) Figs. 6*a* to 6*f* show a perfectly phenomenal change which took place in this child in eleven months' treatment. I know of no set of photographs, among all that have been published on the subject in late years, which shows such an extraordinary transformation in so short a time.

to make in these unfortunate victims, doomed heretofore to live in hopeless imbecility, an unspeakable affliction to their parents and to their relatives. From a large number of photographs which I have received, I have selected a set to illustrate the effect of treatment at different ages, from infancy to the thirtieth year, and illustrating also the influence at periods varying from two years to a year. The series has an educational value, as the pictures tell their own story, not only enabling the practitioner to recognize the victims of this affection, but also emphasizing, as words cannot, the magical transformation which follows treatment.

Fig. 1 *a* shows a patient of Dr. Elsner, of Syracuse, New York. The child was eighteen months old at the beginning of treatment, and the photograph shows a very characteristic state of infantile myxœdema. Fig. 1 *b* shows the state thirteen months after treatment.

Fig. 2 *a* illustrates the case of Dr. Vincke, of St. Charles, Missouri, a boy aged six years. Fig. 2 *b* shows the condition five months after treatment. In a year and a half he grew nine inches.

Fig. 3 *a* shows a patient of Dr. Dickson L. Moore, of Columbus, Ohio, a girl aged nine years. The treatment was begun August 12, 1896. Fig. 3 *b* shows the condition seven months later, March 20, 1897. The child had gained four inches in height, and the entire appearance had changed remarkably.

Fig. 4 presents a typical picture of a sporadic cretin, aged seventeen years, under the care of Dr. J. C. Carson, of Syracuse, New York. Fig. 4 *a* was taken a year before treatment, and Fig. 4 *b* illustrates the condition a year after.

Fig. 5 *a* shows a sporadic cretin at the age of thirty years, a patient of Dr. Sinkler. The height was one hundred and twelve and three-quarters centimetres. Fig. 5 *b* shows the condition a year after treatment. She had grown nearly seven centimetres, and had lost much of the myxœdematous characters. This case is of special interest, as showing the importance of the treatment even in adults.

I know of no single set of photographs which show in quite the same way the phenomenal change as shown in this series of pictures, very kindly sent by Dr. Coyner, of Peoria, Illinois.

Figs. 6 *a* and 6 *b* show the very characteristic appearance of a sporadic cretin, aged twenty-three months: length, twenty-eight inches; circumference of the abdomen, nineteen inches. Fig. 6 *c* shows the change after three months' treatment; the abdomen measured sixteen inches. Fig. 6 *d* illustrates the condition after five and a half months' treatment: height, thirty inches; abdomen measured fifteen inches. Fig. 6 *e* shows the change after seven and a half months' treatment; while the last picture, Fig. 6 *f*, shows, eleven months after beginning the use of the thyroid, a perfectly natural-looking child.

1. *The Character of the Changes.*—(*a*) *Bodily.*—Loss in weight, due to disappearance of the myxœdematous condition and of the fat, is noticed

within a month or six weeks after the commencement of the treatment. The face becomes thinner, the palpebral orifices wider, the puffiness disappears from about the eyes, the flabby supra-clavicular folds melt away, the projecting abdomen diminishes in girth, and the child's figure becomes more shapely. Several of the photographs illustrate this in an interesting manner. This change is much more striking in young children of from three to six or eight years, but it is also well seen in the older patients. Nothing could be more remarkable than the change in the features in Dr. Carson's case; and even in Dr. Sinkler's case, aged thirty, the change as shown in the photograph is most evident. The expression of the face is altered by the recession of the tongue, and in many instances the drooling ceases, as the mouth is kept closed. This relieves in great part the idiotic expression.

Among the constructive and progressive alterations may be mentioned the loss of the waxy pallor of the skin, which becomes softer and much more natural looking. The hair, too, changes and becomes more abundant and finer. Several writers have referred very particularly to this remarkable change in the skin and hair, as though there had been a complete substitution of the old by a new skin and hair. In very young children teething proceeds rapidly; in older subjects, if the second dentition has not begun, the milk-teeth are shed and the permanent ones develop rapidly.

No change is so remarkable as the increase in stature. As Dr. John Thomson remarks, "The natural impulses of growth, which were in abeyance in the thyroidless condition, are let loose." In my first case the little girl grew four inches in a year. Among the most remarkable in the collected series are the following: Dr. Friend's case gained eleven and one-quarter inches in one year and ten months, Dr. Vincke's case gained nine inches in one year and seven months, Dr. Noyes's case gained eight inches in five and a half months, and Dr. Edwin F. Wilson's case gained seven inches in six months.

Fig. 3 illustrates what an extraordinary alteration takes place after seven months' treatment. The loss of the squatness of figure, the apparent disproportion between the head and the trunk, the loss of the characteristic attitude, and the disappearance of the lordosis are well illustrated. It is to be remembered that the rapidity in growth in some cases has led to increase of a lateral curvature, and even to marked bowing of the legs.

(*b*) *Mental Change.*—Even within a couple of months the alteration in the mental condition is noticed. At any rate, the patients look much brighter and the face is not absolutely expressionless. As a rule, the younger the case the more marked is the mental change. Young cretins who have not learned to speak a word soon begin to talk in their play. In children between six and ten the effects are even more remarkable, and with the loss of the myxœdematous condition there is a corresponding awakening of the mental faculties. In older patients the treatment is not so efficacious. In Case II. of my series the girl, aged nineteen, did not seem to be very much benefited, although it is true the treatment was abandoned by the mother after a short time. In other instances, as in Dr. Sinkler's case, the mental condition improved very much, even though the patient was over thirty. I know of no instance in which the treatment has benefited the condition of deaf-mutism.

2. *The Dosage.*—I have usually begun with a grain of the desiccated gland three times a day in young cretins. It should be carefully watched, and the amount reduced if the pulse becomes more rapid, or if there is fever. Older patients may take as much as five grains in the day, and the amount may be increased or diminished as the symptoms indicate. The cases bear the remedy very well; and in a month, if no improvement is noted, larger doses must be tried. Unpleasant effects are less commonly seen than in the myxœdema of adults. The glycerin extract may also be employed. The thyroidin of Baumann is now in the market, and may be used in doses of five minims. It is very fortunate that the active principle of the gland seems effective in all the preparations.

3. *The Question of Continuance of the Treatment.*—After the disappearance of the myxœdema and the establishment of the processes of growth and development, a very moderate dose seems sufficient, one or two five-grain tablets a week. Intermission for a month or six weeks does not seem to be followed by any striking change, but an intermission for a longer time is followed by symptoms indicating a relapse. In my first case the treatment was interrupted for two months last year, and the child became very languid and apathetic, but improved at once when the use of the extract was renewed.

LITHURIA IN INFANCY AND CHILDHOOD.

BY D. D. STEWART, M.D.

IN his interesting article on the uric acid condition in vol. ii. of this work, Dr. Fothergill adopted as a text for his remarks the following quotation from Prout: "Children in general, and particularly children of dyspeptic and gouty individuals, or who inherit a tendency to urinary affections, are exceedingly liable to crystallized lithic deposits from the urine."

Fothergill did not distinguish the quite accidental physical and the common physiological causes which might lead to uric acid gravel, and regarded the occurrence of this condition as indicating a type of reversion to a primitive formation.

Holding this view,[1] Fothergill looked upon the occurrence of lithuria as an indication of a *minus* quantity, as something taken from a healthy child: "The increase of the uric acid formation is the measure of its shortcomings,—its failure to attain to the normal urea formation." Hence he thought it might be expected rather in the enfeebled than in the robust child, and in the children of the gouty and those who are strumous. These were such as showed a distinct tendency to physical deterioration. In consequence, he believed that town-bred children, in whom education of the mental faculties is apt to be conducted at the expense of the physical well-being, and as a result of which enfeeblement of the assimilating organs and stature may easily occur, have a special tendency to retain the early uric acid formation; so that "town products are to a certain extent an inferior race to their country cousins," who outgrow this condition.

Reasoning in this line, Fothergill further held that not only the gouty and scrofulous, but brain-toilers and their children, the neurotics, the migrainous, the lithogenic, and those who take little exercise and eat largely of nitrogenous food, tend to acquire uric acid gravel.

Much light has been thrown on the chemistry and physiology of the uric acid condition by recent investigations. It is this which has cast grave doubts on the practical value of the deductions of Fothergill and others in the direction indicated.

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[1] And by lithuria he merely meant the presence in the freshly voided urine of precipitated urates, or free uric acid, irrespective of the amount excreted, taking wrongly the visible presence always to mean excess.

Unfortunately for the value of these assumptions and much of the therapy built thereupon, they were drawn from theories based on premises now known to be largely incorrect. In the first place, it must be stated that although there is a good deal to be said in favor of the view, not that the uric acid condition is an indication of reversion, but that it is, in the language of Sir William Roberts, a memory of an ancestral condition, yet its importance, other than its being a factor in gout, and often also the cause of renal derangements and disease, has been immensely overrated. In gout and in gravel its action, as will be remarked further on, is strictly a mechanical one,—that of a local irritant,—there existing no valid evidence that it has any toxic qualities whatever. It certainly can no longer be viewed as a toxic agent, and since its mode of formation is, as will be shown, different from what was originally supposed, and since its excess in the urine of infants and children can be usually accounted for by other conditions than that of a diathesis, and since its presence, even in children, as a precipitate in the urinary passages may be accounted for on purely physical grounds, through changes in the urinary secretion other than those which may accompany a diathesis, and since, moreover, uric acid is a constant constituent of the urine, as are urea and the chlorides,—although in amount special to each individual, but never merely in traces, as Fothergill held,—it follows that a great deal that has been written about the uric acid condition, lithuria, and the like, is misleading in its nature and pernicious in its tendency.

The investigations of Horbaczewski, Kossel, Levison, and others tend to show that the commonly accepted notion that uric acid is formed in the body as a result of the direct transformation of food proteids is incorrect, as is that still strenuously held notion that its transformation in the organism into urea by a process of oxidation occurs to any extent in health, and that a partial lack of such transformation is common in the conditions before enumerated.

Apparently the gouty condition—that in which uric acid is supposed to be in excess in the body—is not necessarily dependent upon a too highly nitrogenous diet, and especially on an animal one. For, as is pointed out by Luff ("Gulstonian Lectures on the Chemistry and Pathology of Gout"), gout is not infrequently encountered in poorly nourished individuals, while birds fed entirely on grain develop uric acid concretions about the joints. On the other hand, as Luff also remarks, uric acid is absent from the urine of certain of the carnivora. To indicate that the production of uric acid is not dependent upon diet alone, Luff calls attention to the fact that the same diet in one class of animals (the carnivorous python and boa) will produce uric acid, and in another class (the carnivorous lion and tiger) will produce urea. "Graminivorous birds excrete uric acid and no urea, while herbivorous animals excrete quantities of urea and little or no uric acid."

Horbaczewski has shown pretty conclusively that uric acid, instead of resulting directly from the digestion and transformation of the albuminoids of the food, as does urea, is in reality a product of the decomposition of the nuclein basis of the cells of the body, chiefly the leucocytes, and hence only indirectly related in origin to the ingesta. Foods rich in nitrogenous elements, whether animal or vegetable, through their effect in promoting a digestive leucocytosis, tend to promote uric acid formation merely through the decomposition of the nuclein derived from the nucleo-albumin contained in the nuclei of the leucocytes. This being so, in a dietary free from uric-acid-forming constituents, such as milk,[1] as has been ascertained, uric acid excretion normally is continued in very definite, although much lessened, amount,[2] its source being the nuclei of the cells of the body, and especially the leucocytes, the constant breaking up of which normally furnishes an average of about 0.3 to 0.6 gramme of uric acid daily.[3] It has been ascertained that medicinal substances, such as pilocarpine, phosphorus, antipyrin, salicylic acid, and many other drugs, and especially nuclein-containing substances, such as thymus gland, extract of spleen and bone-marrow, and so on, similarly promote the formation of uric acid. The leucocytosis so induced is succeeded by an increased destruction of the nuclein-containers, with liberation of alloxuric- or uric-acid-yielding substances.

It seems now a well-authenticated fact that normally the circulating blood of neither man nor mammals, nor, probably, of fowls, contains uric acid, either free or combined. The experiments of Garrod, Jaksch, Klemperer, Luff, and others have established this. This fact at first seems difficult of realization from what is known concerning the probable origin of uric acid from nuclein. The explanation apparently is that, although Horbaczewski has demonstrated the fact of the formation of alloxuric bodies, such as the xanthin basis and uric acid, through the agency of the disintegration of nuclein in the organism, it is probable that, normally at least, uric acid is formed in the body, independently of the kidney, only in traces, if at all.[4] Its continued and invariable absence from the blood in health certainly tends to indicate this. In those in whom no considerable decomposition of nuclein occurs there are formed the alloxuric and xanthin bases, from certain of which, in process of excretion by the kidneys, the uric acid is probably derived. According to this view, which is supported by what appears overwhelming evidence, some of these substances may be looked upon as antecedents of uric acid. It is now accepted by the best authorities that, contrary to the old view still urged by Haig, uric acid is in no way harmful, and that in gout it is only through its mechanically irritating qualities that it produces symptoms. On the other hand, certain of the allied nitrogenous waste products, the xanthin derivatives, apparently possess toxic properties, so that our therapy must be directed towards the avoidance of the production of these and to aid their rapid excretion when formed. For it is to these, in conditions other than those described by Jaksch, which are falsely termed "uricacidæmia," that many of the effects erroneously attributed to uric acid are probably due.

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[1] When the excretion of uric acid scarcely exceeds that occurring in the fasting condition.

[2] No evidence whatever exists for the extraordinary statement of Haig, so generally received, that uric acid in health may be readily retained in the system, as in the liver and spleen, its excretion or retention being governed by the reaction of the blood.

[3] See G. Klemperer, Berliner klinische Wochenschrift, 1896, No. 33.

[4] Horbaczewski showed that uric acid could be generated by decomposition of the nuclein contained in the leucocytes of the spleen and in other organs and tissues of the body, and that any nuclein-containing tissue could similarly be made to yield xanthin or uric acid bases. By mixing spleen pulp with blood and maintaining the mixture for some time at blood-heat, he obtained as a result an abundant yield of xanthin bases. Carrying out the same process in the presence of an abundant supply of oxygen, he produced uric acid.

A great deal was made by Fothergill of the fact that uric acid infarcts are not uncommonly found in the kidneys of infants a few days after birth. But this and the occurrence in infants and in young children of gravel, when actually due to excessive secretion of uric acid, can now be explained more rationally by the fact that in the young there is naturally a tendency to a comparatively high uric acid output. This is due in them to a more rapid birth and growth and subsequent decay of newly formed cells, with a resulting increase in the leucocytes or nuclein yielders, and not to any inheritance of a tendency to a reptilian uric acid condition. In early life, as is well known, the proportion of leucocytes to erythrocytes is, normally, considerably greater than in adult life. Schiff, quoted by Cabot, records a case of a healthy infant whose blood an hour after birth showed nineteen thousand five hundred leucocytes per cubic millimetre. After its first meal the count was twenty-seven thousand six hundred and thirty-five, and after its fourth feeding thirty-six thousand. From the third day the leucocytosis, especially the increase resulting from food, underwent a diminution.[1] There is every probability in favor of the view that in this way, through the tendency to rapid growth and decay of cell life, causing normally the presence of a higher leucocyte percentage, and through the tendency to the occurrence of a leucocytosis by virtue of causes less operable in adults, the leaning towards increased elimination of uric acid in young children can be explained. It is unnecessary to regard it as due to any defect in hepatic metabolism or a reversion to a lower type.

It was formerly held by Murchison that the liver was the seat of uric acid formation. Murchison fancied that increased excretion of uric acid or its presence in the blood and tissues indicated functional derangement of the liver, with disturbance in the metabolism of proteids. Charcot held a similar view, which opinion, as remarked, has also dominated the writings of Fothergill and many others. It was thought that since uric acid merely represented a less highly oxidized product than urea, and since from uric acid by oxidation and hydration urea could be derived, the proteids of the food commonly passed through the stage of uric acid formation in process of conversion into the more complex urea. This view, as before remarked, can no longer be regarded as tenable.

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[1] Normally, at the height of digestion of a meal rich in proteids, a leucocyte increase of about thirty per cent. is usual. The marked digestive leucocytosis occurring commonly in the new-born may be accounted for by the fact, as observed by Cabot, that food determines an increase in proportion to its being a novelty to the stomach.

In nephritis, gout, lead poisoning, and numerous other diseases in which excessive nuclein decomposition favors copious alloxuric formation; in certain blood diseases, such as leukæmia and a few primary and secondary anæmias; and in conditions inducing suboxidation, such as heart disease, emphysema, pneumonia, and pleurisy with effusion, uric acid has been found to exist in the blood. In certain of these diseases, more especially in those accompanied by leucocytosis, it is apt also to be excreted in considerable excess by the kidneys, so that, as in leukæmia, there may be an enormous increase over the normal. In these diseases there can be little doubt of its immediate formation from the nuclein of the leucocytes in the spleen and elsewhere.[1]

In health, on the contrary, and even in conditions in which, without the occurrence of a leucocytosis, uric acid is found in the blood, such as in gout, in lead poisoning, and in chronic nephritis, uric acid, as was first suggested by Garrod, is now regarded on the best of evidence as being formed only in the kidney. Thence it is eliminated in the urine, none appearing in the blood save as a result of diseased conditions of the kidney, in which the eliminating power for uric acid, as for other waste products, is diminished. Recent experimental evidence, which shows quite conclusively that uric acid is *never present normally in the blood*, as before remarked, points most strongly to the fact that the seat of its formation is the kidney. The evidence indicates that its formation occurs normally, almost solely, in the kidney, and from the alloxuric and xanthin bases conveyed to this viscus from the blood; urea, apparently, may also furnish a modicum. It seems curious, indeed, that from urea, representing a more complex and more soluble and highly oxidized product, uric acid should be derived; yet, strangely, the evidence is much in favor of the formation of a certain amount, at least, of the uric acid from urea. Some authorities, such as Luff, regard urea as the probable source of all the uric acid excreted. In favor of this he points out that in birds, which excrete their nitrogen as uric acid and not as urea, urea is found in the blood, as is the case in mammals and also in man. In these the blood of the renal artery is much richer in urea than the renal vein. Horbaczewski's and Latham's experiments seem to show that uric acid is formed in the kidney largely from the conjugation of urea and glycocine. Glycocine, which is derived from the glycocholic acid of the bile, is one of the amido bodies, such as taurine, leucine, and tyrosine, which are present in the organism normally, and which are presumed to be converted into urea in the liver. Latham holds that if transformation of glycocine into urea be interfëred with in the liver, a related body, hydantoic acid, and, finally, the soluble hydantoin, will be formed, from which in the kidneys, from its union with urea, uric acid will be produced. Latham gives the following formula representing the formation of uric acid from urea:

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[1] Gumprecht (Centralblatt für allgemeine Pathologie u. pathologische Anatomie, 1896, Bd vii. S. 820) has shown that the related substances, the alloxuric bases, take the place of uric acid in those exceptional cases of leukæmia in which excessive excretion of uric acid does not occur. This same increase of alloxuric bases has been noted after the ingestion of nuclein-yielding substances in conditions other than leukæmia in which the uric acid output was not increased.

$$\underset{\text{Urea.}}{3CH_4N_2O} + \underset{\text{Glycocine.}}{C_2H_2(NH_2)O.OH} = \underset{\text{Uric acid.}}{C_5H_4M_4O_3} + 3NH_3 + 2H_2O.$$

If this view of Latham's be correct, it furnishes evidence that a disturbance in hepatic metabolism may influence the amount of uric acid formation.

Luff regards it as probable that glycocine is concerned in the production of uric acid, since, in the carnivora, the urine of which contains little or no uric acid, the bile contains no glycocholic acid, but only taurocholic acid, and therefore yields no glycocine.

Although urea may thus be a partial source of uric acid, the researches of Horbaczewski indicate that it is very probable, as before stated, that the alloxuric and xanthin bases produced from the destruction of nuclein are the more likely common sources of uric acid. In conditions, such as leukæmia, in which excessive production and destruction of leucocytes occur, and in which uric acid is present in the blood and appears in the urine in large amount, it seems probable that the formation of uric acid occurs not only in the kidney but in the spleen and elsewhere in the body, but also in other diseases in which uric acid in the blood is not dependent upon a marked nuclein destruction, as in nephritis, lead poisoning, gout, and so on, uric acid formation probably occurs only in the kidneys, and its presence in the blood is due to deficient secretory activity in these organs. The most tenable view is that in gout no marked increase of uric acid production occurs, but that the paroxysm of the malady, attended with the deposit of urates about the joints, is due to a temporary or permanent disorder of the uric-acid-eliminating portion of the tubules of the kidneys, leading to the absorption of the acid, and also to an unknown factor.

Levison's[1] observations tend to show that in gout, as has long been suspected, a latent or evident renal disorder, in most cases of organic nature, is present. This, he believes, almost invariably involves the epithelium of the convoluted tubules, which similarly seems to be the primary seat of disease in granular kidney, from which interstitial changes are secondary. It is the epithelium of the convoluted tubules that is probably concerned in the excretion of uric acid.

It is now essential to consider certain chemical problems involved in lithuria, and to indicate the difference between a condition which has hitherto by the old authorities been supposed to be due to the excessive formation of uric acid in the body, and the occurrence of uric acid gravel.

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[1] Zeitschrift für klinische Medicin, 1894, Bd. xxvi. S. 293.

Upon these problems Sir William Roberts, in his "Croonian Lectures on the Chemistry and Therapeutics of Uric Acid Gravel and Gout," has thrown much light. He has clearly shown, in the first place, that *uric acid itself* is not present physiologically either in the body or in the urine, and that pathologically it is encountered only as gravel and calculus in the urinary passages, and as a crystalline sediment in the urine; that when its soluble salt is present in the blood, as in gout, it is injurious only by virtue of its mechanical effects, which result on its deposition as sodium biurate in needle-like crystals in the joints and tissues.

Uric acid is a bibasic acid, containing two atoms of replaceable hydrogen. It forms three varieties of salts, two of which alone concern us; the third, the neutral urate, is never encountered in the economy, and is known only as a laboratory product. The two are, first, the very stable but highly insoluble acid urate or biurate, with the general formula MHu. This salt in the form of acid sodium urate is the chief constituent of the deposits of gout. It is not a physiological component of the urine or blood, nor is it ever encountered, even pathologically, in unchanged urine. The second salt is the physiological combination of uric acid, that which exists normally in the urine and occurs in the blood in the condition in which it is produced in great excess, or, as in gout and in nephritis, in which its elimination is interfered with. This, styled the quadriurate by Bence-Jones, is a compound first investigated by him, and recently more comprehensively by Roberts. The latter has shown it to be a definite physiological salt of uric acid, a chemical union of bi- or acid urate and uric acid in the proportion of a molecule of each. Its formula is represented by the symbol Hu_2,MHu. This is the salt of uric acid which, as sodium quadriurate, converted into the sodium salt by the sodium carbonate of the blood, circulates harmlessly in the blood in gouty subjects until precipitated as sodium biurate, causing a paroxysm of gout.

These same quadriurates have been ascertained by Roberts to constitute the entirety of the urinary excretion of birds and serpents. He regards all the morbid phenomena due to uric acid as arising from secondary changes in these. With the secondary changes, such as may occur within the organism itself, we are not so much concerned here, since gout, the only form in which manifestation of the presence of uric acid in the fluids of the body is evident to us, is extremely rare in childhood; but as they occur in the urinary passages in the young, causing the various phenomena to which the term lithuria, or the uric acid condition, is applied, there is much to be said.

THE CAUSE OF URIC ACID PRECIPITATION IN THE URINE AND OF URIC ACID GRAVEL.

Normal acid urine has an inherent tendency to the liberation and precipitation of its uric acid. This tendency may be regarded as abnormal only when such precipitation occurs prematurely, whilst, as is remarked by Roberts, the urine is still sojourning in the kidneys or the bladder. This view of Roberts's places gravel as merely due to an exaggeration of conditions which prevail normally, but in a less pronounced degree. Uric acid in process of excretion by the kidney, and, subsequently, after excretion in the normal unchanged urine, exists in the form of the soluble quadriurates as a salt of potassium, sodium, and ammonium. The amorphous urate deposit met with in concentrated urines is composed of quadriurates, and the same compound exists in solution in urine free from sediment. The first step in the precipitation of uric acid is the dissociation of the quadriurates, by the action of water, into a bi- or acid urate and the liberation, with precipitation, of a certain amount of uric acid. The less insoluble acid urates remain in solution. Following this, as is shown by Roberts, the biurate is again converted into quadriurate by the action of the monometallic phosphate present in urine, which phosphate in turn is converted into the dimetallic salt, according to the formula,—

$$\underset{\text{Biurate.}}{2(MHu)} + \underset{\text{Monometallic phosphate.}}{(MH_2PO_4)} = \underset{\text{Quadriurate.}}{(Hu_2,MHu)} + \underset{\text{Dimetallic phosphate.}}{(M_2HPO_4)}$$

"These alternating reactions, breaking up of quadriurates by water into biurate and free uric acid, and recomposition of quadriurates by double decomposition of biurate with monometallic phosphate, go on progressively until all the uric acid is set free."[1]

The decomposition of quadriurates in normal urine is inhibited by the contained pigments and the saline ingredients, such as the chlorides, chiefly, but also the sulphates and the phosphates. The salts of potassium have a more inhibiting influence than those of sodium or ammonium. Roberts also found that the addition of a fixed alkali to urine tended to prevent this decomposition, while urea is entirely without influence. The coloring matter of the urine, he found, has a decided power in maintaining the integrity of the quadriurates. Were it not for the urinary pigment and these inhibiting salts, precipitation of uric acid would occur as a usual affair in the urinary passages of all.

The acidity of the urine is not due to the presence of a free acid, but presumably to the acid sodium phosphate (NaH_2SO_4). This salt, derived from the blood, has been thought to owe its conversion in the kidney from the alkaline salt (Na_2HPO_4) to the action of sulphuric, hippuric, and uric acids and carbonic anhydride, through the removal of an atom of sodium and the replacement of an atom of hydrogen.[2]

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[1] Roberts's "Uric Acid Gravel and Gout," p. 42.

[2] Recently doubt has been thrown by Liebermann on the commonly accepted view as to the cause of the occurrence of the acidity of the urine. To quote from Harley (British Medical Journal, March 23, 1895, p. 638): "Liebermann has now shown that the kidney substance contains an acid reacting substance (lecithin-albumin) which is capable of converting an alkaline reacting salt (such as Na_2HPO_4) into an acid one, and that even blood-

The occurrence of a precipitate of uric acid gravel, either in the urinary passages or subsequent to voiding, must be due either to a diminution in the amount of the natural solvents or to the formation of uric acid in excess of that upon which these solvents can be effective. With a diminution in one or more of the salts mentioned, or in the coloring matter, a urine containing even less than the normal quantity of uric acid will show a tendency to precipitation. Poverty of urine in saline ingredients is a very influential factor, as remarked by Roberts, in determining the frequency of stone among the children of the poor as compared with those of the better-fed classes. Concerning this he remarks, "The children of the poor are fed largely on farinaceous articles,—bread, gruel, oatmeal, and potatoes,—with but a scanty allowance of milk, meat, and fish. Wheat flour contains only 0.51 per cent. of mineral matter in proportion to the totality of the dry substance, oatmeal only 2.50 per cent., potatoes only 2.50 per cent.; whereas milk contains 5.50 per cent., and the various forms of meat and fish 5 to 5.50 per cent. Rice, which forms so large a part of the diet of the native of India, only contains 0.39 per cent. of mineral matter in proportion to the totality of the dry substance of the grain. These enormous differences in the amount of saline ingredients in the articles of food must, of course, make a corresponding difference in the proportion of the saline constituents of the urine, because saline matters pass out of the body almost exclusively through the kidneys. On the other hand, the well-known immunity enjoyed by sailors from stone and gravel depends, no doubt, as Mr. Plowright has shown, on the prodigious quantity of salt which seafaring men habitually consume with their food. The same observer has pointed out that the dwellers in a district of Norfolk called Marshland, where the water is brackish, are singularly free from stone as compared with their less fortunate neighbors in the adjacent districts of that county."

But poverty of urine in saline ingredients and pigments accounts for

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serum, or defibrinated blood, when filtered through a layer of lecithin-albumin, is converted into an acid filtrate. An alkaline solution of Na_2HPO_4, when artificially circulated through a freshly extirpated dog's kidney, yields an acid urine. In the normal formation of urine the bases which combined with the acid lecithin-albumin are in their turn broken down by the carbonic acid formed in the tissues, so as again to set free the acid lecithin-albumin for renewed action. If this view is correct, under ordinary circumstances, a sufficient quantity of alkaline (Na_2HPO_4) salts being present in the blood, the renal cells do not break up the soluble urates in their passage into the urine. When, however, this equilibrium is upset by a diminution of the alkaline salts, the soluble urates being in normal quantity, the renal cells will set free uric acid, which will be precipitated in the urinary tubules; or, if the urates are in excess, the alkaline salts may fail to be in sufficient quantity to keep the urates in a soluble form.

"Liebermann found if a saturated solution of uric acid in Na_2HPO_4 be divided into two parts, and one of them diluted with water and the other with Na_2HPO_4, when they are passed over lecithin-albumin the latter contains the most uric acid, from less of it having been retained,—that is, precipitated. An excess of lecithin-albumin in the kidney, it is thus seen, would lead to an acid urine being excreted; and it looks as if the alkaline urine of herbivora is due to the amount of lecithin-albumin in the kidneys not being sufficient to break up the alkaline salts of their diet."

only certain limited groups of calculus cases. There are, as Roberts states, other and larger groups in which, with no deficiency in salt or pigment, the chief determining factors in precipitation are the high proportion of uric acid and the extreme degree of acidity of the urine. The influence of these two factors is decided.

The reaction of the urine is influenced by several factors. During digestion the natural acidity tends to diminish and may entirely disappear, the urine becoming alkaline. The acidity of the urine is increased by fasting, by the ingestion of highly albuminous food, by active muscular exercise, and by the employment of mineral acids.

Although a diet poor in salines increases the tendency to the undue precipitation of uric acid for the reasons stated above, vegetables rich in potash salts of the vegetable acids tend to promote alkalinity of the urine by virtue of the organic acids liberated in process of excretion being converted into alkaline carbonates. A diet largely of potatoes, which are rich in potassium malate, for a similar reason tends to produce alkalinity of the urine. A diet rich in albuminoids and phosphates, whether animal, cereal, or leguminous, tends to raise the acidity of the urine. Allen,[1] in referring to this, remarks that for this reason Bunge views cheese as a highly unsuitable food for those inclined to gravel, since in its manufacture the alkaline salts are lost in the whey, and the casein yields considerable sulphuric acid and phosphoric acid on combustion. He also states that salted meats and fish are objectionable for similar reasons.

SYMPTOMS AND DIAGNOSIS OF THE URIC ACID CONDITION, AND ESPECIALLY OF URIC ACID GRAVEL.

A few remarks are first in order concerning such manifestations in children as may be regarded as due to the presence of uric acid or allied substances in the organism. As previously stated, uric acid is not a normal or usual constituent of the body either as uric acid or as a salt. Its probable mode of production and the rarity of its presence in the blood or tissues of the body have already been mentioned. The views of Haig, which have in recent years gained such wide acceptation, as to the extraordinary far-reaching effects of uric acid as a factor in the causation of disease, are based upon conclusions evolved from premises established, as has been aptly remarked by Sewell, merely in his own consciousness.[2] Haig's views have gained wide notoriety, and have been productive of both good and harm: of good, so far as they have led to a more rational dietary, more especially to the avoidance of undue consumption of animal food; of harm, so far as they have tended to divert attention from the real cause of ailments only indirectly, if at all, related to uric acid. Evil has resulted, too, in consequence of the hold his views have on the popular mind, divert-ing attention from a correct and more comprehensible study of the so-called uric acid condition. Totally oblivious to data overwhelmingly against the truth of his theories, Haig continues to hold that the presence of uric acid in the organs and tissues of the body is the cause of innumerable ailments, a few of which are headache, epilepsy, melancholia, rheumatism, gout, diabetes, abnormally high blood-pressure, Bright's disease, and so on. The investigations of Roberts, and very lately of Luff, seem to have established conclusively that the notion that uric acid may be deposited in certain of the viscera and in the fibrous tissues, owing to diminished alkalinity of the blood, is incorrect. Experiments of Roberts and of Luff show that diminution in the alkalinity of the medium holding sodium biurate in solution does not promote its deposition; conversely, the theory that increased alkalinity of the blood dissolves and sweeps out the fancied accumulated store of uric acid is also shown to be erroneous. It must be insisted upon that no valid evidence exists that uric acid possesses any toxic properties whatever, and that any damage resulting from its presence in the tissues in gout is the result of the mechanically irritating action of its spicules, such, Roberts remarks, as might be produced by the deposition of crystals of any similar mechanically irritating salt. Roberts is inclined to regard the manifestations of irregular gout affecting the stomach, the heart, and the nervous system as due, if to uric acid at all, to actual precipitation of crystals of sodium biurate into the connective and fibrous structures of the implicated organs, or into the fibrous sheaths of the nerves which control their functions. Experimental proof is all against the evidence as to uric acid having toxic properties. Moreover, as Roberts wisely states, "the idea seems opposed to broad biological analogies. Uric acid is the physiological homologue of urea; each of these bodies constitutes, in its separate domain, the final term of nitrogenous metabolism. It cannot be said without an abuse of terms that urea is a poisonous substance, and it would be strange if its homologue, uric acid, differed from it in so important a particular as the possession of toxic properties." It would follow, therefore, that when symptoms occur other than those that may be attributable to the mechanical effects of uric acid, and which seem to be produced by its action as a toxic substance, it is more probable that these are due to the presence in excess of another substance really toxic, such as one or more bodies allied to uric acid, of the xanthin group, about the effects of which we know little, but which we have reason to regard with some apprehension. It is to a study of the action of these that attention should be directed by Dr. Haig and others interested in this line of research. Some of this group, on very plausible grounds, are asserted by Rachford to be the causes of epilepsy occurring in adult life not attributable to syphilis and lead poisoning, and to be the chief factors in the causation of migraine,—a more probable view than that uric acid is so concerned. It is more than likely that symptoms of lithæmia and certain varieties of melancholia may be so caused, and it is also questioned on good evidence whether many of the cerebral manifestations of lead poisoning, and certain of the common symptoms of the gouty state, may not be due to the retention of waste products of the xanthin and its allied series. It has been questioned, indeed, if gout itself may not have as a determining factor some other agent than mere uric acid. Sir William Roberts has properly remarked that there are certain manifestations of gout that cannot be explained solely by the presence of uric acid.

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[1] Chemistry of the Urine, 1895.

[2] See an excellent review of Haig's uric acid theory by Sewell, Medical News, September 16, 1893.

Kolisch,[1] indeed, regards the xanthin bodies other than uric acid as the chief agents concerned in the production of gout. The alloxuric substances and xanthin bases, the ultimate products of nuclein decomposition, are excreted, according to him, largely as uric acid. With disease of the kidney, however, there is less formation of uric acid and a corresponding increase in the production of the xanthin bases. In the urine of the gouty Kolisch has found an invariable increase of these alloxuric substances. He believes that these, especially the xanthin bases, tend to produce renal lesions analogous to those produced by lead. Kolisch also holds that there is probably a constitutional tendency to increased destruction of nuclein inherited by the gouty, but it is only in those in whom the renal function becomes impaired that the more decided manifestations of gout appear. Weintrab[2] has noted a similar excessive excretion of alloxuric bodies in the gouty.

Concerning the symptoms of the so-called uric acid conditions, other than exhibited in the form of gravel and stone, there is little to be said. There are but few instances of gout on record occurring in childhood.[3] Other conditions, which on valid grounds have been attributed to the presence of excess of uric acid in the system of children, need not here be considered. It is not probable, as Fothergill held, that struma is in any way related to excessive formation of uric acid.

The occurrence of uric acid gravel is common almost from birth, due probably most often to an excessive physiological output over that in adults, but also not infrequently to a deficiency in certain of the natural solvents in the urine, or to excessive acidity of the urine. When crystals of uric acid are precipitated in the tubules of the kidneys, if in any quantity, symptoms are quite obvious, although they may be attributed to other causes. Goodhart has pointed out that renal colic in children is often mistaken for stomach-ache pure and simple. Children who are subject to uric acid gravel are apt to be of changeable and nervous disposition. They sleep poorly and have disturbed dreams. Constipation is not uncommon, due, in all probability, to an inhibitory reflex caused by the pain. Periodical attacks of weariness and uneasiness in older children are not unusual, with a mild melancholic tendency. The only symptom of undue uric acid precipitation in the kidney may be pain referred to the umbilicus or to the lumbar region. Although this is unusual, the pain may be so intense as to be accompanied by nausea and vomiting, as in adults, and sometimes, though rarely, save in the passage of a calculus, by hæmaturia. The pain, according to Levison, is not always referred to the umbilicus or to the loins, but may extend into the back and downward to the thighs. There may also be strangury, and frequent urination is very common even in the mildest cases. The attack has usually a sudden onset, the child frequently awakening in the night, crying out with pain. The more severe pain of colic may be absent in milder cases. A dull ache in the loin or back may be alone complained of, and may be mistaken and treated by the unwary, when present in the adult, for lumbago (Horbaczewski). An account of these pains can, of course, be elicited only from older children. In infants a tendency to constant restlessness and crying should lead to a microscopic examination of the freshly voided urine for uric acid crystals. Pain in children may not be complained of at all, and restlessness, disturbed sleep, frequent micturition, and nocturnal enuresis[1] may be the only symptoms suggesting this condition. It is not uncommon to encounter children practically symptomless, save as regards pallor, in whom injury to the kidney is occurring through the undue precipitation of uric acid. A boy of this sort, aged eight years, is now under observation, in whom attacks of acute gastric catarrh have been common: examination of the urine disclosed the presence of a small amount of albumin, and of innumerable mucous cylindroids, and occasionally of fragments of granular casts, evidently due to the precipitation in the tubules of the kidney of uric acid and the acid urates. Besides the occurrence of these in some amount in a freshly voided specimen, hyaline casts and casts formed of urates alone were common, as was the presence of mucous and hyaline cylindroids. There also abounded, as is usual in this condition, leucocytes and erythrocytes. From the foregoing it may be seen that sometimes the diagnosis of the uric acid condition is possible only, when a deposit of gravel is not evident in the urine, by a microscopic examination of a freshly voided specimen. In infants a specimen for such an examination can usually be obtained only by catheterization. This commonly offers no difficulty, a soft catheter of proper calibre being employed. Often pressure over the pubes, when the bladder is moderately distended, will induce urination in infants without the use of the catheter.

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[1] Wiener klinische Wochenschrift, Bd. viii. S. 787, 1895.

[2] Charité-Annalen, t. xx. p. 215, 1895.

[3] Its rarity in children, in whom uric acid is present normally in higher ratio than in adults, is due probably to the fact that antecedent disease of the kidney of the variety common in gout is unusual in early life.

Since the deposit from the urine of uric acid in the form of obvious concretions or in microscopic amounts can be regarded as abnormal only when it occurs in some part of the urinary passages, and since normal urine often tends, soon after passing, to deposit its uric acid, though not present in excess, through decomposition of the soluble quadriurates, it is important to examine the urine, as stated, immediately after voiding. The urine may be clear and entirely free from other sediment than the usual slight cloud of mucus which is apparent after standing. As the microscopic examination for crystals of uric acid and acid urates in those in whom gravel is not obvious should be made at once after voiding, the use of the centrifuge, holding tubes of some capacity, is then imperative in the collection of sediment. By its employment crystals of uric acid and insoluble urates are thrown down, which are readily recognizable by the microscope. The most suitable specimen for examination is that of the morning before breakfast, as the urine is then more strongly acid and is therefore more apt to have deposited some of its uric acid in susceptible subjects.

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[1] Nocturnal enuresis is not infrequently due to the irritation produced by the passage of uric acid crystals.

Should deposit of uric acid be usual in the tubular structure of the kidney, renal irritation may be expected, resulting in the presence not only of small amounts of nucleo- or of sero-albumin, but also usually of hyaline casts and hyaline and mucous cylindroids, and perhaps, though more scantily, a very few granular and epithelial casts. Leucocytes and erythrocytes may be observed in some amount. The occurrence of hyaline casts and mucous and hyaline cylindroids as a result of the irritating action of the precipitated uric acid in the tubules of the kidney is a common observation. Granular and even epithelial casts in these cases are regarded as not infrequent by many observers, notably Levison. Ebstein, as remarked by Levison, showed that uric acid infarcts, so common in the kidneys of the new-born, have a substratum of albuminous matter arranged in concentric layers interrupted by radial sectors, in which substratum the uric acid crystals are embedded. This albuminous substance, in all probability, is simply a nucleo-albuminous exudate from the renal cells in consequence of the irritating action of the uric acid crystals. As the result of examination of infarcts in a number of cases in the newly born, Ebstein found that the granules of the infarction were similar to larger concretions of uric acid, and that they were present both in the pyramids and in the tubules. Levison quotes Martin and Ruge as having found albumin in the urine in almost all of twenty-four very young infants in consequence of uric acid irritation. Hyaline casts were present in fourteen of the twenty-four. In adults the association of casts, and often of small amounts of albumin, nucleo- or sero-, with the presence of free uric acid in the freshly voided urine, is well established.

Mygge, as a result of a study of a large number of cases in which there was a tendency to undue precipitation of uric acid, found that the presence of casts was almost proportionate to the degree of the uric acid precipitation. Levison, who has for several years made a study of the urine in cases of real or suspected lithuria, examining the urinary sediment by the employment of the centrifuge immediately after voiding, has noted that in those in which the lithitic tendency has been persistent, the coexistence of not only hyaline but granular casts with uric acid crystals is usual. He has sometimes observed uric acid crystals embedded in or precipitated on the casts. This he has frequently noticed in himself. A sufferer from uric acid gravel, he can by a slight departure from normal living induce an attack. Several days after its incipience, when macroscopically the urine may be entirely free from sediment, he has often noted the presence of granular casts and numerous leucocytes coexistent with an immense number of uric acid crystals. Casts can be no longer found a few days after his urine has been freed from uric acid crystals by the steady employment of an alkali.

Prognosis.—The very common association of cylindroids, and even of hyaline casts, and less frequently of granular casts, as a result of the irritating action of the precipitated uric acid in the tubules of the kidney is of ominous note, indicating, if long continued, a certain tendency, either in children or in adults, towards secondary renal disease the result of the irritation. Changes both in tubule and in interstitial structure may thus early be originated. Many cases of granular kidney doubtless thus have origin in long-continued and perhaps wholly unnoticed preceding precipitation of uric acid crystals in the tubules of the kidneys. The writer has had numerous such cases among adults under observation. The undue and premature precipitation of uric acid is in consequence either of excessive formation or merely of lessened solubility. In children uric acid precipitation commonly means undue formation, although this may not be the case; but it may be due to a mere lessening in the natural solvents, as in the adult, for uric acid in the urine leads to precipitation. Whenever it is possible to do so, attention should be paid to ascertaining this point. Commonly it is practically impossible in small children to collect the twenty-four-hour urine in order to determine the exact amount excreted. Attention has rather to be paid to the height of color to obtain information as to the probable amount of pigment present, and as to the existence of a fair amount of salts, such as the chlorides.

Treatment.—As to the uric acid conditions other than gravel. The occurrence of gout in children is so rare that space cannot be given to a consideration of its treatment here. Its therapy is on identical lines with this ailment in the adult. It must be stated, in passing, that the treatment of gout is not the treatment of lithuria, and that as concerns diet, although a similar alimentation may be indicated in each condition, the important fact developed by Sir William Roberts must not be lost sight of, that an abundance of table salt is valuable in the latter condition, but harmful in the gouty state. As concerns the special treatment of so-called lithæmia, there need be little said. It is not common in children (if other conditions be not mistaken for it, as is usual in adults, the term being almost as loosely applied as malaria). Lithæmia, when encountered in children, is usually due to disturbed hepatic metabolism the result of faulty nutrition, as in adults. In its treatment, as in lithuria, a lessened intake of animal food if this has been eaten too largely, an abundance of out-of-door exercise, the maintenance of free skin and bowel activity, and the correction of any digestive disturbance present are necessary. Marked benefit is obtained from calomel in single, nightly doses of one or more grains, once or twice weekly, for short intervals, and the further employment of small doses of podophyllum resin, in combination with aloin and extract of hyoscyamus if the bowels are sluggish, and if the use of calomel in the manner described has not been followed by more than transient relief of symptoms. In place of podophyllum, the specific action of which on the liver is too little known, there may be employed, should this drug in suitable doses nauseate, cramp, or otherwise disagree, a combination of equal parts of purified sodium sulphate and phosphate, of which from twenty to thirty grains would be suitable for a child ten years of age. This is given from fifteen to twenty minutes before breakfast in from two to four ounces of water, to which may be added a few drops of tincture of nux vomica or of one of the simple bitters, such as quassia or calumba. The writer has employed this combination of sodium sulphate and phosphate for years, often adding sodium bicarbonate, in conditions in which there are indications of sluggish liver action associated with a tendency to duodenal catarrh.

In these cases of lithæmia, sugars in the form of pastry or "sweets" are especially to be avoided; otherwise the diet is as indicated in cases of lithuria.

There has been great diversity of opinion as to the form of diet suitable in the uric acid condition, and especially in lithuria. Equally competent authorities might be quoted as holding totally opposite views. Formerly, when it was supposed that uric acid represented a step towards urea formation, and that the former was a direct product of the nitrogenous food ingested, it was held that a non-nitrogenous dietary was rigidly indicated. Now that it is accepted that uric acid is formed almost solely from the decomposition of the leucocytes, a somewhat modified view prevails. What has, however, led to confusion as to a suitable dietary in dealing with adults subject to lithuria, and caused curious contradictory results to be obtained, is the failure to recognize the fact that, as concerns uric acid gravel at least, the form of diet should depend a great deal upon whether the too early appearance of uric acid precipitation is due to excessive formation or indirectly to a condition of the urine interfering with proper solution. This point is practically impossible to ascertain with certainty in many cases of the disease in children, but attention can be paid, as previously stated, to conditions influencing the solubility of uric acid, such as the approximate daily output of urine, the comparative amount of saline ingredients, and the degree of acidity. It may be inferred that, given normal output, somewhat high color, and low acidity, the undue precipitation is the result of increased secretion.

As to the question of diet again, Goodhart quotes Sir Alfred Garrod as stating that he has not observed that meat-eaters are those who are most addicted to the passage of uric acid. Goodhart remarks, "Now surely you must all agree with me that you have long known from the observation of this very point in childhood that it is certainly not the meat-eating children who pass uric acid, but it is the largely farinaceous feeders. And if you want to control this defect of assimilation, you will do so very little by the exclusion of meat from the diet. . . . And this is strongly confirmed by what I think used to be a matter of common teaching when I was a student, that stone was so usual in Norfolk because of the largely farinaceous diet that the agricultural laborer lives upon. The enormous frequency of stone among the natives in India is, I fancy, still more strongly to the point." Both Goodhart and Garrod failed to distinguish between excessive output and the normal with lessened solubility. The frequency of stone in India and among the Norfolk residents is due to the form of diet, as previously stated, not permitting of a sufficient amount of urinary salts to maintain the uric acid as soluble quadriurates.

The facts all point to the necessity in childhood for great moderation in nitrogenous food, and especially animal nitrogen, since in children, from the extreme activity in their vital processes, excess of nuclein-yielding elements readily originates increased uric acid formation. It may be laid down as a rule that the diet in childhood should be one especially to prevent undue uric acid formation. For this reason the diet suggested by Klemperer[1] for uric acid gravel seems most suitable,—*i.e.*, foods containing little nuclein-forming principles, such as milk, the cereals and vegetables, and white of egg. On the contrary, such foods as thymus, liver, brain, kidney, pancreas, and veal should be allowed sparingly, if at all, because they are especially rich in nuclein. Kossel remarks that the paranuclein of yolk of eggs has a similar effect to other nuclein-containers, so that it, too, should be excluded. Ordinary meats may be permitted in moderation, provided sufficient out-of-door exercise is taken. Children, fortunately, do not require meats, as do adults. It is unquestionable that under a meat diet there is increased uric acid output, although not to the extent observed when the nuclein-forming foods mentioned are eaten. Still, under a meat diet, the phosphoric and sulphuric acids form in excess, tending to lower the alkalinity of the blood and increase the acidity of the urine. As is remarked by Mordhurst, in discussing this question,[2] the urine of the carnivora is always acid, whereas that of the herbivora is neutral or alkaline.

As previously stated, the oxidation of the nuclei of leucocytes and other cells of the human body furnishes on any diet in the adult from 0.3 to 0.6 gramme of uric acid daily. The uric acid excretion is scarcely greater when milk alone is taken than it is during fasting. Klemperer found that after the ingestion of two litres of milk, without other food, the urine contained from 0.5 to 0.6 gramme of uric acid; after five hundred grammes of meat, the urine of the same person contained from one to two grammes of uric acid; after five hundred grammes of thymus, it contained about three-quarters of a gramme more of uric acid. Klemperer remarks that there is no exact quantitative relation between the nuclein in the food and the uric acid in the urine; the nuclein may also be excreted as urea, just as uric acid may be when taken by the mouth. The difference in different individuals seems to depend, according to Klemperer, on some being able to transform the uric acid formed more readily into urea than do others. Klemperer points out that the ingestion of xanthin-containing bodies leads, as does the ingestion of nuclein, to an increase of uric acid in the urine. Hence tea, coffee, and meat extracts must be interdicted.

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[1] Berliner klinische Wochenschrift, 1896, S. 33.

[2] Verhandlung des Congress für Innere Medicin, Bd. xii., 1893, S. 495.

To maintain the proportion of salts in the urine, it is important, in cases of too easily precipitable uric acid, to see that the diet contains an adequate amount of saline elements. Food rich in salts, therefore, should be taken, such as milk and fresh salads and garden vegetables. Fresh fish is also advisable. An abundance of table salt should be allowed in these cases, with the idea of increasing the solvency of the quadriurates in the urine. As regards vegetables, asparagus, which contains a body of the xanthin group, should be avoided. Asparagus has been erroneously stated to be a diuretic, the increased frequency of micturition caused by it through urinary irritation being mistaken for increased diuresis. The large white variety is thought to be more irritating to the kidney than the smaller green form.[1] Fresh fruits are beneficial by virtue of the vegetable acids and the salts contained in them, which in the organism are oxidized into carbonates and serve to alkalize the urine. The vegetable acids, if craved, may also be allowed for the same reasons. As may be supposed, the administration of mineral acids, on the contrary, tends to increase urinary acidity. Nitro-hydrochloric or hydrochloric acid, should the condition of the stomach seem to demand the one or the other, is not contra-indicated. Small doses of dilute hydrochloric acid *before* meals are often of singular utility in relieving an oxaluria accompanying the acid condition. The coincident occurrence of oxaluria should demand increased caution as to diet and the tabooing of such articles of food as contain calcium oxalate in any amount. These include sorrel, spinach, garden rhubarb, and so on. Tea and cocoa are also, according to Esbach,[2] extraordinarily rich in oxalic acid, containing, per kilogramme, even more than sorrel and spinach.

Fatty foods are permissible if they cause no digestive disturbance. The same may be said of starches. As concerns sugar in the form of pastry, jam, and sweets, although they have no direct influence on the production and excretion of uric acid, clinical evidence, at least, is against their general use even in lithuria.

Alcohol is to be avoided in subjects of gravel, as, by increasing the decomposition of nuclein, it unquestionably, according to Levison, increases uric acid excretion, and, as phosphoric acid is liberated by this nuclein decomposition, the urine is rendered more acid thereby. Chittenden's experiments on dogs confirm the observation that uric acid excretion is increased by alcohol. Levison has repeatedly observed the same effect in man. He has noticed an increase from 0.6 gramme to 1.0 gramme under the moderate use of alcohol. In susceptible subjects free uric acid crystals may be readily made to appear in the urine under the ingestion of moderate doses of alcohol. Moreover, alcohol has a direct irritating action on the kidney in process of excretion.

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[1] It is said that this variety contains an excess of a principle inhibiting urinary secretion which resides only in the stalk of the green variety. In the green portion the glucoside asparagin is chiefly found, which may have a slightly diuretic effect.

[2] Bulletin de Thérapeutique, 1883, t. v., civ., p. 385.

Undue acidity of the urine is, in Roberts's opinion, the chief immediate determining factor in the precipitation of uric acid gravel, and the paramount indication for treatment is to diminish the acidity. He points out that much may be done by watching the regular fluctuations in "acid" and "alkaline" tides, so carefully studied by Bence-Jones and himself. The percentage of uric acid excreted is always highest during the hours of the more prolonged fast, such as that between the evening meal and that of the morning. Sleep is not only a time of fasting, and hence of hyperacidity of the urine, but also of recumbency and bodily immobility. Since the tendency to precipitation of uric acid is greatest during a fast, when the acidity of the urine is at its maximum, other things being equal, the intervals between meals should not be too prolonged. Roberts remarks that each meal acts on the urine as a dose of alkali and also as a diluent, and in both of these ways co-operates as a protection against uric acid precipitation. After the meal is absorbed and assimilated, the urine again becomes increasingly acid and concentrated, and, as a consequence, increasingly prone to deposit uric acid. As the first meal of the day is commonly quickly digested, the interval between breakfast and the mid-day meal should not be over four or five hours, unless a hearty breakfast has been eaten, which is inadvisable. Four light meals at moderate intervals are most appropriate in the treatment of lithuria. Roberts has found that at no time during the waking hours does the acidity of the urine tend to rise so high and its volume to fall so low as in the latter part of the interval between the first and the second meal. He further points out that the essential matter in the prophylactic treatment of gravel is to guard the urine from precipitation in the renal passages. If we can delay the precipitate whilst passing the tubules, any subsequent precipitation is practically harmless.

The employment of special and antacid remedies is of the greatest utility in the prevention or removal of uric acid precipitation. In mild cases of gravel a single full dose of an antacid taken at bedtime is sufficient to prevent precipitation and the occurrence of colicky pain. Of the antacids an excellent remedy is potassium citrate, a single dose of which for an adult is from thirty to sixty grains, and for a child of ten years from five to twenty grains. This should be taken well diluted. In more pronounced cases Roberts, who prefers potassium citrate to other alkalines, administers a second dose about the middle period of the hours of sleep, if it can be so arranged. Lithium carbonate or citrate may be used in place of potassium citrate if desired. The carbonate may be administered in effervescence, with lemon-juice. This salt is, in at least some cases, apparently more active even than lithium citrate.[1] It seems to be more diuretic, and is also mildly laxative. Kirk has noted that in adults a daily (before breakfast) dose of five grains was more efficient in curing a gouty gingivitis than was lithium citrate. Contrary to this opinion of Kirk, Mendelsohn[2] holds that lithium citrate is the most diuretic of the salts of lithium. Lithium carbonate is soluble only in effervescing solution, so that it is best administered in some aerated water, or it may be given in combination with the ordinary effervescing powder of sodium bicarbonate and tartaric acid, or in one of the many proprietary forms of granular effervescing salts. The lithium salts have the important advantage that while as efficient as potash, they are far less toxic, and so are immensely better suited for habitual use in the uric acid condition. They are a trifle more apt to cause gastric irritation, and must be administered in small quantities, a dose of from five to eight grains three times daily being suitable for an *adult*. One of the lithium salts mentioned is preferable also to a salt of potash in that by its use in the small dose indicated (grains two to three) for a child ten years of age undue alkalization of the urine is avoided, and thus the danger of the occurrence of phosphatic calculus minimized. As remarked by Levison, who refers to the above danger,[3] so long as the sediment of the freshly voided urine obtained by centrifugalization contains no uric acid crystals, and its reaction is but faintly acid, all is obtained that could be desired. To avoid the dangers resulting from excessive doses of alkalies, Van Noorden prefers calcium carbonate. This salt is not eliminated by the kidney, but by the bowel, uniting with phosphoric acid, is finally eliminated as calcium phosphate. Klemperer employs sodium bicarbonate in preference to the various alkalies mentioned. The writer, however, sees little use for the salts of sodium or calcium, almost habitually employing those of lithium or potassium, and preferably the latter.

Among the remedies proposed for the uric acid condition, and especially for uro-lithiasis, piperazin, piperazin hydrochlorate, and a derivative of piperazin, lycerol (dimethyl-piperazin-tartrate), deserve favorable mention. The writer was probably the first in this country to investigate the physiological and clinical effects of these in cases of chronic gout and in cases of uric acid gravel and stone. Although the physiological action of piperazin and its salts rendered their *modus medendi* obscure, the result in a sufficient number of carefully studied cases in the writer's hands justified, in his opinion, the therapeutic claims made for them. Although no uric acid increase, as uric acid, can be said to occur under their administration, that they render more soluble hitherto insoluble uric acid is undoubted, and, in the writer's opinion, they also favor the excretion of deposits of uric acid in another form than as a uric acid salt of piperazin.[1]

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[1] See Kirk, Lancet, 1894, vol. i. p. 1614.

[2] Deutsche medicinische Wochenschrift, September 30, 1896.

[3] St. Petersburger medicinische Wochenschrift, 1897, Nr. 1, S. 1; Nr. 2, S. 9.

That piperazin should be regarded as of more than theoretic value in the gouty condition was shown by the experiments on piperazin of Biezenthal,[2] who, to quote from my paper before referred to,[3] "induced artificially the deposit of uric acid and acid urates in the joints and tissues of doves by the well-known method of Ebstein, that of gradual destruction of the renal secreting structure by subcutaneous injections of neutral potassium chromate. To a large number of pigeons thus experimented upon piperazin was coincidently administered, with the object of noting if this drug would prevent the formation of these deposits. The results obtained were most interesting. In a very large percentage of the doves which received piperazin, it is stated that none of the characteristic deposits of urates occurred, indicating that piperazin actually possesses the power which test-tube experiments attribute to it."

I further said, "It may not be improbable that a difference in the degree of alkalinity of the blood in birds and in man will be found to account for the striking results obtained with piperazin in these experiments,—results not occurring from its use in many cases in man. As is well known, birds and serpents excrete their nitrogen not as urea, but as uric acid. The alkalinity of the blood of the herbivora is greater than in man, but I am unaware of any observations as to this point made on the blood of birds. It seems likely, however, that a similar condition may obtain with the latter, in order that the passage of uric acid through the blood and its free excretion by the kidney may be readily accomplished." That the view here expressed is probably a correct one is shown by the fact that in cases of chronic gout more pronounced results were obtained by the combination of piperazin with a salt of potash, such as the citrate,—such results as could not be obtained by the use of either drug singly.

In certain cases of uric acid gravel the writer obtained remarkable results from the employment of piperazin. In one case that had resisted the usual methods of treatment, in which indications of uric acid stone were very evident succeeding the long-continued passage of gravel, and in which operation had been at first urged by Professor Keen and myself, a complete and permanent cure rapidly followed the steady employment of piperazin.

The dose of the basic piperazin for an adult is from five to ten grains, three times daily, taken, largely diluted in water, on an empty stomach.

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[1] Concerning this action of piperazin, see my second paper on the subject, "The Influence of Piperazin on the Urine, and especially on Uric Acid and Urea Excretion," Therapeutic Gazette, February, 1894, p. 91.

[2] Berliner klinische Wochenschrift, 1893, S. 805. These experiments were originally made by Meisels (Archiv für Medicin, 1893) and subsequently confirmed by Biezenthal. Meisels found that lithium carbonate, etc., were without effects in this direction, and later experiments with uricidin have yielded negative results.

[3] Therapeutic Gazette, February, 1894.

The hydrochlorate is somewhat weaker in effect, and should be given in a trifle larger dose. The writer has had little experience with piperazin in children.[1]

Lycetol, which I was the first in this country to employ, is a more agreeable and less toxic piperazin salt. It is apparently of equal value with piperazin, and should be administered in the same or a trifle larger dose. It is of pleasant taste, and is non-hygroscopic.

Lycidin is a recent addition to the piperazin group. It is asserted that its power of dissolving uric acid is five times that of piperazin. It is a freely soluble, hygroscopic, crystalline substance, of somewhat unpleasant taste. Its chief employment thus far has been in cases of gout, in which good results are claimed from its use. In daily doses of from one to five grammes in an adult, Grawitz[2] noted no untoward effect. The writer has had no experience with it or with *uricidin,* another recent candidate for favor as a powerful uric acid solvent. Uricidin is not a definite salt, but a combination of lithium and sodium citrate with sodium chloride and sulphate. Its clinical trials are said not to justify the claims put forward for it. Meisels found that the excretion of uric acid was diminished by it, and that it was without effect in preventing or removing uric acid deposit in the tissues of birds first experimented upon by the method of Ebstein before mentioned, while piperazin had a decided effect in this direction.

The employment of the much-advertised lithia waters as uric acid solvents can give little better results than the use of ordinary soft water.[3] To avoid concentration of urine in cases of lithuria, pure water should be allowed in abundance. The frequent use of salines or drastic purgatives should be avoided, as these tend to concentration of urine. Klemperer prefers the use of water enemas for constipation. These favor diuresis as well. Calomel in moderate doses, as before mentioned, is unquestionably of the greatest service through its action on metabolic processes, and especially on the liver, the indirect source of uric acid formation.

An out-of-door life is, of course, essential in these cases, with judicious physical exercise. Immoderate exercise must not be allowed, as it apparently tends to increase the secretion of uric acid. Levison observed that in a case studied with a daily average excretion of 0.6 gramme, some hours' cycling or horseback-riding always raised the output considerably, as from the usual output of 0.6 gramme to, on one occasion, 0.981 gramme, and on another to 1.089 grammes. This increase may be explained by the production of a leucocytosis by the exertion. Undue exercise, also, by causing too free skin activity, leads to concentration of urine, with heightening of its acidity.

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[1] The writer observed toxic effects in adults when a dose of a drachm and over was taken daily for several days (see Therapeutic Gazette, February, 1894, p. 92, "Untoward Effects of Large Doses of Piperazin"). The dose for a child of ten years should not at first exceed two and one-half grains.

[2] Deutsche medicinische Wochenschrift, 1894, Nr. 20, p. 786.

[3] The most accurate test for the elucidation of the real effects of any mineral water as a uric acid solvent, as is well stated by Levison, is, instead of ascertaining its mere solvent power in the ordinary way, that proposed by Pfeiffer: "A healthy person is made to drink seven hundred cubic centimetres (one and a half pints) of the mineral water, and the urine which he evacuates after some hours is examined with regard to its solvent force. Two hundred cubic centimetres (six and three-quarters ounces) of the urine is poured upon 0.5 gramme (seven and a half grains) of dry uric acid and digested with it for fifteen to twenty minutes at a temperature of 37° C. (98 6° F.), when the uric acid is separated from the liquid, dried, and weighed."

Should a tendency to uric acid precipitation be increased under even moderate exercise, this should not be interdicted, lest more decided injury result to bodily nutrition. This increase under a moderate diet and the use of other methods laid down will probably disappear, especially if the dose of the alkali employed be properly graded.

In children in whom the undue precipitation of uric acid is apt to depend upon excessive secretion, in all likelihood due to a condition of leucocytosis, it is, as Levison advises, well to employ remedies in addition to uric acid solvents which restrain the exaggerated formation and decomposition of leucocytes. Such remedies are iron and quinine, and more especially arsenic. As concerns this last drug, Levison states that in severe cases of gravel in children in which alkalies were not sufficient to prevent the attacks of pain, he obtained good results by combining the use of arsenic with the alkali. He found that after a short time the urine did not contain as much uric acid as before.

THE TONSILS, THE PHARYNX, AND THE UVULA.

BY SAMUEL G. DABNEY, M.D.

THE TONSILS.

Anatomy.—The tonsils are masses of glandular tissue situated between the pillars of the fauces on each side of the throat. A similar collection is often found in the naso-pharynx and at the base of the tongue, the whole thus constituting an incomplete lymphoid ring. In shape they conform more or less to the space between the pillars containing them, being normally rather long and narrow. They vary much in size, even in health, but they should not project beyond the faucial arch. Each tonsil presents on its surface from five to ten openings, which lead down into small pouches or crypts. There is great diversity of opinion as to their limits in health. Wilson[1] believes that normally there is no such organ as the tonsil, which really consists of a mass of mucoid and lymphoid glands bound together by connective tissue as a result of frequent inflammation. Bosworth[2] emphasizes the trivial importance of the tonsils in health, but admits that they form an intimate part of the normal throat. The main blood-supply is from the tonsillar branch of the faucial artery, which enters at about the lower third of the organ. "The internal carotid artery is one and a half centimetres, the external two centimetres, distant from its lateral periphery" (Delavan). Harrison Allen[3] states that a large pocket is often found in the lower portion of the gland. He names the part above this the velar tonsil. He recommends that incisions for the reduction of hypertrophy should be made in the direction of growth, and not transversely; that diseased crypts and canals should be laid open, and that attempts should be made to restore the organ to its normal structure.

DISEASES OF THE TONSILS.

The most common diseases of the tonsils are acute tonsillitis, hypertrophy of the tonsils, usually but not always synonymous with chronic tonsillitis, and peritonsillar abscess, which, though involving rather the tissues around the tonsil than the organ itself, is usually classed with its affections.

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[1] New York Medical Journal, No. 63, p. 509.

[2] Diseases of the Throat, p. 15, 1892.

[3] American Journal of the Medical Sciences, January, 1893.

Acute tonsillitis may be either (*a*) follicular or (*b*) superficial.

(*a*) ACUTE FOLLICULAR TONSILLITIS.

Definition.—This is the disease called by Bosworth[1] "croupous tonsillitis," and by Casselberry[2] "infectious pseudo-membranous tonsillitis." It is commonly, though erroneously, known to the laity as "ulcerated sore throat," and many physicians, rather unfortunately, designate it as "diphtheritic" or "diphtheroid."

Etiology.—That this affection is the direct result of the action of micro-organisms there can be no doubt. Its clinical history and frequent epidemic prevalence confirm in this respect the results of bacteriological examination. By some authorities[3] it is maintained that the several types of tonsillitis depend on the special microbe responsible in each case. By others the variation is said to depend (1) on whether the organism affects the surface or the deeper layers of the tonsil or the subtonsillar cellular tissue, (2) on the virulence of the microbes and the resistance of the individual. This is the opinion of Viellon,[4] and seems to be more plausible. In twenty-four cases he found the streptococcus pyogenes virulens, usually associated with less virulent pneumococci and staphylococci. The germ theory of its causation accounts for the frequent origin of this disease in defective sewerage, cesspools, etc., for, as stated by Bosworth,[5] in the processes of decomposition which take place in sewers the microbe finds a field favorable for its propagation. Follicular tonsillitis is contagious, but only to a very limited degree. The most important predisposing cause is hypertrophy of the tonsils, which is found in the great majority of persons subject to recurring attacks of such inflammation. The enlarged tonsil offers an irregular and protruding surface for the reception of the germs, and, since hypertrophied tonsils are usually also diseased, a field of diminished resistance for their activity. Tonsillitis is most common in those of rheumatic diathesis, but the relation is most striking with quinsy, in connection with which this subject will be more fully considered. There is no question that errors in diet are a predisposing cause of this as well as other forms of sore throat. In many cases, especially in children, abstinence from cake, candy, pastries, and raisins decidedly lessens the liability to tonsillitis. Exposure to cold plays but a small part, if any, in causing this affection, though it frequently produces the simple superficial form of tonsillar inflammation.

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[1] Loc. cit., p. 161.

[2] American Text-Book of Diseases of Children, p. 434.

[3] Wolfenden, Journal of Laryngology, August, 1894.

[4] Sajous's Annual, 1895.

[5] Loc. cit., p. 162.

Morbid Anatomy.—Usually both tonsils are involved, and frequently the lymphoid tissue of the naso-pharynx also. In addition to the ordinary results of inflammation there is an exudate, which the microscope shows to consist of fibrin entangling in the meshes of its fibrillæ leucocytes and epithelial cells. The pathogenic organisms above mentioned are found in this exudate, as well as in the tonsillar structure. In some cases the exudate is very slight in amount, and the symptoms, especially the deep-red color, the swelling, and the tenderness, resemble the variety which some writers describe as parenchymatous; but suppuration in the tonsil itself is rare, if we except those minute and often multiple abscesses which are occasionally found in a closed crypt.

Symptoms.—The attack usually begins with a distinct chill or chilly sensations. There is fever ranging from 100° to 104° F., occasionally higher. Headache, backache, and pain in the limbs are generally present. Sore throat and difficulty in deglutition vary much in severity. In young children these symptoms are often so slight that the seat of the trouble is overlooked. Rotch[1] wisely advises that examination of the throat should be made as a routine measure in acute febrile affections of children, even when there are no symptoms to indicate disease there. On inspection the tonsils, usually already hypertrophied, appear swollen and reddened. This stage is soon followed by an exudation of varying character. It appears usually as whitish or yellowish-white deposits at the mouths of the tonsillar crypts and dotted as disseminated spots over the tonsil. A similar appearance is often presented on the pharyngeal wall, but only on its glandular structures. It is especially common in the chain of muco-lymphoid glands which is situated behind the posterior palatine fold. When posterior rhinoscopy is possible, the same affection is frequently found in the lymphoid tissue of the naso-pharynx. Indeed, I have had the opportunity in several cases to observe its beginning here and descent along the pharyngeal wall to the tonsil, as Bosworth[2] suggests is frequently the case. Occasionally a thin, pale, translucent membrane spreads over the surface of the tonsil. It lacks the tough, fibrinous character of diphtheritic membrane, and is easily wiped off. Moreover, it never extends to the anterior faucial pillar or the soft palate, as diphtheria is prone to do. Enlargement of the lymphatic glands in the submaxillary region is common. Transient albuminuria is not uncommon.[3] The fever and acute symptoms seldom last longer than three or four days, but the local inflammation and exudate may not disappear for a week or more. Cases characterized by marked swelling and redness and tenderness, with very slight exudate, sometimes persist for two weeks or longer, or they subside and return. In these cases there is often a slight fever, with fluctuations of a degree or more. Sometimes minute abscesses will be found in the follicles, and very often such cases yield best to antirheumatic treatment. When paralysis of the palate supervenes it is usually thought proof positive of diphtheria. Probably in the great majority of cases this is true, but the evidence of many competent observers seems to show that such paralysis may be a sequel of non-diphtheritic tonsillitis. Occasionally a peritonsillar abscess follows immediately on an acute follicular inflammation. Prompt treatment will do much to prevent this complication. After recovery from tonsillitis there is often decided weakness, and I have frequently observed a subnormal temperature.

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[1] Pediatrics, p. 810.

[2] Loc. cit., p. 168.

[3] Wolfenden, Bosworth, and others.

Fütterer[1] reports an interesting case of paresis of the ocular muscles, with various other symptoms, among which was severe lumbago, which he attributed to a septic tonsillitis. It does not seem to me, however, that this causal relation was well established.

Diagnosis.—In acute sore throat in children it is of great importance that the patient be separated from other children, even before a diagnosis can be made. This point is well emphasized by Dukes[2] in his experience at Rugby School.

At present the trend of opinion is decidedly in favor of the microscope as the only reliable means of diagnosis between lacunar inflammation of the tonsil and diphtheria. I agree fully with Holt,[3] however, that in at least four-fifths of all cases a certain clinical diagnosis can be made at the end of twenty-four hours from the onset of the disease, and that the remaining one-fifth is important chiefly from the danger of communicating the disease to others. In these exceptional cases the microscope is of great value. The opinion of Browne and Casselberry[4] that diphtheritic membrane almost invariably extends to the anterior faucial pillars or the soft palate is not sustained by general experience. Such extension, though pathognomonic when it occurs, is often absent, and is of more value in prognosis than in diagnosis. Holt in one hundred and nine cases in the New York Infant Asylum found the membrane to remain limited to the tonsil in twenty-seven. Our chief reliance must be on the character and situation of the membrane and the ease with which it is detached. An exudate which, twenty-four hours after its appearance, is still thin, filmy, more or less translucent, and can be easily wiped off without leaving a raw surface beneath it, is not diphtheria. Diphtheritic membrane in its very beginning may have these characteristics, but in from twelve to twenty hours it becomes thicker, tougher, whiter. The fact that the exudate is spread as a thin layer over the tonsil and is dotted along the glandular structures in the lateral fold of the pharynx should not lead to the diagnosis of diphtheria if it continues to possess the marks just described. Less likely to be mistaken are those cases of follicular tonsillitis in which the exudate consists simply of deposits scattered here and there over the tonsil at the mouths of the crypts. Koplik, it is true, claims to have found the Klebs-Löffler bacillus even in these cases. In both diseases enlargement of the lymphatic glands in the submaxillary region and at the angle of the jaw is common. As a rule, the fever and muscular aching are more apt to be severe in tonsillitis than in diphtheria, but too much importance must not be attached to this in diagnosis. Albuminuria is rather indicative of the graver malady, but occurs too often in the milder also to be of distinct diagnostic value.

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[1] Annals of Ophthalmology and Otology, vol. v. p. 479.

[2] Lancet, February 15, 1896.

[3] Diseases of Infancy and Childhood, p. 972, 1897.

[4] Loc. cit., p. 435.

Prognosis.—Favorable. Though this disease may be followed by peritonsillar abscess, and rarely by paralysis of the palate, I cannot agree with Bosworth[1] that it is ever followed by membranous laryngitis. Holt[2] is doubtless correct in saying that when this complication occurs we have had to deal with diphtheria.

Treatment.—Treatment must be general and local. Notwithstanding the opinion of Haig[3] that calomel given in tonsillitis with uric acid diathesis often, by forming the insoluble urate of mercury, produces rheumatic symptoms elsewhere, I would still commend this drug highly in the beginning of the attack. To a child three years old a grain may be given in doses of from one-eighth to one-fourth grain every half-hour. If the bowels do not act freely, it should be followed by a laxative. The fever and muscular aching are best controlled by phenacetine in doses of one or two grains every three or four hours to a child four years old. With this salophen may be combined, and is especially indicated when there is a rheumatic tendency. Tincture of iron (one part) to glycerin (eight parts) is of value both locally and constitutionally (Bosworth). These remedies I have found more effective than aconite, belladonna, or red iodide of mercury. In the convalescent stage, tonics, such as quinine, strychnine, and cod-liver oil, are often indicated.

Local Treatment.—An ice-bag to the neck is often comforting to older children, especially when there is involvement of the cervical lymphatics. Holding cracked ice in the mouth, or a hot soda-and-water gargle, is sometimes pleasant.

Guaiacol has been highly praised for its analgesic effect, as well as for its power to lower temperature and abort the attack. Raymond[4] applies it, full strength, on a cotton swab, being careful not to let any run into the larynx. He finds that combining it with cocaine rather aggravates the smarting it produces. Laurens[5] applies it combined with olive oil in the proportion of one to twenty. When the follicles are distended, if the patient is tolerant of manipulation, it is well to empty them with peroxide of hydrogen on a cotton-wrapped probe. Cleansing the surface of the tonsil by an alkaline and antiseptic spray is pleasant, and perhaps hastens cure. The same solution can be advantageously applied through the nose to the naso-pharynx.

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[1] Loc. cit., p. 174.

[2] Diseases of Infancy and Childhood, p. 974, 1897.

[3] Quoted by Chenoweth, Journal of Rectal and Gastro-Intestinal Diseases, January, 1897.

[4] Medical Record, March 24, 1894.

[5] Annales des Maladies de l'Oreille et du Larynx, February, 1896.

Prophylactic Treatment.—Most children who suffer from recurring attacks of tonsillitis have enlarged tonsils. They frequently possess that tendency to enlargement of the lymphoid structures to which the name "lymphatism" is given. Often they indulge in injudicious diet, and are improperly clothed and live in unsanitary homes. It is in these directions that preventive treatment must be carried out.

Hypertrophied tonsils should be excised; the flat and diseased one should have its crypts opened and the electro-cautery applied; adenoid growths should be removed.

For lymphatism syrup of iodide of iron, in doses of from ten to fifteen drops in water after each meal for a child four years old, is valuable. Excessive sugar, pastries, candy, tea, and coffee should be forbidden for children. Houses are more often heated too much than too little. Abundant out-door life should be insisted on. Where there are recurring attacks in certain premises the sewerage should be carefully inspected.

(*b*) ACUTE SUPERFICIAL TONSILLITIS.

In this variety of the disease the tonsil is reddened and somewhat swollen, but there is no exudate. The pharynx adjacent is involved, and sometimes the uvula is œdematous.

Etiology.—There is no proof of the action of micro-organisms here. The most common causes are exposure to cold and digestive disturbances.

Symptoms.—The attack usually begins with chills and fever, the latter ranging from 100° to 103° F. The tongue is often coated, and the bowels costive. There is pain in the throat, with difficulty in deglutition. Pain occasionally radiates to the ear, and there is sometimes a complication of otitis media.

Prognosis.—Favorable. Duration, from two or three days to a week.

Treatment.—Calomel, followed by a laxative if needed. Phenacetine or lactophenine for the pain. Ice compress to throat, and cracked ice in the mouth, and in the later stages, if the palate is relaxed, an astringent gargle.

HYPERTROPHY OF THE TONSILS.

A chronic enlargement of the tonsils, usually synonymous with chronic tonsillitis.

Etiology.—(1) As the tendency to enlargement of the lymphatic structures, known as lymphatism, to which reference has already been made, is hereditary, and as it is a predisposing cause of tonsillar hypertrophy, it is common to find several children of a family with enlarged tonsils and adenoid growths. (2) Enlarged tonsils are seldom, if ever, congenital. It is said[1] that they are not uncommon in the first few months of life. The youngest age at which I have seen sufficient enlargement to demand treatment was about twelve months. Most frequently the enlargement takes place between the third and sixth years. After puberty there is a tendency to diminution in size, but even in adults hypertrophy is not rare. (3) With the infectious diseases, such as scarlet fever, diphtheria, and measles, chronic tonsillar enlargement often begins. (4) Recurring attacks of acute tonsillitis are sometimes the cause, though more frequently the result, of chronic enlargement. They produce the variety described by Bosworth[2] as hyperplastic. (5) Debilitating influences of all kinds tend to increase the hypertrophy. McGuire[3] reports a case of tonsils so enlarged after parotitis as to demand tracheotomy. Inflammation of the tonsil secondary to that of the parotid is very rare.

Morbid Anatomy.—As a rule, both the faucial tonsils are involved, and frequently also the pharyngeal. Rarely one tonsil only is affected, and this to an enormous extent. In such cases I have occasionally seen the tonsil attached by a narrow pedicle. Knight classifies hypertrophy into (1) tonsils so decidedly increased as to interfere with deglutition and respiration; (2) flat diffused tonsils, projecting but slightly, except during retching, and often associated with rheumatism; (3) tonsils which encroach on the pharyngeal space, but do not project beyond the faucial pillars, the latter being adherent to and spread out over them, sometimes very thin and sometimes thickened. Tonsils usually become harder and more fibrous in proportion to the age of the patient. The anterior pillars are frequently adherent, and it is important that such adhesions be detached before operating. Much has been written of late concerning tuberculosis of the tonsils. Gottstein,[4] Nicoll,[5] Dieulafoy and Cornil,[6] and Mouret[7] conclude that in a small proportion of cases enlarged tonsils are tubercular, and that the tuberculosis may be either primary or secondary. I fully agree with Wright,[8] however, that "every laryngologist's observation has been wide enough to convince him that there must certainly be some mistake in the assertion that any considerable number (of enlarged tonsils) are more than simple hypertrophies of pre-existing lymphoid elements."

Symptoms.—A large group of the symptoms formerly attributed to hypertrophy of the faucial tonsils are more decidedly due to adenoid growths. Thus the obstruction to respiration, the deafness, the peculiar voice, the impaired general nutrition, and cough are all more frequently due to the post-nasal enlargement. There can be no question, however, that the faucial tonsils often contribute to these symptoms, besides producing certain effects peculiar to themselves. Cough, though not the most common, is still not a rare symptom of tonsillar hypertrophy. According to Furet,[1] cough may result from any form of tonsillar disease. It is often violent, spasmodic, and even painful, but without expectoration. He attributes it to the "tonsillar plexus," which Andersch describes as consisting of the glosso-pharyngeal, lingual, spinal, and pneumogastric nerves. Difficulty in deglutition is occasionally present, though rarely in my experience. North,[2] however, relates a case of a boy nine years old who had never been able to swallow solids. He was immediately relieved by tonsillotomy. Frequent attacks of acute tonsillitis are common. Impaired digestion and malnutrition are often seen, and it is not unusual to find such cases improve rapidly after tonsillotomy when tonics, change of air, and the best hygienic surroundings have been tried in vain. Mouth-breathing, especially at night, is the rule where the tonsils are much enlarged. In some cases it is produced by them alone, but more frequently is chiefly due to the adenoid growths with which they are associated. Examination of the crypts will usually reveal very offensive cheesy masses contained in them, and it seems reasonable to suppose, as Bosworth[3] suggests, that they may contaminate both air and food. Stirling describes three cases of bony growth invading the tonsil. He believed them to be elongated styloid processes. No surgical interference was justified. An interesting case is reported by Boulay[4] as follows. A boy twelve years old suffered for two years with nocturnal crises, characterized by sudden awakening with anxiety, tingling of tongue, loss of consciousness, and convulsions of tongue, lips, and face, and often of the four limbs; respiration was embarrassed, and asphyxia threatened, the whole attack lasting from five to ten minutes. The child had immense tonsils and adenoid growths. From the day on which the tonsils were removed the attacks ceased, and never returned. The adenoids were removed later.

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[1] North, Toledo Medical and Surgical Reporter, July, 1895.

[2] Loc. cit., p. 132.

[3] Medical News, May 14, 1893.

[4] Berliner klinische Wochenschrift, August, 1896.

[5] Glasgow Medical Journal, January, 1896.

[6] Bulletin de l'Académie de Médecine.

[7] New York Medical Journal, November, 1896.

[8] Ibid., September 26, 1896.

Prognosis.—The tendency of hypertrophied tonsils is to become smaller after puberty. Before this diminution occurs, however, irreparable harm will often have been done to the general development and nutrition, and sometimes to the sense of hearing. Most important it is to remember that enlarged tonsils increase the liability to scarlet fever and diphtheria, and augment their danger should they occur.

Treatment of hypertrophied tonsils is surgical. Medicinal applications are rarely effective, and have been sufficiently discussed in vol. ii. of this work. Tonics and hygienic directions are often indicated. The question which usually presents itself is whether operation is necessary. The classification adopted by Holt[5] is, in the main, commendable. He divides hypertrophy into (1) tonsils so large that they nearly meet in the median line,—operation always indicated; (2) tonsils projecting not more than one-fourth inch beyond the pillars,—operation rarely necessary; (3) intermediate cases,—operation must be determined by the presence of symptoms, such as tonsillitis, cough, or impaired hearing. As a rule, I should say that where the tonsils project more than one-fourth inch beyond the pillars excision is advisable. We must remember, too, that tonsils may be hypertrophied without projecting beyond the faucial arch. The treatment of these cases will be discussed later.

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[1] La Presse Médicale, May 13, 1896.

[2] Toledo Medical and Surgical Reporter, July, 1895.

[3] Loc. cit., p. 137.

[4] Journal of Laryngology, July, 1896.

[5] Loc. cit., p. 273.

Method of Operation.—It is well to have at hand a saucer of cracked ice and a bottle of McKenzie's tanno-gallic mixture (tannin, six drachms; gallic acid, two drachms; distilled water, one ounce; shake and sip, for hemorrhage). It is advisable to have also a long pair of dressing-forceps or an applicator, such as is figured on p. 479 of vol. ii. A piece of oil-cloth should be pinned around the child's neck and around its arms and body. A few children will submit quietly to the operation, but usually the child should be seated in the nurse's or father's lap, who takes its legs between his knees and holds his arms around its body with the back of the child's head against his shoulder. An assistant, standing behind them, steadies the head. The patient should be seated, not facing the light, but with his back towards it, and the surgeon should reflect the light into the throat with the head-mirror. As a rule, no gag is necessary. I usually introduce a tongue-depressor, but this may be, and in very small throats should be, dispensed with. Ordinarily, Mathieu's tonsillotome is the best instrument, and it is well to have at least two, or, better, all three, sizes. In rather flat, soft tonsils the McKenzie instrument is often preferable. With the patient seated as described, and with the mouth opened, if need be, by holding the nose or pressing in the cheeks between the teeth, the surgeon reflects the light into the child's mouth. The guillotine is introduced flat into the throat, and turned to the vertical position when the tonsil is reached. Care should be taken to carry it well down to the lower end of the tonsil, to avoid cutting the anterior pillar (which should be previously detached, if adherent), and to press it firmly to the side so as to cut off the tonsil even with or deeper than the pillars of the fauces. It is rarely necessary to press at the external angle of the jaw. The most common accident is to wound the anterior pillar. This increases the pain and hemorrhage at the time of operation and the subsequent sore throat. It is said that the uvula has been sometimes caught in the loop and partially amputated. This would seem to be due either to insufficient illumination or to lack of dexterity. In children it is best to remove both tonsils at the same sitting, but operation on the second should not be undertaken till bleeding from the first has ceased. The technique described is the one I find most satisfactory, but some surgeons prefer the snare, and some recommend excision of the entire tonsil. Marcel,[1] after an experience with it of five hundred cases, prefers the cold snare. In hard tonsils (relatively rare in children), Wilson[1] uses the hot snare. Coulter,[2] Loeb,[3] and North[4] advise removal of the whole organ, believing that only by this means is the tendency to tonsillitis cured. North considers the danger from hemorrhage less after complete than after partial excision. It cannot be denied that in some cases, even in children, hemorrhage may be alarming.

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[1] Jahrbuch für Kinderheilkunde, 1895.

Barkan[5] reports a death from secondary hemorrhage twelve hours after removing the faucial tonsils with guillotine, and the pharyngeal with curette, in a strumous boy six years old. Jessop[6] describes a case of secondary hemorrhage occurring four days after operation on a child ten years old. The patient was blanched and almost speechless. He first injected brandy, raised the legs, and placed hot bottles in the bed. Sucking ice checked the bleeding for a time. He then scrubbed off the tonsil with absorbent cotton, and held against it a pledget saturated with perchloride of iron. He attributed the hemorrhage to a slough from an unhealthy wound. I have myself[7] reported a case of secondary hemorrhage in a little girl, aged eight years, three days after removing tonsils. At the meeting of the American Laryngological Association in 1895, Dr. J. W. Farlow[8] exhibited an écraseur for the removal of tonsils in adults, and in children when adenoids are to be operated on at the same sitting. He wisely suggested that in delicate children the loss of even a small quantity of blood may be very undesirable. In the discussion that followed, Daly, Shurly, Casselberry, and Ingalls agreed that serious hemorrhage, even in children, is not very rare, and that practitioners may place too much reliance on the guillotine as a means of averting it.

In adults often, and in children occasionally, we meet with cases of hypertrophied tonsils which are flattened and deep-seated in the pillars of the fauces and cannot be made to project beyond them. It is this variety in which Knight, quoted above, often finds a rheumatic factor. The best local treatment is to detach adherent pillars, to open up the tonsillar pockets and crypts, and to make a number of incisions with the electro-cautery.

PERITONSILLAR ABSCESS.

Synonyms.—Quinsy, Suppurative tonsillitis.

This disease is relatively rare in children, and therefore will be rather briefly considered.

Definition.—A phlegmonous inflammation in the tissues around the tonsil, usually proceeding to suppuration.

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[1] New York Medical Journal, vol. lxiii. p. 509.

[2] Ibid., November 21, 1896. [3] Ibid.

[4] Toledo Medical and Surgical Reporter, July, 1895.

[5] Occidental Medical Times, March, 1894.

[6] British Medical Journal, January 3, 1894.

[7] American Practitioner and News.

[8] New York Medical Journal, November 16, 1895.

Etiology.—The most important predisposing causes are the rheumatic diathesis and enlarged tonsils. These influences explain the hereditary tendency to quinsy. As to its relation to rheumatism, there is some difference of opinion. Thus, Hare[1] declares that "if there is a causal relation between the two diseases, tonsillitis is most likely to stand as the first link in the chain." This is also the opinion of Sacaze.[2] He relates a case in which inflammation of a wrist, the subject of former injury, followed a tonsillitis, and, as he believed, was due to infection transmitted from the tonsil. Sir Willoughby Wade[3] also advocates this theory, and considers tonsillitis a "primary infective disease of the lacunæ; rheumatic fever a secondary disease from absorption of micro-organisms or their products." Wagner[4] was confirmed in this belief by finding in the synovial fluid and urine germs identical with those found in the tonsil, which he looked upon as the point of invasion. Notwithstanding these authorities, however, the facts (1) that quinsy is of such frequent occurrence in rheumatic families; (2) that it often attacks persons subject to rheumatic affections at other times without being itself followed by rheumatic symptoms; and (3) that antirheumatic treatment is of pre-eminent value and, used promptly, is often prophylactic, tend to support the generally accepted belief that rheumatism is the underlying cause. The direct cause of the suppuration has been found by Logucki[5] to be streptococci and staphylococci, the latter predominating in tedious cases. It is especially a disease of early adult life. Casselberry[6] states that three per cent. of the cases occur under ten and six per cent. under fifteen years of age. In adults it often returns several times a year, constituting a quinsy habit.

Pathology.—My experience confirms that of Logucki,[7] that abscess situated between the tonsil and the posterior pillar is of longer duration and of less acute symptoms than when the collection is beneath the anterior pillar. The pathogenic micro-organisms found in the pus have been already mentioned.

Symptomatology.—The onset may be insidious, beginning as a slight sore throat, but more frequently it begins with a decided chill, and with fever ranging from 102° to 105° F. The *pain*, often involving all that side of the neck and *radiating to the ear;* the *dysphagia*, often intense; the *regurgitation of fluids* through the nose; the *thick speech;* the *swelling* and *tenderness* at the *angle of the jaw* on the side affected; the *difficulty in opening the mouth;* the *offensive breath;* the *stringy, tenacious mucus* in the pharynx and naso-pharynx; the *bulging* either of the anterior portion of the soft palate or of the posterior pillar backward and downward; the

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[1] Quoted by Chenoweth, Journal of Rectal and Gastro-Intestinal Diseases, January, 1897.

[2] Le Bulletin Médical, July, 1894.

[3] Gaillard's Medical Journal, August, 1896.

[4] New York Medical Journal, No. 830, 1894.

[5] Archives of Laryngology and Rhinology, vol. ii. p. 244.

[6] Loc. cit., p. 437.

[7] Loc. cit.

œdema and *elongation of the uvula*, most marked when suppuration is imminent or has taken place, and sometimes causing *cough* and *distress in breathing*; the *sallow color*, and the *anxious countenance*, constitute a typical group of symptoms. Attacks vary much in severity and duration, but, as a rule, it is from six to eight days from the beginning of the attack till suppuration is so evident as to indicate incision. The tendency is to point a little above and to the outside of the tonsil. An abscess in the tonsil proper and opening on its surface is a rarity, unless we speak of those minute abscesses, often multiple, due to the closure of a crypt.

Prognosis.—Favorable. The possible dangers have been sufficiently elaborated on p. 451, vol. ii., of this Cyclopædia.

Treatment.—There is usually constipation, and it is well to begin with a good dose of calomel and follow it by a saline laxative. The most valuable remedies are the preparations of salicylic acid. Newcomb,[1] from an analysis of one hundred and sixty-nine cases in which salol, salicylate of sodium, and guaiac were tried, concludes that salol gives most prompt relief. This has been my experience also, but more recently I have been using salophen, and I think it less likely to produce disagreeable results than salol. To an adult I give ten grains of salophen and four grains of phenacetine every four hours, and to a child in proportion. Lactophenine in similar or somewhat smaller doses is a highly praised recent remedy. If salol is used, the urine should be observed, and the dose diminished when it becomes slightly greenish or olive-colored. An alkaline antiseptic spray (*e.g.*, of Seiler's tablets) to the nose and hawked out through the naso-pharynx is often helpful in removing the tenacious mucus. An opiate may be demanded to procure sleep at night. Guaiac, either as a lozenge or as the ammoniated tincture, is valuable. The application of a paste of bicarbonate of sodium to the tonsil has been highly recommended, but I have found it useless. Hot or cold applications to the neck, and ice or hot water in the mouth, are sometimes grateful. It seems almost incredible that a quinsy can be aborted by application of a three per cent. solution of cocaine, as claimed by Fox.[2] It is curious to note that the same statement was made by Haviland Hall[3] so long ago as 1888. Fluctuation should be carefully watched for, and as soon as it is evident an incision should be made with a bistoury wrapped with absorbent cotton or adhesive plaster to within half an inch of its point. Bitter tonics and iron are often indicated in the convalescence.

CONGENITAL SYPHILIS OF THE PHARYNX.

This disease is rare, and yet not so rare as is supposed. John McKenzie[4] truly says that "a great deal of confusion prevails concerning this question, in consequence of the persistent adherence to the old superstition which regards this affection as of 'scrofulous' origin. It is impossible to exaggerate the *rôle* of congenital syphilis in the production of the deep, destructive pharyngeal ulceration of childhood."

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[1] Journal of the American Medical Association, December, 1892.

[2] Lancet, No. 6, 1896.

[3] British Medical Journal, vol. i. p. 1060.

[4] Morrow's System of Genito-Urinary Diseases, etc., p. 332.

There may be either mucous patches or deep ulceration; the latter is the more characteristic. The disease may appear at any age between the first few weeks of life and the period of puberty. Quite frequently it appears about the twelfth year, and it may be the only expression of the inherited diathesis. Females are attacked oftener than males. The most common seat of ulceration is the posterior portion of the hard palate; next in frequency the fauces. The tendency is to rapid caries and necrosis. The ulcer is deep, ragged, and often surrounded by a thickened infiltrated area. Ulceration of the tongue from congenital syphilis is very rare.

Diagnosis.—The appearance of the ulcer, the age of the patient, and the absence of any acute systemic disease, such as diphtheria or typhoid fever, scarcely leave room for doubt. Of course the history of syphilis in a parent is valuable when it can be obtained, but failure to elicit it, for various reasons, is common, and does not add much to the difficulty of diagnosis. Very frequently there are associated other evidences of syphilis, as, for instance, interstitial keratitis, cicatrices in the skin, and notched teeth. As to this last symptom, however, Morell Mackenzie calls attention to its frequent absence where the throat is affected.

Prognosis.—This is said to depend on the age at which the disease appears and on the prompt recognition of its true character. The earlier ulceration occurs the graver the case.

"Pharyngo-laryngeal ulceration occurring within the first year is almost invariably fatal" (John McKenzie). As illustrating the gravity of this disease, even in older children and under most vigorous treatment, I may cite the following case. A girl of about fourteen, belonging to the upper ranks of society, was brought to me by her uncle for treatment. Her father and mother were both dead, the former, I was told, having died of a syphilitic affection. The child at the time of her visit to me was absolutely deaf, all communication with her being carried on by talking on the fingers or by writing. Her sight was greatly impaired from a former inflammation of the cornea and iris, the evidences of which remained in opacities of the cornea and an irregular pupil. In her throat there was a deep, ragged ulcer, involving the left anterior pillar, extending laterally down the pharynx, with some ulceration of the larynx. I was informed that she had lost her hearing at the time of the inflammation in her eyes, when she was about eight or nine years old; that she had from the first been under the most active antisyphilitic treatment, administered by specialists of high repute, in conjunction with her family physician. Tonics and a residence in the country were also tried with but slight improvement. This young lady was under my observation for several years. Various forms of specific medication and general tonics were administered; but, in spite of all, the ulceration would frequently break out anew, and towards the last produced such a degree of cicatricial stenosis of the larynx as to leave an opening not large enough to admit an ordinary lead-pencil. She died in her eighteenth year, seemingly of malnutrition and debility.

Such instances are, fortunately, rare. They illustrate the fearful ravages of inherited syphilis of the most obstinate type.

Treatment.—Treatment must be carried out on the same principles that guide us in syphilis elsewhere. In the deep ulceration our chief reliance must be on iodide of potassium. Mercury is valuable, I think, in rather small tonic doses, long continued. Of almost or not quite equal importance is the building up of the general health with cod-liver oil and iron, good diet and fresh air, and, where practicable, a season at the seashore. Locally, for the mucous patches, nothing is superior to the stick of nitrate of silver, and for the deep ulcers, iodoform. The treatment of adhesions is rarely satisfactory, and, unless the results are serious, should not be attempted. The tendency to recurring stenosis is great. In cutting through a cicatrix in the naso-pharynx a catheter should be introduced through the nose as a guide. Delavan found that the application of monochloracetic acid to the raw surfaces prevented their reunion, even though they remained in contact.

We owe to John McKenzie much of our knowledge of this important affection, and I have taken the liberty to quote freely from his article on the subject in the text-book above mentioned.

ELONGATION OF THE UVULA.

This condition generally attends some other disease of the pharynx. It is often found in follicular pharyngitis, and it is aggravated, if not induced, by the efforts at hawking in naso-pharyngitis, and sometimes in adenoid growths. It frequently exists with the general relaxation of acute pharyngitis. I cannot agree with Bosworth,[1] that in most instances it is primarily a congenital affection, but think, with Lennox Browne, that it is to be attributed to the same causes as acute pharyngitis. It is relatively rare in children. The two most conspicuous symptoms are tickling in the throat, with the sensation of a foreign body there, and cough. Very frequently these annoyances are most marked on lying down. There is occasionally a distressing spasm of the glottis, and there may be impairment of the voice. Browne calls attention to morning nausea. I have seen this strikingly illustrated in a girl about thirteen years old. For weeks she had suffered from sick stomach on rising, and had vomited her breakfast. There was no cough or other symptom referable to the throat, and yet the clipping of an elongated uvula gave immediate and permanent relief. The diagnosis would appear simple, and yet, because of a considerable variation in the normal length of the uvula, and because of the tendency to gag which often characterizes these cases from over-irritability of the pharynx, it is not always easy. The average length of the uvula in the adult is not quite half an inch. The inclination of most patients on the first examination of the throat is to hold the breath, distort the face, and draw up the palate. Thus we are prevented from observing the throat in its natural position, and cannot tell whether at rest the uvula hangs on the tongue. The patient should be instructed to breathe quietly and to let the face be in repose. The tongue should be depressed just enough to allow a thorough view. We often find then that the uvula rests on the tongue, and in many cases its mucous membrane hangs as a tip below the organ proper, constituting the condition known as "stripped uvula."

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[1] Diseases of the Throat, p. 93.

Treatment.—Of course the cause should be first removed. In recent cases an astringent gargle is often sufficient. I question whether anything is better than a solution of tannin in glycerin and water,—say thirty grains of tannin, half an ounce of glycerin, and water enough to make three ounces. When the elongation persists, or returns frequently, uvulotomy should be practised. Only enough should be removed to reduce the uvula to the normal length. A five per cent. solution of cocaine should be carefully applied with a cotton-wrapped probe two or three times; then, after a few minutes' delay, the tip of the uvula should be caught with long forceps held in the left hand and drawn gently forward; the scissors, which should be long, stout, and blunt-pointed, are then directed so that the cut surface looks upward and backward. By this manœuvre the raw surface is not irritated by the passage of food. Care should be taken not to exert much traction on the uvula, for by so doing too much mucous membrane will be removed and an ugly and rather painful stump left to heal. A few instances of alarming hemorrhage have been reported, but they are so exceedingly rare that the danger of this complication need not be considered. There is often a good deal of difficulty in swallowing for a few days, together with pain which shoots up towards the ears. Holding cracked ice in the mouth is helpful, and, when the dysphagia is decided, the family or patient may be instructed how to apply a little of a four per cent. solution of cocaine to the raw surface just before eating.

DEFORMITY AND DEFECTIVE INNERVATION OF THE UVULA.

A certain interest has recently been aroused in this subject by an article by Dr. Charles L. Dana in *The American Journal of Insanity* of April, 1896. In an examination of one hundred and eight insane patients Dr. Dana found almost fifty per cent. with a uvula subject to deformity or defective innervation, while in one hundred normal persons the examination was entirely negative. He concludes that "a uvula twisted to one side, and not innervated, forms an anatomical and physiological stigma of degeneration."

Somers[1] also reports an interesting case of bifid uvula with degeneracy. He quotes Berens as having, in an investigation of three thousand throats, found the uvula abnormal in only eighty-four cases, fourteen of which were deeply cleft.

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[1] New York Medical Journal, November 21, 1896.

STENOSIS AND TUMORS OF THE LARYNX.

By WILLIAM K. SIMPSON, M.D.

STENOSIS OF THE LARYNX.

INASMUCH as the following articles on stenosis of the larynx and laryngeal growths are supplemental in their nature, the reader is referred to the original work (vol. ii.) for many of the salient points. In these supplementary articles the writer will endeavor to consider only the newer aspects of the subjects, and will avoid any repetition of the original except where it is deemed necessary to more clearly emphasize the points under consideration and to preserve a continuity of context. The article on stenosis of the larynx will refer to the chronic type in contradistinction to the acute type as represented by diphtheritic or membranous croup.

The dividing line in the description of the same morbid conditions as they exist in children and adults is often a matter of great nicety to determine, and any consideration will naturally partake somewhat of repetition; and, although there are many diseases peculiar to childhood alone, nevertheless there are many which appear both in children and adult life, varying only in their characteristics and severity commensurate with the difference of development and susceptibility in the two ages.

The infantile larynx being such a very small organ in proportion to the importance of its vital function, any marked malformation or constriction shows itself very readily in interference with its function of respiration, and it is for this reason undoubtedly that we see so few instances of congenital deformities of the larynx. This fact may also account for many of the sudden suffocative deaths shortly after birth.

The comparative rarity of congenital laryngeal deformity is further demonstrated by the testimony of those who have had large experience with intubation for acute stenosis in children. Here the educated finger quickly determines any deviation from the normal contour, especially in the upper portion of the larynx, while any obstinate impediment to the passage of the tube below would probably indicate stricture in the subglottic region.

It is a noteworthy fact that the landmarks for our guidance in intubation in children are seldom obliterated by congenital deformities. Aside from tumors, the principal causative factors in congenital stenosis are hereditary syphilis, tuberculosis, external glandular enlargements, and arrest of development. These may find their immediate expression in tumefactions, ulcerations, abscesses, loss of substance, paralysis, spasm, and external pressure.

J. H. Bryan, of Washington, D.C.,[1] reports a case of laryngismus due to congenital valvular formation of the upper orifice of the larynx. The child was subject to tonic laryngeal spasm from the time it was two weeks old. The epiglottis was found to be irregular in outline and bent backward over the laryngeal cavity; spasm was attributed to a binding of the epiglottis, causing the aryepiglottic folds to come almost in apposition, producing a slight stridor during inspiration; the child had phimosis, and was fed on cow's milk. On diluting the milk and relieving the phimosis, the respiration lost its spasmodic character and became normal.

Meinhard Schmidt, of Cuxhaven,[2] reports a case of stenosis from congenital compression of the sides of the epiglottis. The stridor was violent soon after birth. The child lived seven months. The epiglottis was found bent so that the two lateral sides were strongly in apposition, causing the laryngeal aperture to resemble the figure 8, leaving a small breathing opening back and front.

Through the courtesy of Dr. John O'Dwyer, of New York, the author recently examined a child four months old who had had noisy and restricted laryngeal breathing since birth. The examining finger detected a bound-down and distorted epiglottis and adherence of the arytenoid cartilages. The condition was relieved by the passage of sounds and forcible divulsion by means of the finger.

Deformities of the epiglottis are considered by some as among the common forms of congenital malformation.

H. Ashley[3] reports a case of tubercular laryngitis in a child of twenty months, with urgent laryngeal dyspnœa, for which intubation was successfully employed. The child died six weeks later from measles. Autopsy showed tubercular ulcerations in the larynx.

Emil Müller[4] reports the sudden death of a child five months old, due to pressure of a large thymus gland and the escape of milk into the air-passages, filling the larynx, trachea, and bronchial tubes.

Rhemdorff[5] reports a fatal case of tubercular laryngitis with stenosis in a child thirteen months old.

Clegg,[6] of Liverpool, reports a case of congenital respiratory obstruction in an infant four days old, from swelling of a nævus at the base of the tongue. Tracheotomy was performed, and death occurred thirty-six hours later from a complicating pneumonia.

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[1] Lancet, London, October 3, 1891.

[2] Centralblatt für Chirurgie, Leipzig, August 3, 1892.

[3] British Medical Journal, April 25, 1891.

[4] Gaz. Méd. de Strasbourg, April 5, 1890.

[5] Jahrbuch für Kinderheilkunde, Leipzig, Bd. xxxiii., 1891.

[6] British Medical Journal, London, January 9, 1892.

Chiari, of Vienna,[1] reports a case of hereditary syphilis in a boy four and a half years old. There was severe dyspnœa for six months; the laryngeal mucous membrane was reddened and thick below the vocal cords; the right arytenoid cartilage was very thick, adducted, and stationary; the right vocal cord was deeply ulcerated in the middle third. Mercurial treatment was used and tracheotomy performed on the third day after being seen. The canula remained in seven weeks, at which time, under mixed treatment, the larynx became normal.

Other than the immediate onset of symptoms due to hereditary or congenital causes, the urgent symptoms may be delayed for an indefinite period, and we may have later stenosis from additional causes, among which may be mentioned primary œdema, stenosis associated with acute laryngitis, acute infectious diseases, the exanthemata, phlegmonous diseases, rachitis, Bright's disease, traumatisms, spasm or paralysis from central disease, pressure reflex from nose, naso-pharynx, or pharynx, and malnutrition, stenosis from foreign bodies, and also stenosis due to retained tracheal canulæ and from pressure effects of intubation.

Acute primary œdema in children is a comparatively rare affection. F. Barjou[2] considers it as an infectious disease, stating that streptococci and pneumococci have been found in a number of instances, and also giving the opinion that cold and traumatisms are but occasional causes favoring the penetration of germs within the organism.

Felix Peltesohn[3] reports only 8 cases of acute œdema in a total number of 5161 throat cases treated in his clinic, and none of them in children. In the report of 3887 autopsies, œdema of the larynx was noted in 210 instances,—149 in men, 40 in women, and 21 in children. Of the whole number, 44 cases occurred in regional and 166 in systemic disease.

Fasano[4] reports a severe case of stenosis in a boy of ten years, occurring a few hours after an attack of erysipelas.

Dr. de Scroizelles[5] reports instances of stenosis in children from simple laryngitis.

Vladimir A. Padütcheff[6] reports a case of intense œdema of the ary-epiglottic folds and of the epiglottis suddenly developing in a boy of eleven years, who was ill with measles. Tracheotomy was performed.

Boris I. Kotelausky[7] also reports a case of stenosis occurring one week and a half after an attack of measles in a boy of eleven.

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[1] Archiv für Kinderheilkunde, Stuttgart, Bd. xv., 1893.

[2] Gazette des Hôpitaux, Paris, May 19, 1894.

[3] Berliner klinische Wochenschrift, November 4, 1889.

[4] Archiv für Gynäkologie, Berlin, July, 1894.

[5] Jour. de Méd. et de Chirurg., 1887.

[6] Journal of Laryngology, London, January 24, 1892.

[7] Centralblatt für Laryngologie, 1887.

Contrary to the prevalent opinion that œdematous laryngitis is common in the course of Bright's disease, Dr. de Havilland Hall considers it to be quite rare. Sir Morell Mackenzie in a report of two hundred cases of Bright's disease found no œdema. Dr. George Johnson[1] states that with a very large experience he never saw a case of œdema of the larynx with Bright's disease.

Whipham and Delépine[2] report a fatal case of stenosis occurring in a boy fourteen years of age. In the discussion there was some doubt thrown on the diagnosis, the opinion being advanced that it was a coexisting lesion of syphilis and tuberculosis.

Dr. C. Lewis[3] reports a case of abductor paralysis of the right vocal cord, in a child of eight years, from acquired syphilis.

Strauss,[4] of Berlin, in a study of late syphilis in children, reports three cases only as having occurred in eight years in the clinic of B. Baginsky of over two thousand patients yearly, thus indicating a comparative rarity. These occurred in a girl of seven years, a girl of twelve years, and a boy of twelve years, and are fully reported.

Though the reported cases may be rare, I consider it necessary always to keep syphilis in mind as a cause in progressive stenosis, as the timely administration of mercurials and iodides may avert a more serious condition.

The relation between rachitis and laryngeal spasm in children is emphasized by P. Masucci.[5] He confirms the opinions of others in this relation, and reports the presence of rachitis in sixteen out of twenty cases of glottic spasm noticed by himself.

Dr. Thesch, of Frankfort,[6] considers improper nourishment and hygiene a most potent factor in the production of glottic spasm in children.

William Squire, of London; Kossowitz, of Vienna, and Bull[7] give very positive evidence of this relation.

Foreign bodies may cause stenosis primarily from their mechanical presence and from the secondary inflammatory reaction, tumefaction, granulations, or abscesses.

G. H. R. Holden[8] reports a case of a child fourteen months old who drew a hook from his mother's dress into his larynx. Symptoms came on twenty-two hours afterwards; recovery followed removal after laryngotracheotomy.

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[1] Journal of Laryngology, London, January 24, 1892.

[2] British Medical Journal, March 16, 1889.

[3] Glasgow Medical Journal, 1887.

[4] Archiv f. Kinderheilkunde, Stuttgart, Bd. xiv. H. 1, 1892; Journal of Laryngology, September, 1892.

[5] Archivo Italiano di Pediatria, Naples, vol. xii., No. 2.

[6] Berliner klinische Wochenshrift, 1887.

[7] Annual of the Universal Medical Sciences, 1895, vol. ii.

[8] British Medical Journal, March 12, 1892.

West[1] reports a case of a gill-plate of a herring lodged in the larynx of a child nine months old. Removed several days after a preliminary tracheotomy.

Desvernine[2] reports a case of a pin in the larynx of a child four months old, producing a fibrinous cast of the larynx and trachea. Recovery after tracheotomy.

Cases illustrating the tolerance of the larynx to foreign bodies are reported by Skinner.[3] A child twelve months old coughed up an orange-seed after forty-six days' retention in the larynx.

Helen S. Childs[4] reports a case of a clove being expelled after a residence of five weeks in the larynx of a boy five years old.

E. de Pradel[5] reports a case of pseudo-whooping-cough in a child of five years, who, after two months' retention, coughed up a pebble, with cure of spasmodic symptoms.

Symptoms.—The subjective symptoms of progressive laryngeal stenosis in children are hoarseness, cough of a croupy character, and dyspnœa on exertion, which are at once sufficient to draw attention to the larynx. These symptoms, with occasional spasm of the glottis, precede the more constant and well-known inspiratory stridor and accessory respiratory muscular action of a permanent and dangerous stenosis. It must always be borne in mind that severe laryngeal spasm may at any time complicate a stenosis of whatever nature.

The means at our disposal for detecting the nature of the stenosis are the laryngeal mirror, digital examination by the index finger, transillumination of the larynx, forced laryngoscopy,[6] and by the aid of the Röntgen rays.

A thorough examination of the pharynx is also often very necessary for the detection of any lesion that may originate or extend into the pharynx.

The mirror, when it can be utilized, affords the best means of detection. In grown children this can, as a rule, always be accomplished; in younger children the difficulties of its use are necessarily greater, but with expertness and care, even in very young children, a satisfactory examination may be made, a glance being often all that is necessary to the practised eye. Cocaine applied to the fauces greatly aids in the tolerance of the mirror. Aside from "gagging," the great tendency to the generation of mucus, and even vomiting, may render the examination impossible, and we may have to resort to narcosis; chloroform should be preferred. Under narcosis the tongue must be well drawn out. The epiglottis may have to be depressed, and the accumulation of mucus must be constantly wiped away.

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[1] Lancet, London, April 9, 1892.

[2] Revista de las Ciencias Médicas, Habana, September 5, 1889.

[3] British Medical Journal, May 7, 1892.

[4] Doctors' Weekly, New York City, February 6, 1892.

[5] La France Médicale et Paris Médical, Paris, April 5, 1895.

[6] See article on Autoscopy, p. 600 of this volume.

Walter F. Chappell, of New York,[1] refers to a method of administering belladonna to the extent of dryness of the fauces, followed by opium, in order to facilitate examination and endolaryngeal operations in children. He used it successfully in removing papilloma in three children, two, three, and three and a half years old.

Digital examination by the index finger is a very valuable means of detecting superior stenoses, and should be practised when the mirror cannot be used; it also serves to locate any pharyngeal cause of obstruction. Intubation has taught that much can be accomplished by such examination.

Electric transillumination by L. Voltolini's[2] method, and modified by Freudenthal,[3] may be of service in certain instances.

Skiagraphy (X-rays) will undoubtedly become an efficient means of examination, especially in detecting the location of foreign bodies and the more dense varieties of tumors. J. Walker Downie[4] reports a case of detection and removal of a pin in a young man nineteen years of age with the assistance of skiagraphy.

Escat[5] describes a special instrument for laryngoscopy on young and frightened children: "The instrument is conceived on the principle of a retractor, and serves, by pulling forward the base of the tongue, to open the pharynx antero-posteriorly." A minute description follows.

The differential diagnosis between laryngeal and pharyngeal respiratory obstruction is of great importance. Tonsillitis, faucial diphtheria, lingual swelling, or any acute or chronic inflammatory processes of the pharynx may give rise to obstructed breathing. Perhaps the most common chronic condition requiring differentiation is post-pharyngeal abscess. The prominent points characteristic of pharyngeal obstruction are difficulty of swallowing, thick (pharyngeal) voice, and open mouth. These with the aid of pharyngoscopy and digital examination will determine the exact character.

George M. Swift, of New York,[6] refers to slight narcosis by chloroform in order to differentiate between laryngeal spasm and obstruction from other causes: the former will give way under chloroform, while if due to obstruction the symptoms will continue.

Sajous and Massei[7] point to the great necessity of differentiating between sub- and infra-glottic stenosis in order to intelligently direct proper treatment.

The prognosis of laryngeal stenosis must, of necessity, depend entirely on the cause of the obstruction and the amenability of that cause to treatment, bearing in mind always that all progressive obstructive lesions are very prone to acute exacerbation, either of spasm or acute inflammatory action, thus increasing the danger of an unfavorable prognosis. It must also be borne in mind in the case of chronic stenosis that one can breathe through a very small laryngeal space as long as that space remains unencroached upon.

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[1] New York Eye and Ear Infirmary Reports, January, 1895.

[2] Monatsschrift für Ohrenheilkunde, November, 1888.

[3] Medicinische Monatsschrift, November, 1889.

[4] Glasgow Medical Journal, November, 1896.

[5] Journal of Laryngology, Rhinology, and Otology, London, March, 1897.

[6] New York Medical Journal, April 15, 1893.

[7] Annual of the Universal Medical Sciences, 1896, vol. iv.

Treatment.—Aside from treatment of the constitutional and extra-laryngeal causes upon which the stenosis may primarily depend, treatment is also called for against the immediate attack, and, secondly, with the view of permanently overcoming any chronic resultant intra-laryngeal changes which keep up the progressive stenosis.

Where spasm is a prominent feature under the first heading, there is nothing better for immediate and lasting effect than some form of opium or morphine, limiting the dose according to the age of the child and the effect produced. Chloroform may also be used for an immediate effect.

To ward off an anticipated attack of spasm, full doses of bromide of sodium and chloral hydrate may be administered, but their effect is too slow for immediate use.

Kurt[1] reports a case of chronic tonic spasms in a girl six years old, a sequel to whooping-cough, cured by irritation of the conjunctivæ and nasal mucous membrane, the theory being that irritation of quiescent branches of a nerve will control reflection and contraction in other branches. He used a feather dipped in a mixture of quinine and sugar.

For acute œdema, either primary or of the nature of a complicating acute inflammatory stenosis, the external application of cold by means of the ice-bag or ice-water coil is one of the best means at our disposal. Where there is much dry mucous secretion, the careful inhalation of steam, either plain or impregnated with sedative substances, may be used, but its indiscriminate use must be guarded against.

A. H. Buckmaster, of New York,[2] reports the successful use of injection of hot water (120° F.) against the œdematous parts, thereby averting a tracheotomy.

Betz,[3] in a case where tracheotomy was refused in a child eighteen months old, with croup, reports a cure with the assistance of inhaling every fifteen minutes three drops of a mixture containing ether sulphate, three parts; acetic ether, one part; menthol, one-tenth part.

In older children, or where access may be had to the larynx, scarification may be employed in suitable cases; but this, with all other principles of interference, must depend on our ability to employ them.

If the stenosis persists, tracheotomy or intubation must necessarily be performed. The choice of operation will somewhat depend on our knowledge of the character of the stenosis, but preferably the latter if a competent intubationist be at hand, resorting to tracheotomy in case of failure of intubation.

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[1] Medical Press and Circular, London, June 3, 1891.

[2] New York Medical Journal, 1887.

[3] Medical World, Philadelphia, March, 1893.

Dr. Gläser,[1] of Hamburg, reports an instance of cure by prophylactic tracheotomy in a rachitic child one year old who had violent spasms of the glottis.

For overcoming permanent or progressive stenosis some means of intra-laryngeal pressure must be invoked, and, when it can be accomplished, there is no better method than intubation as originated by Dr. O'Dwyer, of New York. The introduction of intubation has undoubtedly revolutionized the subject of intra-laryngeal dilatation. By its use we can at the same time secure sufficient breathing space and permanent dilatation, and the tube can be worn for an indefinite time, thus doing away with the necessity of oft-repeated and transitory dilatation by the older methods, and in the great majority of instances preventing the necessity of a previous tracheotomy. It may undoubtedly be necessary in certain very close, dense, and irregular strictures to perform tracheotomy both for immediate relief and to allow of dilating the stricture from below, or some intra-laryngeal cutting operation for the liberating of cicatrices or webbed-like tissue, after which intubation may be performed for its permanent effect.

Recent literature records the very successful use of intubation in relieving not only the stenosis primarily originating in the larynx, but also the secondary stenosis due to wearing the tracheotomy tube, for whatever cause it may have been employed, and where it is desirable to dispense with its permanent use. It is well known that in certain cases of tracheotomy, especially when performed high up, it becomes necessary, for various reasons, to retain the canula for an indefinite period, and the longer it is retained the greater is the danger of its permanent use becoming indispensable, resulting in loss of development, intra-laryngeal atrophy and ankylosis from disuse of the organ, and also producing a secondary stricture from more or less closure of the larynx at the superior aspect of the tracheotomy wound. In all cases of tracheotomy in children we should endeavor to dispense with the canula at the earliest moment, and, if practicable, to anticipate the possibilities of its continuance by resort to intubation. The technique of intubation and shape of tubes for chronic stenosis are, in the main, the same as those employed in croup, varying according to the demands of the individual case. For the details of intubation the reader is referred to the article on the subject.

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[1] Wiener medicinische Wochenschrift, 1887.

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F. E. Waxham, Journal of the American Medical Association, October 22, 1892.

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Charles H. Knight, New York, Boston Medical and Surgical Journal, July 7, 1892.

TUMORS OF THE LARYNX.

The subject of tumors of the larynx is so thoroughly treated in the original work that no attempt will be made in the supplementary article, other than to illustrate or emphasize some of the leading points previously considered by the reports of cases and opinions bearing on the various aspects of the subject. The principal innovation during the time elapsed is the treatment of papillomata by the intra-laryngeal pressure effect of intubation.

Desvernine[1] reports an instance of nine multiple congenital papillomata in a scrofulous child of seven years, cured by tracheotomy and removal of the growths.

Rees[2] reports a very interesting and unusual case of congenital laryngeal papilloma in a child of ten and a half years, associated with ordinary warts on the hands. They resisted all treatment, including astringents, cauterizations, and evulsion.

Gougenheimer[3] reports congenital confluent papilloma in a child of five years with excellent general health. Tracheotomy and crico-thyrotomy were performed, but growth recurred in spite of electro-cautery at the point of insertion.

Moritz[4] describes cases of twin-sisters aged twenty years with papillomata of the larynx. One began to suffer from hoarseness when two years old. He advances the theory that papilloma of the larynx may be caused by inhalation of amniotic fluid mixed with gonorrhœal discharges during birth.

Lavraud, of Lille,[5] attaches great importance, in the etiology of laryngeal papillomata, to repeated irritation and a catarrhal process which may in a measure explain their occasional retrogression after tracheotomy, the irritation being thus removed. I cannot but believe that there are determining causes in the production of laryngeal growths in children other than local catarrhal irritation, owing to their great infrequency as compared with the great frequency of catarrhal diseases in children. Papillomata represent by a large majority the type of laryngeal growths as seen in children; other growths are comparatively rare, while malignant growths are, as Sir Morell Mackenzie says, "a pathological curiosity." Of the more recent reported cases of other types, L. Grunola, of Munich,[1] mentions a case of mixed tumors in a child three years old. He removed a fibroma from the base of the epiglottis after tracheotomy and subsequently a cauliflower papilloma by laryngo-fissure. At a later date there was a recurrence of the papilloma from the base of the epiglottis, which was removed by the snare.

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[1] Revista de las Ciencias Médicas, Habana, September 5, 1889.

[2] Journal of Laryngology, London, July, 1891.

[3] Annual of the Universal Medical Sciences, 1892.

[4] British Medical Journal, November 26, 1892.

[5] Revue de Laryngologie, d'Otologie, et de Rhinologie, Paris, August 1, 1893.

G. Zuffinger[2] describes a peculiar bilobed morbid growth, keratosis, in a young girl sixteen years old, arising from the anterior commissure and involving the inferior surface of the vocal cords. The growth was covered with small bristle-like appendages.

A fatal case of *abscess* is reported by Jacobs[3] occurring in a child seven years old. There was embarrassment in expiration; nothing was shown by the laryngoscope, and a diagnosis of subglottic tumor was made; a mass of pus followed a tracheotomy, and the child died of pneumonia.

A case of scleroma of the left vocal cord is reported by Zuffinger[4] in a girl of ten years.

Eugène Révelliard[5] reports an instance of polyp of the trachea, following tracheotomy for croup, in a child of two and a half years. It was removed with cure after a second tracheotomy. This was undoubtedly due to irritation of the tracheal canula.

The symptoms subjectively considered are those common to any progressive involvement of the larynx leading up to a mechanical obstruction. There are no subjective symptoms absolutely characteristic of laryngeal growths in children, and the best that can be done is but to conjecture until their true objective character is revealed by the finger or mirror, or the coughing up of fragments, as in some cases of papilloma.

The prognosis as to their fatality is not necessarily bad, if tracheotomy can be performed early enough, in case of serious stenosis; and speaking of papillomata, the prognosis as to their recurrence must always be guarded, as under any form of treatment there is a marked tendency to recurrence, often necessitating frequent operations, which, though resulting finally in the cure of the growth, may injure the phonatory function of the larynx.

The choice of treatment in papillomata of the larynx in children always presents points for mature deliberation. The two main questions are whether to adopt the endo- or extra-laryngeal methods, and which variety, if either. In their choice much will depend on individual and accumulated experience, as it is adapted to the case in hand. There must be taken into consideration the age of the patient, the amenability to manipulation, and the extent and seat of the growths. It is always a temptation to try the endo-laryngeal operation, which, if it gives promise of success, is to be preferred to the extra-laryngeal methods; but there are unquestionably instances where, from the character of the case in all its phases, the endo-laryngeal methods should not be employed. No absolute line can be drawn. The methods of endo-laryngeal procedure are mainly by forceps, either after the Mackenzie type or the tube forceps, the latter, from my own experience, being in the main preferable; the snare, either cold wire or galvano-cautery; direct application of caustics; application of astringents, including alcohol, which of late has been used with doubtful success; and, finally, the pressure effect of intubation. The points favoring the endo-laryngeal methods are: a child of sufficient age and tractability and where an accurate command of the larynx is possible, a rather isolated and limited area of growth, the more pedunculated type, the ease of access, and where after the removal the galvano-cautery may be applied with equal precision, for I consider that in all attempts at removal the base of operation should always be cauterized. Hemorrhage, though not necessarily excessive, may interfere with a proper continuance of the operation, and great care must always be taken lest adjacent tissue be injured. The galvano-cautery snare, though unquestionably efficient when it can be used, demands an extreme nicety of application which is rather difficult in children. I do not consider the primary application of caustics practicable except in a most restricted type of growth; it is too slow a process. In using the galvano-cautery, especially on the lateral aspect of the larynx, the cautery point should be guarded to prevent cauterizing the opposite side. It is unnecessary to say that in operating by the endo-laryngeal methods without general anæsthesia cocaine should be used, remembering, however, that children are very susceptible to its influence; hence a small quantity of the weakest possible solution should be employed. It is a good procedure to previously cocainize the pharyngeal cavity with a very weak solution in order to facilitate the use of the mirror, and then to isolate a small quantity of a stronger solution—from two per cent. to four per cent.—in the larynx by means of a syringe, a swab, or a form of spray that will enter the superior laryngeal cavity. There have been a number of reported instances of the treatment of laryngeal papilloma by intubation with the idea of producing absorption of the growths by means of the pressure of the tube. While unquestionably this method will be of service in certain cases, its success will depend on the favorable location of the growths, so that pressure can be intelligently exerted, remembering that from its narrowness of calibre the subcordal space is best adapted to this type of pressure, while the supracordal space is much larger and yielding, and will necessitate an enlargement of the tube corresponding to this region in order to properly exert the required pressure.

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[1] Munich medicinische Wochenschrift, No. 41.

[2] Wiener klinische Wochenschrift, No. 47, 1891.

[3] Paris Médical, March 16, 1891.

[4] Journal of Laryngology, London, July, 1891.

[5] Revue Médicale de la Suisse romande, Geneva, March 20, 1891.

Fenestrated tubes have been constructed by Lichtwitz,[1] the opening coming opposite the growth, so that it will project into the tube and can be removed by forceps through the tube. Dr. O'Dwyer, of New York,[1] has constructed a fenestrated tube especially adapted for subglottic growths in children. Dr. O'Dwyer[2] has also devised cylindrical tubes for foreign bodies in the larynx or trachea. The tubes are much shorter than the regular ones and much larger in calibre and circumference, hence should not be left *in situ* too long on account of possible ulceration by pressure of the tube.

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[1] Journal of Laryngology, London, February, 1893.

Bonam,[3] of Brest, reports a case of a child fifteen months old in which a fragment of nut-shell was lodged in the larynx or trachea, and the patient was cured by expelling it into the intubation tube.

Baldwin,[4] of Columbus, Ohio, reports a cure of papillomata by intubation in a child of eight years. The growth was large. Cases are also reported by A. Rosenberg[5] (two cases) and Thrasher,[6] of Cincinnati.

It has occurred to me that intubation could be subsequently used to exert absorbing pressure on the remaining parts of growths left by partial removal by endo-laryngeal methods.

When, from the youthfulness of the patient, inability of control, and large or diffuse extent of the growths, extra-laryngeal methods are undertaken, which methods consist of thyrotomy with a thorough removal of the growths and subsequent cauterization and tracheotomy either as palliative or with the idea of recession of the growths, as undoubtedly happens in a certain number of cases, and of which a number of instances have been reported, I think that if there is a tendency to recurrence after other operations, tracheotomy with the above idea in view is certainly worthy of recommendation. The propriety of recommending it as a first operation is questionable, as we do not know how long we would have to submit the child to the inconvenience of wearing the canula before the growth would atrophy.

J. A. White[7] reports a case of tracheotomy becoming necessary in a boy of five years after a quick and luxuriant recurrence following a number of operations. Three months after tracheotomy the growths had almost disappeared, and in three years entirely so.

Garel, of Lyons, France,[8] reports a case of spontaneous recession of a papilloma in a girl of four years, following tracheotomy.

F. Massei,[9] though admitting the tendency to recurrence, speaks also of the tendency to disappearance after tracheotomy. For this reason he deprecates thyrotomy and laryngectomy, and considers operations through the mouth and tracheotomy entirely sufficient.

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[1] Sajous's Annual, 1894.

[2] Annual of the Universal Medical Sciences, 1891.

[3] Revue mensuelle des Maladies de l'Enfance, Paris, July, 1895.

[4] Medical Record, New York, March 8, 1895.

[5] Deutsche med. Wochenschrift, Leipzig, August 31, 1893.

[6] Journal of the American Medical Association, August 22, 1892.

[7] Ibid., October 22, 1892.

[8] La Semaine Médicale, May 6, 1891.

[9] Annales des Maladies de l'Oreille, du Larynx, du Nez, et du Pharynx, Paris, December, 1893.

F. Semeleder[1] reports a papilloma the size of a pea at the anterior commissure in a child of eight years. Laryngo-fissure was performed. Recurrence took place, and the child died during a paroxysm of suffocation in pulling out the canula.

E. Fletcher Ingalls, of Chicago,[2] reports a case of multiple papilloma in a boy of three years. Endo-laryngeal operations were performed; recurrences took place, and death occurred from bronchitis in ninety-six hours.

Borneman, of Berlin,[3] reports four cases of multiple papilloma in children with results:

(1) Laryngo-fissure; voice remained impaired.

(2) Tracheotomy and laryngo-fissure; sharp spoon and curette; recurrence and death by pneumonia.

(3) Laryngo-fissure; recurrence.

(4) Endo-laryngeal methods with forceps; successful.

R. Kohler[4] reports a recurrence with subsequent death from diphtheria in a child fourteen months old with multiple papilloma, after superior tracheotomy and laryngo-fissure.

A successful case of removal of multiple papilloma with stenosis, without precautionary tracheotomy, in a boy of ten years, is reported by De la Sota.[5]

Clinton Wagner, of New York,[6] reports a case of thyrotomy in a child of eighteen months with laryngeal growth.

F. T. Paul and Middlemass Hunt,[7] of Liverpool, report a case of **spindle-celled sarcoma** of the larynx in a boy of twelve years. The tumor was very large, filling the entire larynx. It was removed by endo-laryngeal methods and thyrotomy. The main seat of the growth—viz., the anterior part of the right vocal cord—was thoroughly scraped, and the child made an excellent recovery. Paul lays great stress on "the utility of thorough removal of these growths and a differential recognition of their histological character, previous clinical experience having shown that sarcoma in the larynx is generally mild, thus presenting a modified character of malignancy when compared with sarcoma of other parts."

The view above quoted, though possibly not universally maintained, should have its influence against laryngectomy for sarcoma until a thorough removal by other means has been employed.

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[1] Gaceta Médica de México, January 15, 1891.

[2] Journal of the American Medical Association, February 7, 1891.

[3] Deutsche med. Wochenschrift, Leipzig, April 9, 1891.

[4] Ibid., June 11, 1891.

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[6] New York Medical Record, November 7, 1891.

[7] Revue Médico-Pharmaceutique, Constantinople, January, 1894.

PSEUDO-MEMBRANOUS LARYNGITIS.

By DAVID BOVAIRD, M.D.

The bacteriological studies of recent years have led to an entirely new classification of this subject. On the basis of the bacteriological findings we speak of,—

1. Pseudo-membranous laryngitis due to the presence and growth of the specific germ of diphtheria, the Klebs-Loeffler bacillus. This constitutes true diphtheria of the larynx. Owing to the almost universal distribution of staphylococci and streptococci, a greater or less number of these bacteria are regularly found accompanying the diphtheria bacillus in cultures from cases of true diphtheria. As a rule, the staphylo- and streptococci are present only in small numbers, and seem to play no active part in the inflammatory process. In certain cases, however, they equal or exceed in number the diphtheria bacilli, and their growth and development seem to constitute an essential feature of the pathological process. These cases are sometimes spoken of as mixed infections. At the present time they are considered in all their relations with the cases of true diphtheria; but it seems probable that in the near future it will be found of advantage to consider them entirely by themselves.

Of two hundred and eighty-six cases of primary pseudo-membranous laryngitis examined by the New York Board of Health, eighty per cent. showed the presence of the diphtheria bacillus, while in only fourteen per cent. was it clearly demonstrated to be absent. Further experience has fully confirmed these results, so that in the absence of positive evidence to the contrary all cases of primary pseudo-membranous laryngitis should be looked upon and treated as true diphtheria.

2. Pseudo-membranous laryngitis due to the presence and growth of bacteria other than the diphtheria bacillus, for the most part the streptococcus pyogenes, sometimes the staphylococcus, sometimes both. This constitutes pseudo-diphtheria of the larynx. The pseudo-membranous laryngitis which occurs as a complication of measles, scarlet fever, and other infectious diseases is usually pseudo-diphtheria; it may, however, be true diphtheria.

Clinically both true diphtheria and pseudo-diphtheria of the larynx are characterized by the development of pseudo-membrane within the larynx and the symptoms of severe laryngitis. They cannot be differentiated from each other by the character of the membrane, by the degree of swelling of the cervical lymph nodes, or by any other clinical feature. Nothing but a culture from the affected throat will tell us which bacteria are the exciting cause of the pathological process.

In making cultures from laryngeal cases it is rarely possible to reach the larynx itself with the swab, and practically it is found that the bacteria which are exciting the inflammatory process are present also in the pharynx and will regularly be obtained in cultures made from that part. While in the first days of the disease, owing to accidental causes, cultures from the throat may either result negatively or show only strepto- or staphylo-cocci, a second or third attempt will demonstrate the presence of diphtheria bacilli.

The pseudo-membranous laryngitis which develops secondarily to a pseudo-membranous inflammation of the tonsils, pharynx, or nasal fossæ is simply an extension of the primary process, and it may, therefore, be justly assumed that the bacteria found in the initial focus are the cause of the laryngitis. They may be either the diphtheria bacilli or the pyogenic cocci.

The teachings here outlined constitute the reply of scientific investigation to the long-debated question as to the relation of croup and diphtheria. The great majority of the cases of primary croup are diphtheritic; a small part may be due to the action of other bacteria. Practically there is no croupous inflammation of the larynx in which bacteria are not concerned. The so-called traumatic croup resulting from burns or scalds is found to be regularly accompanied by the growth of strepto- and staphylococci.

Pathology.—The pathology of croupous or pseudo-membranous laryngitis does not differ in its fundamental features from that of croupous inflammation occurring elsewhere. For the details on this subject the article on Diphtheria may be consulted. The peculiar features of pseudo-membranous laryngitis are fully set forth in vol. ii. of this Cyclopædia.

Symptomatology.—The symptoms of pseudo-membranous laryngitis have been so fully discussed in the original article that only a brief summary need be here given. In the early stages we find a croupy cough, hoarseness, a temperature of from 101° to 102° F., with a corresponding elevation of pulse and respiration and a moderate prostration. So long as the process is limited to the larynx the constitutional symptoms are not prominent. When the inflammatory process extends to the adjacent parts, either upward or downward, the temperature rises, the pulse and respiration are increased accordingly, and the prostration becomes more marked. In the purely laryngeal process the local symptoms constitute the important feature of the case. As the swelling and formation of pseudo-membrane increase, the hoarseness deepens into aphonia, inspiration, then expiration, become stridulous, and the respiration becomes more rapid and labored. At any point the inflammatory process may cease to increase or may be checked by treatment, and improvement begins. But if it continues to advance, sooner or later, to the obstruction of the larynx due to the swelling and pseudo-membrane there is added the element of spasm of the glottis, which introduces the phenomena of rapid strangulation so graphically described in the original article. Progressive, unremitting stenosis of the larynx is the characteristic feature of pseudo-membranous laryngitis, but some remission of the symptoms may occur.

Stenosis may develop suddenly, even in mild cases or late in the course of the disease.

In its severest form pseudo-membranous laryngitis is fatal within forty-eight hours. Most of the cases run their course within one week, but the disease may be protracted for several weeks by complications. Of these pneumonia is by all means most frequent, and is usually the immediate cause of death.

Diagnosis.—The great majority of cases of primary croup are diphtheritic. Only as a complication of the acute infectious diseases is the affection generally pseudo-diphtheria. The distinction can be made positive only by means of cultures from the affected throat. But in many cases of pseudo-membranous laryngitis, in the early stages the pseudo-membrane is confined to the larynx, and is therefore not seen on inspection. Unfortunately, the employment of the laryngoscope is not practicable at the age at which croup is most common, from the second to the fifth year. Failure to see the pseudo-membrane does not exclude pseudo-membranous laryngitis. In many cases the exudate is so slight that even after death it appears only as a thin film upon the interior of the larynx.

We must therefore be prepared to distinguish pseudo-membranous laryngitis from the simple catarrhal or spasmodic affection. The latter affection is characterized by a higher temperature, with less prostration, than a pseudo-membranous laryngitis, the marked remission of all symptoms during the day, with the return of cough and dyspnœa at night.

But here again the wisest course would be to employ cultures from the throat as a safeguard in diagnosis. The necessity of employing cultures as a means of diagnosis in all cases of acute inflammations affecting the larynx must be insisted upon. It is impossible to determine the bacteriology of a pseudo-membrane by looking at it. Furthermore, pseudo-membrane may be present in the larynx and not be visible on inspection; and, finally, experience has fully demonstrated that some cases of simple catarrhal inflammation are due to the action of diphtheria bacilli and should be treated as true diphtheria.

Prognosis.—Pseudo-membranous laryngitis, whether it be true diphtheria or pseudo-diphtheria, is always a serious and often a fatal affection. Even the mild cases must be viewed with concern. They may at any time develop dangerous stenosis. The constitutional depression is severer, and consequently the danger to life is greater, in true diphtheria. While, as a rule, the same is true of the local symptoms, we have often seen pseudo-diphtheria of the larynx, complicating measles or scarlet fever, present a clinical course as rapid and fatal as that of true diphtheria.

Treatment.—The advances in the treatment of pseudo-membranous laryngitis include as chief factors the use of antitoxin and calomel fumigations.

Antitoxin.—The discussion of antitoxin as a remedy in diphtheria belongs to the description of that disease. We shall here speak only of its application to the treatment of the laryngeal process. Being derived wholly from the action of the diphtheria bacillus, it is at once evident that it can be expected to act curatively only in the cases of true diphtheria of the larynx. However, since the great majority of the cases of primary pseudo-membranous laryngitis are true diphtheria, and the value of injection in such cases has been clearly shown, while the employment of the remedy is practically devoid of danger, it should be made a rule to administer antitoxin in every case of primary pseudo-membranous laryngitis the moment the diagnosis is made. Formerly we waited for the results of cultures from the throat to be made known: now there should be no delay. Life may be saved by promptness. No harm is done if the case proves to be pseudo-diphtheria. Owing to the gravity of the disease, the maximum dose is employed under these circumstances. After consideration of the report of its special committee on antitoxin, the American Pediatric Society at its last annual meeting recommended the use of from fifteen hundred to two thousand units of antitoxin in every case of laryngeal diphtheria. At the Willard Parker Hospital, under control of the New York Board of Health, never less than two thousand units are now employed. In cases of severe stenosis, Monti recommends from two thousand to three thousand units, while in the gravest cases Janowski[1] advocates even from four thousand to five thousand units. Whatever quantity is employed may be repeated after twelve or twenty-four hours, according to the indication.

The evidence presented in the report of the American Pediatric Society is overwhelmingly in favor of the use of antitoxin in these laryngeal cases. Of 1256 cases of laryngeal diphtheria, 691, or more than one-half, recovered without operation (O'Dwyer). In many of the cases reported to the society symptoms of severe stenosis disappeared completely after the injection. Many physicians considered its action in this respect almost marvellous. The final results of the treatment of these laryngeal cases were far beyond anything previously attained. Of 691 non-operative cases, 128 died; mortality, 18.5 per cent. Of 533 cases intubated, 138 died; mortality, 25.9 per cent. Of 32 tracheotomized, 12 died; mortality, 37.5 per cent.

There is no need of deducting moribund cases to make these statistics most favorable to the new remedy. They cannot be approached by any former method of treatment. Antitoxin shortens the course of the disease, limits the extension of membrane, relieves the stenosis, combats the toxæmia,—in short, shows itself a specific curative agent.

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[1] Archiv für Kinderheilkunde.

It has been rightly urged that the use of antitoxin in no way interferes with the employment of any other rational method of treatment. Calomel fumigations, steam inhalations, or the bichloride treatment described in the previous article may be employed.

So far as the laryngeal process is concerned, in children local applications cannot be employed.

In combating the stenosis of pseudo-membranous laryngitis emphasis should be laid upon the fact that the obstruction is not wholly due to the amount of pseudo-membrane. At autopsy one is often greatly surprised at the small amount of membrane found in cases presenting the severest degree of stenosis. Spasm, congestion, and œdema have much to do with limiting the calibre of the glottis; it is not simply blocked by membrane. All these factors are aggravated by excitement or unrest. Perfect quiet is therefore essential, and many a threatening attack of stenosis can be relieved by the use of opium in some form. In the experience of the writer and his associates in the Foundling Hospital, Dover's powder in solution is the best preparation for such cases. While having a sedative effect, it does not dry the throat by checking secretion. To a child of two years two grains may be given; from the third to the fifth year, three grains. The dose should be repeated whenever necessary. There is no danger of respiratory depression from such doses.

In all cases great care should be taken to give abundant fluid nourishment, and heart stimulants, especially alcohol and strychnine, should be freely used.

Calomel Fumigations.—Just previous to the introduction of antitoxin, calomel fumigations were a favorite method of treatment. A tent or canopy was rigged over the crib in any convenient way. Within this from fifteen to sixty grains of calomel were volatilized over an alcohol lamp at intervals of from one-half to two hours. After the volatilization the tent was kept closed for fifteen minutes. The room was then thoroughly aired. For the volatilization special apparatus can be had, but is not required. This method of treatment undoubtedly produced anæmia in the patients, and frequently salivated attendants, but seemed to be productive of good results. Since the use of antitoxin has become general, it has been practically abandoned.

For the methods of giving steam inhalations the article in vol. ii. may be referred to.

Whenever in any case the advancing stenosis cannot be relieved by palliative measures, and the child begins to struggle for air, operative relief should be given. The long contest between tracheotomy and intubation has been decided, on the basis of practical experience, in favor of the latter.

When employed together with antitoxin an impartial observer speaks of it as the "ideal operation for the relief of the great majority of the cases of croup requiring operative interference."[1]

For the details of the operation and after-treatment the article on Intubation may be consulted.

Finally, we may summarize the treatment of pseudo-membranous laryngitis thus:

Antitoxin for all cases that may be true diphtheria.

Calomel fumigations, steam inhalations, sedatives, heart tonics and stimulants, and careful feeding as adjuvants.

Finally, when the indications arise, intubation.

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[1] Welsh, Transactions of the Association of American Physicians, 1895.

INTUBATION.

BY DAVID BOVAIRD, M.D.

INTUBATION has not only maintained the ground occupied at the time of the publication of the first volumes of the Cyclopædia, but has advanced to the occupation of a new field. It is now necessary to speak of,—

1. Intubation for acute stenosis of the larynx.
2. Intubation for chronic stenosis of the larynx.

INTUBATION FOR ACUTE STENOSIS.

Indications for the Operation.—Practically the acute stenosis requiring intubation is that excited by a pseudo-membranous laryngitis, either diphtheritic or pseudo-diphtheritic. The operation has been required in some cases of acute catarrhal or spasmodic laryngitis, but such cases are rare. Whenever in the course of a laryngitis there develops an increasing stenosis which, unrelieved by palliative treatment, reaches a point where insufficient air is admitted through the narrowed larynx, intubation should be done promptly. The signs of such obstruction are rapid, labored, and stridulous respiration, recession of the supra-clavicular and supra-sternal tissues and the epigastrium during inspiration, diminished respiratory murmur over the lower lobes behind, and the rational consequence of insufficient aeration of the blood, cyanosis. In rare cases the stenosis may be entirely due to swelling and infiltration of the tissues of the larynx below the vocal cords, and respiration will be almost noiseless; but, as a rule, stridulous dyspnœa is characteristic of laryngeal stenosis. Increasing restlessness goes hand in hand with advancing stenosis, and in severe cases the patient will present all the frightful phenomena of rapid strangulation. To wait for such symptoms before operating is only to invite a fatal issue. Whenever it becomes evident that the stenosis is advancing, despite palliative treatment, and the struggle for air becomes a tax upon the vitality of the patient, intubation is indicated. Delay is dangerous and may be fatal.

The Instruments.—No better evidence of the thoroughness with which the problems involved in intubation had been studied and the indications fulfilled could be asked than the fact that the instruments employed stand to-day almost exactly as they came from the inventor's hands. Not that [illegible] proposed; in the early years of trial they were [illegible] the operators. The tubes have been the special ob-[illegible]. Longer tubes, shorter tubes, tubes with artificial epiglottis [illegible] of larger calibre,—all manner of modifications,—have [illegible] as O'Dwyer says of them, they have only added dangers to [illegible]. No one of these modifications has stood the test of [illegible]. No new principle has been added to those recognized by the [illegible] in the construction of the tubes first given to the profession. The success of the operation has been endangered not only by unwise [illegible] originality, but also by the difficulties in the way of those who would gladly have copied the instruments as O'Dwyer fashioned them. On this point the inventor says, "I have never seen a tube made outside of New York which was not imperfect in one or more points; and even in New York there are but two instrument makers that have ever made tubes that I would recommend, or from which I would expect to get the best results. Even these two makers require constant watching, otherwise they grow careless and make tubes that ought never to be inserted into the human larynx. I have never found any workman, however intelligent and skilful, who could make an exact copy of one of these little tubes without having previously had special instruction." Most of the tubes sold as O'Dwyer's are, therefore, only caricatures of the inventor's work. It is right to emphasize the proper construction of the tubes, for no operator, however skilled, can attain good results without attention to this point.

The changes made in the instruments in the last eight years have been few. The mouth-gag and introducer remain the same. The heads of the two smallest tubes have been hollowed out to facilitate extraction. The extractor has been made heavier, and the duck-bill points broader, to give it a firmer grasp, and also to lessen the chances of lacerating the tissues.

For use in the cases where there is much loose membrane in the trachea or bronchi, O'Dwyer has contrived a set of tubes which are simply short, hollow cylinders of large calibre. They are designed to extend beyond the cricoid and leave as large a lumen as possible for the escape of the membrane. In each set there are seven tubes, graduated in size in a manner similar to the regular set. They are introduced by a special instrument with a longer curve at the neck to compensate for the shorter length of the tubes. Having no retaining swell, these tubes are simply wedged into the larynx, and from the resulting pressure can be retained only for a short time. As soon as the loose membrane has been expelled they are removed, and, if necessary, are then replaced by the regulation tube. Some operators say that with antitoxin these special tubes are less frequently required; but, on the other hand, they have been employed more often of late in the Willard Parker Hospital because of the rapid exfoliation of the false membrane attributed to the use of the specific remedy.

Technique.—The technique remains as described in vol. ii., but the experience of ten years has abundantly proved that intubation is a safe operation only in the hands of those who have been trained by practice upon the cadaver. Without such practice no man can intubate without danger of serious injury to the larynx.

Even long practice upon the cadaver will not make safe operators of many men. Since respiration is practically suspended from the moment the finger touches the larynx, the operation to be successful must be done very quickly. With the mouth-gag in place, not more than ten seconds should be required to insert the tube into the larynx. Such quickness demands not only practice, but inherent skill. For those who have had no practice in intubation, tracheotomy remains the safer operation.

Time for Removal.—Often the child settles this question by coughing out the tube and not requiring its reintroduction. Up to the present antitoxin era the average time for the retention of the tube has been five days. Some operators have claimed good results from the daily withdrawal of the tube, either reinserting it immediately after cleaning it, or waiting till the return of stenosis required it. Conservative judgment cannot commend this procedure. Unless there is some direct indication for removal, such as blocking of the tube by membrane or secretions, the tube should be allowed to remain until the remission of the symptoms of the disease or the lapse of time renders it probable that it can be dispensed with. The other method only exposes the patient to exhaustion and the repetition of the dangers inherent in the operation. Just in proportion as it shortens the course of diphtheria itself, antitoxin seems to shorten the time for the use of the tube. The evidence upon this point is not yet conclusive, but in a recent article[1] the reports of a number of observers show a reduction of from eighteen to seventy-one hours. In many cases the tubes can be removed within three days (O'Dwyer).

Retained Tubes.—Occasionally it happens that after all other symptoms of the laryngeal disease have disappeared, the removal of the tube is followed by immediate return of the stenosis, and reintubation is necessary. One case in the New York Foundling Hospital was intubated forty times before the tube was finally dispensed with. These cases are spoken of as "retained tubes." The cause of such retention is ulceration of the larynx, due to the use of too large or improperly made tubes. In some cases the lesion is accompanied by the growth of granulation-tissue about the upper margin of the larynx. For such cases tubes with a prolonged or built-up head are required. Again, the difficulty may be a paralysis of the vocal cords due to pressure. In such cases the tubes are repeatedly expelled by coughing. They call for tubes with a narrow neck to lessen pressure upon the cords, and a larger retaining swell to keep them in place. But most commonly the ulceration is below the level of the vocal cords, and the only indication is for the use of smaller-sized tubes. Tracheotomy is the worst possible treatment for these cases of retained tubes, for in the end intubation will probably be required to get rid of the tracheal canula (O'Dwyer).

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[1] Rosenthal, Medical and Surgical Reporter, vol. lxxiv. p. 22.

In certain parts of the Continent it is the established practice to perform a secondary tracheotomy upon all cases of intubation in which the tube cannot be dispensed with at the end of some arbitrary period of time, usually four or five days. The only justification for a secondary tracheotomy is the failure of the tube to relieve the stenosis. The employment of tracheotomy without reference to this point has no rational basis, and its only result has been a great increase in mortality.

After-Treatment.—The great problem in this connection has always been to feed the patients. The presence of the tube interferes to some extent with the closure of the larynx and allows food to pass into and irritate parts already sore. Troublesome coughing and choking naturally follow. The problem of avoiding such disturbance can be solved in most cases by adopting the procedure suggested by Casselberry, of Chicago. This consists in feeding the child while lying upon an inclined plane with the head downward. This is best done by placing the child supine across the nurse's lap and allowing the head to fall backward over her thigh. In this position even fluids can be taken without harm. Some children, however, will not feed in this position. In such cases spoon-feeding should be insisted upon, so that after each swallow the child will cough and clear the larynx of anything that may have entered it, or nasal feeding may be employed. In the latter procedure a velvet-eyed silk catheter is passed through the nose into the œsophagus and liquid nourishment poured through it. This method is comparatively easy and very efficient. Feeding is so essential a part of treatment that no pains should be spared to accomplish it.

A second problem in after-treatment is the prevention of pneumonia. The opinion of Roux, Martin, and other French writers that pneumonia is largely due to local hygienic conditions resulting in the infection of hospital wards and sick-rooms with pneumonia-producing bacteria is generally accepted. Perfect cleanliness and thorough ventilation are therefore to be insisted upon. Pneumonia may also in some cases be due to the aspiration of the products of inflammation from the trachea or bronchi into the lungs. To avoid this danger, it has been suggested that the foot of the bed of an intubated patient be kept elevated in order to drain the respiratory passages by gravity.

Dangers of the Operation.—In introducing the tube, any one of several accidents may seriously affect the success of the operation:

1. Prolonged attempts to intubate by an unskilled operator may result in asphyxia.

2. The lower end of the tube may enter one of the ventricles of the larynx, and serious harm be done in attempting to push the tube onward. This danger was early recognized, and to lessen it the lower end of the tube was enlarged and given its present bulbous form. To escape it altogether, the operator need only see that the child is held perpendicularly and keep

the axis of the introducer exactly in the median line. So long as these rules are followed, this accident cannot occur; whenever the introducer deviates even slightly from the median line the danger is imminent.

3. Loose membrane in the trachea may be pushed down before the tube, blocking its lower end and preventing respiration. This pushing down of membrane has been the ready explanation of most cases of serious accident in intubating. O'Dwyer met with it only three times in over two hundred operations, and the careful analysis of the deaths reported from this cause shows very clearly that most of them have been due to the accidents previously mentioned,—namely, too prolonged attempts at intubating or penetration of one of the ventricles,—and not to loose membrane.

If the tube is blocked by membrane it should be immediately withdrawn. The resulting cough may at once clear the trachea and justify the reintroduction of the tube, or the cylindrical tubes previously described may be employed until the membrane has been expelled. The signs by which we may determine the presence of loose membrane in the trachea are (1) a croupy cough with a tube in the larynx, (2) a flapping sound, (3) sudden obstruction to expiration, especially during coughing.

During the wearing of the tube it may become plugged by dried secretions and calcareous deposits. Such obstruction will make itself evident by steadily increasing dyspnœa. The tube should be removed and cleaned. No metal tube should be allowed to remain continuously in the larynx for more than five days.

The ulceration produced by badly fitting tubes has already been spoken of. This can be avoided by attention to the construction of the tubes and by making a rule in any doubtful case to employ a small rather than a large tube. If too small a tube is employed it may slip below the vocal cords, but, unless the disparity is extreme, the head of the tube cannot pass through the cricoid cartilage. It will therefore lodge at that point. There are no rational signs by which this accident can be recognized, and the tube cannot be felt through the larynx or trachea. The only means of demonstrating the presence of a tube in the larynx below the vocal cords is to pass a sound into the larynx and elicit a metallic click from the displaced tube. An effort may be made to remove such a tube in the ordinary way. This failing, tracheotomy is required.

Advantages of Intubation.—The battle of statistics between intubation and tracheotomy has been long and fiercely fought.

Columns of figures might easily be quoted clearly showing the better results from intubation, but the question at issue is not to be answered by comparison of tables of operations and deaths. The indications for the operations are the same, but the fact remains that in the great majority of cases parents prefer intubation, and will submit their children to it when tracheotomy will not be entertained. The experience of the city of Brooklyn is in point. With four or five hundred deaths from croup each year, the leading tracheotomist was called upon to operate only sixty-six times in seventeen years. In the same city one intubator in four years operated upon one hundred and forty-two cases. One operator saved twenty-two cases in seventeen years, the other forty-two in four years. In the face of such facts, which could be corroborated by all our large cities, further argument over statistics of operative results is absurd.

INTUBATION FOR CHRONIC STENOSIS.

Within recent years intubation has been employed for every form of chronic stenosis of the larynx. In children the operation has been most often resorted to in order to get rid of retained tracheal canulæ. The principle upon which intubation has been employed in these cases is that of gradual dilatation, a tube which could be easily inserted being first employed, and replaced by larger ones as the stricture relaxes. The instruments and methods are the same as in acute stenosis.

The operation is making steady progress both in this country and abroad. Every year brings new reports of its successes. The results are so satisfactory as to justify progress and insure its continuance. The application of the operation belongs so distinctly to the field of the laryngologist that it does not seem advisable to enter into the details in this place.

BRONCHO-PNEUMONIA.

By F. GORDON MORRILL, M.D.

Synonyms.—Catarrhal or lobular pneumonia, Capillary bronchitis.

Definition.—Inflammation of the terminal bronchi and air-vesicles, which in severe cases may involve all the component elements of the lung. In young children the disease may kill so quickly that a bronchiolitis is the only gross lesion discovered by post-mortem examination. Microscopic investigation has, however, proved that the air-vesicles are always complicated to some extent; and, to avoid confusion, it must be remembered that "capillary bronchitis" (bronchiolitis) has no claim to recognition as a distinct disease, and the term should be used only to define a stage or condition which is invariably present in broncho-pneumonia.

History.—Leger was the first to notice, in 1823, that a form of pulmonary inflammation is frequently observed in children which differs from the ordinary pneumonia of adults, with which it had been previously confounded. A few years later the lobular character of the disease was demonstrated by Leger, De la Berge, and others. In 1838 Rilliet and Barthez established the fact that there are two distinct varieties of pneumonia to which children are liable: one lobar, which they considered essentially primary, and the other lobular, which they regarded as secondary. In this same year the term "broncho-pneumonia" was originated by Seifert.

A few years later Barrier described three varieties of the affection, disseminated, diffused, and pseudo-lobar. Fauvel, Legendre, and Bailly (1840–44) contended strongly that "capillary bronchitis" was the essential element of the disease, and that the lesions involving the air-cells were either mechanical or due to frank pneumonia of a disseminated type. Bartels and Ziemssen (1860–69) regarded every lesion, aside from the bronchiolitis, as purely mechanical. Subsequently, Charcot demonstrated the fact that lesions of the parenchyma were of two kinds, mechanical—including atelectasis and emphysema—and inflammatory; at the same time he showed the marked difference between the latter and the fibrinous exudate of frank pneumonia. It remained only for the bacteriologists to discover the micro-organisms which are to-day regarded as essential factors in the etiology of frank and broncho-pneumonia, diseases which differ as widely as any two affections of the same organ possibly can.

Varieties.—The type which often proves fatal before the inflammation has obviously affected the air-vesicles was formerly known as "suffocative catarrh," or capillary bronchitis.

That in which the aggregation of affected lobules may render a large portion of one or more lobes solid is called "pseudo-lobar" or "mixed" broncho-pneumonia.

In any exhausting illness the patient's sensibility may become so blunted as to permit the passage of particles of nourishment through the larynx, and this may give rise to the "deglutition" or "inspiration" form of the disease.

The frequent association of tubercle with broncho-pneumonia justifies the use of the term "tubercular" in connection with a class of cases which are quite common.

Prevalence and Mortality.—See vol. ii. p. 617.

Etiology.—Youth, climate, season, exposure, filth, poverty, overcrowding, hospitalism, and the presence of measles, diphtheria, tubercle, and whooping-cough are all important etiological factors, inasmuch as they prepare a favorable soil for the infection. Filth and overcrowding are perhaps the most potent underlying causes of the disease in its most fatal form. Death from broncho-pneumonia among well-to-do people in private houses is extremely rare.[1] The causative influence of measles is very marked, irrespective of age, while that of whooping-cough is confined, as a rule, to very young children. The prevalence of the disease as a complication of grippe varies widely in different epidemics.

Bacteriology.—Much attention has been paid to the bacteriology of the disease, particularly in Paris during the past five years; but, in spite of a great deal of careful work, no one micro-organism has yet been definitely connected with any form of broncho-pneumonia. The fact that any or all of the bacteria usually found in the lungs (Talamon-Fraenkel diplococcus, streptococcus pyogenes, staphylococcus aureus et albus, Friedländer's bacillus pneumoniæ, and the Klebs-Loeffler bacillus) may be discovered in normal mouths, noses, and throats justifies us in terming the disease a *non-specific* and (usually) *auto-infectious* one. Its frequent association with other affections (notably whooping-cough and measles) is accounted for by the increased virulence of the micro-organisms in the presence of another infection which is sufficiently potent to overcome the natural mechanical and physiological powers of resistance. I refer to expectoration, the expulsive action of ciliated epithelium, and phagocytosis. Broncho-pneumonia may also be hetero-infectious (contagious), and can be contracted by inhaling the micro-organisms contained in the dried nasal and pulmonary secretions of those already having the disease. Sevestre (of Paris) has reported cases in which the bacterium coli commune was the infecting germ. In cases which are secondary to diphtheria the Klebs-Loeffler, alone or together with other micro-organisms, may be discovered. In a case which died under my care at the Boston Children's Hospital and had contracted diphtheria just as a sudden fall of temperature announced convalescence from frank (croupous) pneumonia, the autopsy showed the presence of broncho-pneumonia in the opposite lung. In this instance pneumococci were found in the lung originally affected, and streptococci and Klebs-Loeffler bacilli in the lesions of the secondary infection.

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[1] See vol. ii. p. 621.

Morbid Anatomy.—The lesions discovered by post-mortem examination vary in accordance with the type and severity of the disease. In the extremely acute and fatal form which masquerades in the guise of a "capillary bronchitis" the gross lesions may be those of a bronchiolitis only. In the usual form[1] one observes, scattered throughout the posterior and lower portions of the lungs, areas of collapsed and inflamed vesicles, some of which latter may contain a little fibrin surrounding the epithelial cells and leucocytes which constitute the usual inflammatory products of the disease. In pseudo-lobar or "mixed" cases the extent of lung involved and abundance of fibrin may render the macroscopic aspect so like that of frank pneumonia as to deceive the ablest pathologist. As a rule, however, such a large consolidation is accompanied by one or more small areas of inflammation in the opposite lung,—a condition far more common in the disease under consideration than in the other. Occasionally the true nature of a case can be decided only by microscopic examination.

In tubercular cases the pathological conditions may be classed under four principal types:

1. In "quick" or "galloping" consumption cheesy deposits, scattered or discrete, are found in the small bronchi and air-vesicles, in addition to the usual lesions of broncho-pneumonia. There is also marked enlargement of the bronchial glands, the contents of which are apt to be of the color and consistency of cream.

2. In "bronchial phthisis" it is the glands which are chiefly involved, so far as tubercle is concerned, and they are the starting-points of the acute inflammatory lesions of which the child dies.

3. When tubercle is deposited in a lower lobe, the accompanying consolidation (which may be quite extensive) is that of broncho-pneumonia of rather a low grade of inflammation, and corresponding pathological changes are found.

4. The lesions present may not differ essentially from those of the usual disseminated form, and recourse must be had to the microscope to determine that the case is one of acute pulmonary phthisis masked by a broncho-pneumonia. Or miliary tubercle is found scattered throughout the lung and on the pleuræ. In a vast majority of instances the tubercular infection precedes the broncho-pneumonia; but that the reverse occasionally happens when the course of the disease is (as at times it may be) indefinitely pro-

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[1] See vol. ii. pp. 622 *et seq.*

longed is undoubtedly true, as tubercle is prone to fasten upon those whose powers of resistance are weakened from any cause.

Symptoms.—In babies, and often in older children, intense dyspnœa and a sudden rise of temperature (104° to 105° F.) may mark the beginning of the most acute and infectious type, outbreaks of which are liable to occur in hospital wards, among children ill with measles, diphtheria, or whooping-cough. If the patient has pertussis, the paroxysms may be greatly modified, or even cease entirely. In any event, the rational signs of a severe pulmonary complication are quickly observed: fever, rapid breathing, dyspnœa, retraction of the intercostal muscles and epigastrium, together with a painful, hacking cough. The physical signs are those of an acute diffused bronchitis of the posterior surfaces of both lungs. Death frequently occurs before any evidence of consolidation is obtained, and is preceded by a diminution of the distress, cyanosis, and, quite frequently, a marked fall of temperature. Recovery is accompanied by slower respiration, and the child's attention can be diverted from its struggle to obtain oxygen enough to sustain life, on which all its powers have been concentrated. Like symptoms, but of slower development, are observed in the ordinary acute form of the disease. Death or recovery occurs at a later stage, while physical signs of scattered areas of consolidation are often present. In the pseudo-lobar form the onset is usually sudden, and the symptoms, both rational and physical, may closely resemble those of an acute frank pneumonia,—the same quick accession of fever, cough, and dyspnœa, together with evidence of a large, one-sided consolidation. I doubt if recovery ever follows under the above circumstances. The tendency is to marked cyanosis, continued high temperature, and death, the latter at an early stage in babies or very young children.

A more protracted form of broncho-pneumonia may occur in the midst of an attack of what appears to be an ordinary bronchitis. It attacks, as a rule, children who are at least four years of age, and who are often able to expectorate the thick, tenacious mucus which accumulates in the bronchial tubes. It frequently resolves itself into a one-sided affection of somewhat gradual development. Here the struggle is prolonged, and the child may remain ill for weeks or months before death (from tubercular phthisis) or recovery (complete or with fibroid induration, bronchiectasis, etc.) ensues.

In cases of bronchial phthisis a slight œdema of the face, together with a spasmodic cough, may give rise to a suspicion, in the absence of a definite history, that the broncho-pneumonia which succeeds the primary affection is a sequel to whooping-cough. In very young children, tubercle, if present, is usually disseminated, and the secondary acute inflammation is rapidly fatal. In such cases an extremely irregular temperature and the presence of enlarged glands, together with the child's miserable aspect, may enable one to anticipate the abundant evidence of tubercular infection presented at the autopsy. In children, pulmonary phthisis occasionally (although much less frequently than in adults) begins with signs of extensive consolidation of a lower lobe. This fact has long been recognized by French observers, and is called phthisis *à forme de pneumonie.* Here the consolidation is that of broncho-pneumonia, but the temperature is not so high as in the more usual pseudo-lobar form, and the physical signs (dulness on percussion, crepitant râles, and a soft, blowing respiration) are less marked. If, under these circumstances, the lung should clear up quickly, the obvious inference is that the case has been one of abortive frank pneumonia (oftener observed at the apex), which may cause similar physical signs.

Diagnosis.—The few (published) results of examination of the blood in broncho-pneumonia show that a leucocytosis is present, but nothing sufficiently definite to aid in the diagnosis has yet been ascertained.

The acute and extremely fatal type, so-called "capillary bronchitis," is easily distinguished from bronchitis by the sudden access of grave symptoms. From frank (croupous) pneumonia it differs in being a double-sided affection and in the fact that it is apt to be secondary. In frank pneumonia the signs of pretty extensive consolidation in one lung are usually present at an early stage; and, while there may be râles in the opposite lung, from a coexisting bronchitis, they are not remarkably plentiful, as a rule, and the quiet type of the dyspnœa is very different from the acute respiratory distress of acute broncho-pneumonia. Bronchial respirations are common and, indeed, usual in croupous pneumonia, while in an ordinary acute broncho-pneumonia only the signs of scattered areas of consolidation are obtained if the child survives the first shock of the disease. The fact that broncho-pneumonia is by far the commoner previous to the third year of life is also of assistance.

To distinguish between the pseudo-lobar form and frank pneumonia is at times very difficult, perhaps impossible. The mode of invasion and the physical signs may be absolutely identical. Perhaps the cyanosis is apt to be greater in the "mixed" form, although it is usually very marked when croupous pneumonia occurs in a young child. Frequently, however, the extension of dulness on percussion into and in front of the axillary space (far more common in pseudo-lobar than croupous pneumonia), or the presence of a solid patch in the opposite lung, may lead to a very strong suspicion of the real nature of a given case.

The more protracted form (which is, perhaps, the one as frequently seen as any outside of hospitals) is apt to follow some ordinary affection of the upper respiratory tract, and is easily distinguished from croupous pneumonia by its more gradual mode of invasion, indefinite duration, and ultimate disposition to become a one-sided affection. This is the classical form of broncho-pneumonia formerly described in text-books, and it is in this type that sudden changes from dulness on percussion to comparative resonance have been so often described and are so seldom heard. That dulness and a deficient respiratory murmur are occasionally produced by defective inflation and partial but temporary collapse in weak children I can readily believe. That subsequent reinflation may cause these signs to disappear is, I think, also true. But I cannot credit the statement that in so complex a condition as that of a broncho-pneumonia, and during the acute stage, phenomena of this kind occur to an extent to be appreciable to the ear. That resonance may suddenly change to dulness goes without saying, but not the reverse. Occasionally a very acute attack will end in a condition of prolonged semi-convalescence with symptoms which resemble those of the later stages of the more ordinary protracted kind. This is more particularly apt to be the case in broncho-pneumonia following pertussis, the emphysematous condition of the lungs causing resolution to be tedious. In either case the question to be determined is whether tubercle is present or not, the diagnosis of chronic subacute broncho-pneumonia being, as a rule, easily made.

It may be said, in a general way, that the physical signs of thoracic diseases in children are less reliable than in adults, while the affections themselves are more complex; and experience teaches that if one's clinical views accord fairly well with the lesions demonstrated in three-quarters of the cases which are autopsied, it is all that can be reasonably expected.

Prognosis.—In babies the disease proves speedily fatal in a vast majority of all cases. Between the second and third years the mortality is probably not far from seventy-five per cent. In older children the outlook is more favorable and two out of three recover. In protracted cases the question resolves itself into recovery or phthisis, tubercular or fibroid. If a positive diagnosis is not made by examination of the sputa or the injection of tuberculin, there is always room for hope so long as the apices are not affected. Cough, emaciation, abundant muco-purulent expectoration, and physical signs of diseased lung may all eventually disappear, and an apparently hopeless condition be changed to that of restored health.

The prognosis is, of course, influenced by the type, as well as the age, of the child; and it may be roughly stated that "suffocative catarrh" (capillary bronchitis) kills a great majority of its victims in two or three days, and that acute pseudo-lobar broncho-pneumonia is almost invariably fatal. For special symptoms of importance in estimating the chances of recovery in a given case, the reader is referred to vol. ii. p. 636.

Prophylaxis.—The nasal and bronchial secretions of children ill with broncho-pneumonia should be carefully looked after, and destroyed by fire or rendered harmless by disinfectants. Cases occurring in wards where measles or whooping-cough is present should be immediately isolated. Good proof of the contagiousness of the disease under the above conditions has been obtained. The opening of special wards for cases of measles at the Enfants Malades in Paris was immediately followed by an increase of twelve per cent. in the mortality, nearly all deaths being due to broncho-pneumonia among the children there herded together. At the Enfants Assistés, Sevestre, by timely isolation and the employment of strict antiseptic measures, reduced the mortality from cases of broncho-pneumonia complicating measles and diphtheria nearly fifty per cent.

While the disease is certainly more contagious in the presence of the two last-named diseases, yet an equally hetero-infectious and perhaps more fatal type occasionally invades wards containing young children ill with whooping-cough;[1] and the isolation of any case whose spasms become suddenly modified, while the temperature ascends, is imperatively called for, because these symptoms can have but one signification. Wards and rooms which have been occupied by cases of broncho-pneumonia coming on in connection with either of the diseases which I have referred to above should be as carefully disinfected as those vacated by scarlet-fever patients. Slow and careful feeding, together with cleanliness and the use of antiseptic mouth-washes, are the means to be employed in guarding against the "deglutition" form of the disease. In cases of diphtheria which have been operated upon, the use of antiseptic gauze over the canula is a safeguard against the entrance of micro-organisms which, if inhaled, may be the means of producing broncho-pneumonia in a child who might escape auto-infection.

Treatment.—Now that the disease has been proved to be due to the presence of micro-organisms planted in a favorable soil, we naturally look for relief in a treatment which shall bring in direct contact with them substances whose germicide properties are established. Experiments in this direction have, however, proved unsuccessful thus far. Creosote, guaiacol, and eucalyptol have all been faithfully tried by inhalation, hypodermic injection, and internal administration, and no good results obtained. As to an antitoxin, the variety of micro-organisms which may cause the disease, together with the uncertainty as to which of them may be present in any particular case, renders relief from this source extremely problematical. There is, however, something to be hoped for in the use of a streptococcus antitoxin, inasmuch as this germ is the predominating one in a majority of cases, and Claisse and Hutinel have reported quite a number of instances in which the symptoms were those of acute septicæmia and the streptococcus the only micro-organism found at the autopsies. The pulmonary lesions were often insignificant in extent, and death evidently resulted from poisoning by the toxins of the streptococci, which, according to Claisse, do not themselves enter the circulation to any extent, as a rule. Here, then, would seem to be an opportunity of testing the value of an antitoxin treatment, and with a chance of favorable results.

Baths have proved of real service, and the indications and rules for their employment are fairly well settled. Tepid baths, which may be repeated three or four times a day, reduce the temperature and relieve dyspnœa and nervous symptoms. The child is slung in a large bath towel, and gently lowered into a bath of a temperature of 95° F., which may be gradually reduced to 85° F. After remaining ten minutes it is wrapped in a blanket and replaced in bed. Cold baths (75° to 60° F.) reduce the tem-

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[1] I have no personal knowledge of an outbreak of this kind in America, but I witnessed one in Paris in 1893, in which the disease spread very rapidly and was extremely fatal.

[illegible], and, what is far more important, produce a nervous reaction which rouse the child from the condition of apathy and somnolence which often precedes death. The action of the heart is strengthened, arterial tension increased, and expectoration produced. It is the only method which has yielded uniformly good results in acute cases of very young children and infants,—a fact which is so well recognized that it forms the leading feature of routine treatment at all the children's hospitals in Paris at the present day. The child is given a stimulant, or a hypodermic injection of caffeine (three to five grains), and plunged into the bath, where it remains, carefully watched, for five or ten minutes, and is then wrapped in a blanket and put to bed. I believe that life is saved in this way, and I am sure that it is often prolonged. As a substitute for baths, the wet pack may be used, the temperature of the water being made the same. The child is wrapped in a wet sheet, covered with blankets, and remains "packed" for an hour or two. I do not believe that in cases which show a marked tendency to somnolence and cyanosis the effects of this measure can compare with those of the cold plunge; but it is certainly a useful procedure, and excites no spirit of opposition on the part of the attendants. The cold bath and wet packs are repeated every four or six hours, as may be required.

There are but few drugs which can possibly do good in a case of acute broncho-pneumonia which suddenly attacks an infant. Expectorants are worse than useless. Emetics hasten death. Brandy, digitalis, and caffeine are beneficial, and should be given from the start. Quinine as an antipyretic is apparently impotent in the presence of a temperature of from 105° to 106° F., while the effects of the coal-tar preparations are very temporary. To support the heart and reduce the temperature are the obvious indications, and these are best accomplished by the cold bath and employment of heart tonics and stimulants.

℞ Sodii benzoat.,
Caffeinæ, āā ʒss;
Aquæ dest., ʒiss.
Fifteen to twenty minims by subcutaneous injection every six hours.

Brandy and digitalis should be given in small but frequently repeated doses. Both digitalis and caffeine increase diuresis, and in this way aid in the elimination of the poison. In more protracted cases good evidence of accumulation of mucus in the tubes is an indication for emetic doses of ipecac, and the relief obtained in this way is quite marked. Stimulating expectorants may also prove useful here; and when convalescence is prolonged, and the physical signs show that definite areas of lung are obstinate in healing, counter-irritation is of value, and may be readily effected by other means than the tedious process of painting with tincture of iodine. The skin is washed clean with sulphuric ether, and the part to be counter-irritated defined and limited by a line of vaseline around its edges. Equal parts of carbolic acid and alcohol are applied with a soft brush; the skin is observed to turn white two or three minutes later, and the superfluous acid is then rapidly removed with an absorbent cotton swab dipped in pure alcohol. The result is not a blister, but an intense congestion of the superficial capillaries, which lasts ten days or more and serves the purpose excellently. In case of an unusually tough skin the proportion of acid may be increased. Some slight pain attends the application, but it is temporary, and by the time a layer of cotton-wool has been placed next the skin as a protection, and the child is ready to lie down, all discomfort has ceased. Tonics and digitalis—the latter by strengthening the arterial circulation and relieving venous stasis—both aid in the repair of the more chronic lesions of broncho-pneumonia. Cod-liver oil, iron, malt, strychnine, and preparations of tar are useful. When the child is so fortunate as to live through the acute stage of the disease, somatose and predigested starch preparations should be given promptly. The latter may not be ideal substitutes for breast-milk, but are certainly nourishing and easily absorbed. Moreover, few children young enough to nurse live through the third day of a broncho-pneumonia.

To recapitulate. Brandy in every case; digitalis in most cases. Tepid baths or the cold wet pack when cyanosis is not marked and nervous agitation is present. Cold baths and hypodermic injections of caffeine, brandy, or ether when cyanosis, somnolence, and a very high temperature (separately or together) are observed. Emetic doses of ipecac and stimulating expectorants when there is evidence of accumulation of mucus in the bronchi. Counter-irritation, digitalis, and tonics to hasten absorption in protracted cases.

PULMONARY TUBERCULOSIS.

By A. JACOBI, M.D.

THE following pages are meant to supplement, not to repeat, the article on "Phthisis," and, as far as it concerns pulmonary tuberculosis, that on "Tuberculosis," contained in the second volume of the "Cyclopædia of the Diseases of Children." Hence the remarks on the varieties and the pathological anatomy as well as on the symptomatology of pulmonary tuberculosis will be found brief. The attention of the reader is mainly asked for the chapters on etiology and on the preventive and curative treatment, which may be found interesting when read in connection with those on the same subjects in the publication of 1889.

I. NATURE AND DEFINITION.

The question raised in the article "Phthisis" in vol. ii. p. 670, concerning the identity of "phthisis" and pulmonary tuberculosis, was answered affirmatively. Still, as is stated on p. 165, vol. ii., Malassez and Vignal succeeded in the experimental creation with or without co-operation of bacilli, by zooglœa, of tuberculous masses and small "tubercles" in which nothing but zooglœa were found. Similar microbes were met with in bone-abscesses by Castro and Soffia two years later (in 1885). Ribbert examined in 1884 miliary tubercles and found no bacilli, but big and giant cells, and preferred the name of multiple lymphoma for that reason. Biedert, however, when having the same experience, suggested that the occasional presence in the blood or in pus or serum of some additional alkali, which might interfere with perfect staining, explained the absence of bacilli. Eberth produced in guinea-pigs tuberculosis which contained no bacilli and was therefore called pseudo-tuberculosis. Eppinger found in such tubercular infiltrations *not* bacilli, but cladothrix, and called his product pseudo-tuberculosis cladothrichica. But all these exceptional facts or products should not militate against the acceptance of the essential unity of the tubercular process, which cannot exist without the bacillus tuberculosis (Koch), no matter whether it is found in the lungs, bones, glands, or other tissues.

Tubercle bacilli retain their infecting power a long time. But they *proliferate as parasites* only, and even become less virulent by passing, in the course of experimentation, through a series of living bodies. They also suffer from putrefaction and persistent desiccation to such an extent as to cause merely local affections with a tendency to heal, but no general infection; and those originating in the animal body seem to be less dangerous than those raised in the human body. Thus a certain comparative safety is afforded, after all. When an invasion of bacilli has taken place, their first effect is a copious proliferation of tissue cells, particularly of those of the connective tissue. Epithelial and giant cells are formed in large numbers; their nests show a sharp delineation; leucocytes increase in their neighborhood by emigration. These processes, together with the formation of a surrounding capsule, take from ten to fourteen days after invasion (inoculation); then the "tubercle" is perfected. When there are many round cells in the composite mass the product is called a lymphoid tubercle; when there are but few of them (particularly in the periphery), the mass is accordingly called a large cell (or epithelial) tubercle. The enclosed bacilli are sometimes destroyed by pressure and become harmless; if not, a new infection may take place in the neighborhood, or bacilli are spread by the blood, and still more by the lymph-currents. The tubercle is devoid of blood-vessels, and therefore a disintegration of the central mass is of frequent occurrence. This disintegration spreads in the direction of the periphery and causes the tubercle to look yellowish white. This "*caseous*" degeneration is mainly observed in larger tubercular masses, but also in the smaller "miliary" deposits. Caseation is very liable to terminate in softening, rarely in calcification. The latter is equivalent to complete recovery; the former is a constant source of local irritation, of new bacillary invasion, of repeated bronchial and peribronchial congestion and diffuse infiltration of the pulmonary tissue (caseous pneumonia). It should not be forgotten, however,[1] that caseation does not necessarily mean tuberculosis, for it is also met with as the final transformation of suppuration, or of cancerous or of typhoid infiltration. Nor are giant cells characteristic of tuberculosis. Without the presence of the bacillus tuberculosis the diagnosis of tuberculosis should not be considered complete.

II. DISPOSITION.

The *disposition* to tuberculosis of the lungs is partly local, partly general. The relative smallness of the heart (Brehmer, Fels) and the contraction or narrowness of the pulmonary artery render the tissue of the lungs anæmic. Shortness of the cartilage of the rib, found to be congenital by Freund forty years ago, and rhachitical alterations of the chest interfere with respiration and circulation. The rhachitical depression, mainly acting upon the lower part of the chest, cripples particularly the lower lobes of the lungs and prepares them for inflammations and tubercular deposits. Infants puny at birth, twins, babies born in rapid succession or of anæmic and poverty-stricken parents, overcrowding in ill-ventilated, smoky dwellings and school-houses, previous catarrhal pneumonia, whooping-cough, measles, or influenza, trauma by blow or fall, glandular enlargements, bone-disease, or cold abscesses anywhere in the body, persistent eczema or furunculosis, chronic nasal catarrh or ulceration, pharyngitis, amygdalitis ("tonsillitis") of various kinds, sometimes even chronic gastritis or enteritis, offer under favorable circumstances just as many inlets to pulmonary tuberculosis. Other infectious diseases, such as scarlatina, croupous pneumonia, or typhoid fever, do not so frequently create a predisposition. Like rhachitis, which does not act by its mechanical results only, but by the general anæmia caused by it, scrofulosis, not identical with tuberculosis, prepares the soil for it by its constitutional anomalies; for scrofula is characterized by vulnerability of all the tissues, the long duration of and slow recovery from superficial or deep-seated lesions, the frequency of relapses, the rapid disintegration of newly formed tissues, and the enlargement of glands and their tendency to caseous degeneration. It is particularly the latter organs which are often connected with the first symptoms of pulmonary tuberculosis. In two ways the bronchial (and other) glands may become responsible for them: either the bacilli enter the vulnerable glandular tissue with greater facility (for even healthy epithelia and mucous membranes allow bacilli to penetrate), or the capsulated and dormant bacilli, unless destroyed by pressure, are waked up and disseminated by the chronic congestion of the parts.

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[1] See vol. ii. p. 165.

It has been intimated above that every disturbance of general or local metamorphosis creates a disposition to tuberculosis by impairing blood and tissues. That is why bad innervation from loss of blood, slow convalescence, care and trouble (in the older child and the adolescent), and also why colds, have their bad influence and predispose to pulmonary tuberculosis. Indeed, the dangers of sudden exposure and of abrupt changes of temperature, which alter the circulation both of the surface and of the deep tissues, either directly or by reflex action, are too evident to be reasoned or smiled away by the fad of recognizing nothing but microbes as the sole and omnipresent sources of every ailment or malady.

In close connection with this subject is that of the danger arising from bad or insufficient *air*. Density of population, crowded rooms, uncleanliness, and closed windows work in two directions. By the exhaustion of oxygen and increase of carbonic acid and noxious gases they impair the blood and tissues. By the accumulation of bacilli on floors and walls and in the air they disseminate the almost ubiquitous malady, and the other detrimental influences—dirt, improper food, and so on—are liable to go hand in hand with those enumerated above. This is equally the case in animals and in man. The report of Dr. E. W. Hope, medical officer of health of Liverpool,[1] is particularly interesting. According to him, the cow shipments in Liverpool are under the immediate supervision of two well-qualified inspectors, who attend to the legal requirements as to light, ventilation, and cleanliness, while no such supervision is required in the country. The result is that of one hundred and forty-four samples of milk taken from sources within the city, in three, or 2.8 per cent., was found the bacillus tuberculosis, while of twenty-four taken at the railway stations, 29.1 per cent. infected the guinea-pig.[1]

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[1] British Medical Journal, July 17, 1897.

A disposition is also created by a *defective condition of the respiratory tract.* Though bacilli may be swept through a normal tissue, it ought to be taken as a fact that, as long as a bronchial mucous membrane is covered with normal mucus and protected by vibrating epithelium, foreign bodies, from particles of metal and carbon to bacilli, are liable to be expectorated. *Only the air-cells which have no fimbriated epithelia allow bacilli to rest and develop with greater facility.* All the other surfaces of the respiratory organs are endowed with means of self-defence. This is, however, greatly interfered with either by the abnormal structure of the integuments or by actual lesions. The latter need not be direct, as in whooping-cough or measles; they may be indirect. For but lately Kohler[2] published a case which makes it probable that the disposition to tuberculosis may be increased or a previous affection may relapse as the result of a trauma, but also that when an injury or a concussion of the thorax is not very conspicuous, their effects may be long delayed.

Abnormal structure of the integuments may either be inherited from parents suffering from chronic infectious diseases, such as tuberculosis or carcinosis, or acquired by previous exhausting ailments, anæmia, or chlorosis (vol. ii. p. 169).

The question whether *vaccination* may cause tuberculosis or scrofulosis has been in Germany the subject of an official inquiry, and replied to lately by Gerhardt and Leyden. They refer to the fact that the discovery of the bacillus excludes the possibility of such a causal connection except in those rare cases in which the fever attending vaccination may be assumed to favor the proliferation and dissemination of bacilli previously lodged in tubercular lymph-bodies, or in which erysipelas, sepsis, or syphilis is caused by a gross mistake in vaccination. They also point to the frequency of tuberculosis at all times, and emphasize that it has certainly not increased since vaccination was made compulsory; that, on the contrary, in the armies of the German empire, since vaccination was made obligatory, the mortality from tuberculosis has decreased. These experiences and the conclusions therefrom tally with mine as stated in vol. ii. p. 171.

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[1] Some of the facts reported to prove the absolutely favorable influence of fresh air are rather startling. When Hutinel makes the statement that among eighteen thousand children in charge of the Paris "assistance publique" there were but twenty cases of tuberculosis, we are—considering the immense mortality of these children—tempted to ask whether they lived long enough to develop the disease. S. Bernheim reports the cases of three pairs of twins born of three women: one of each pair was supplied with a wet-nurse and kept at home, and died of tuberculosis; the others were fed artificially, but in the country, and remained healthy. Such results, if not too surprising to be believed, are too exceptional to establish a rule. It looks as if they proved too much.

[2] Viertelj. f. gerichtl. Med., July, 1897.

The small vaccination wounds do not count. They cannot be compared in any way with those of *circumcision* or other operations whose occasional influence in admitting tuberculosis was discussed in vol. ii. p. 170, nor with the long-lived erosions and sores of *eczema* and *impetigo*. Whenever bacilli are admitted in this way, the result is more apt to be a generalized miliary than a pulmonary tuberculosis.

III. ETIOLOGY.

The origin of pulmonary consumption is in almost all instances attributed to the *inhalation* of bacilli. As they are certainly deposited on bedding, clothing, and on the floors and walls of rooms, nothing appears to be easier than that the long-lived microbes should be admitted to the air of the room and thus be inhaled. In this way the contagion of acute exanthems is certainly disseminated. Tubercle bacilli, like everything solid, when floating in motionless air, are certain to sink gradually, and the inference is that children are more liable to inhale them, a mode of infection which I have claimed to be probable in cases of diphtheria.[1] This mode of propagation has been taken to be the principal one in pulmonary tuberculosis. In vol. ii. p. 169 I said that the bacillus, "to be inhaled, must be dry. As long as sputum is moist, or, after having been dry, is again exposed to moisture, it cannot be mixed with the air and thus enter the lungs of another person. . . . The phthisical patients in the wards of a hospital are uninjurious as long as no expectoration is permitted anywhere but in a spittoon containing some water." To such an extent has this belief controlled the teachings of medical men that the rules and regulations of health departments have concerned themselves with this mode of transmission only. Experiments, however, appear to prove that the air-currents usually found in a room are not sufficient to detach dry bacilli fastened with their surrounding sputum to the walls or floors. It is only strong currents, such as are caused by sweeping, beating, brushing,—perhaps even by violent slamming of doors,—that will float them. Under these latter circumstances it is certainly possible that dry bacilli may be detached in this way and infect those present. But experiments on animals have not yet proved that they could be infected by inhalations thus conducted; and it is quite possible that boards of health will have to alter or rescind both their opinions and the practical rules built on the foundation of former knowledge.

But lately Flügge[2] published a long series of experiments and observations which appear to be able to stand accurate tests. Crying, sneezing, coughing, even talking, detach sputum in more or less invisible quantities. Everybody's experience yields such instances—palpable ones—in the sick and the well. Such *moist particles*, mostly infinitely small, were proved to remain in the air of a room five hours. Indeed, an air-current of from one to four millimetres in a second (equal to from twelve to fifty feet an hour) sufficed to float them for that length of time. In this manner the contagiousness of pulmonary tuberculosis is even more pronounced than by assuming the dry sputum to be the only means of conveying the disease, and the direct transmission from husband to wife or children, or between patients in a hospital ward or sanitarium, becomes almost a matter of course, so that the medical and humanitarian devices planned on hitherto imperfect knowledge require a far-reaching revision.

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[1] Treatise on Diphtheria, 1880.

[2] Zeitsch. f. Hyg. u. Inf. Krankh., Bd. xxv., 1897.

However, the occurrence of *direct contagion* is difficult to demonstrate as long as one-seventh of the population of the temperate zone dies of pulmonary tuberculosis, and as long as the inroads into the circulation on the part of bacilli are so numerous, indeed, and often so mysterious, that Bollinger speaks of "cryptogenetic origin." It will be stated in another place that the initial lesion need not correspond with the locality of invasion; subcutaneous infections cause pulmonary lesions; lymph-nodes are diseased without an affection of the corresponding mucous membranes, and represent the "latent" form of tuberculosis. It has been the tendency to underestimate the amount of direct contagion. A committee of the French Academy examined the cases of two hundred and thirty-three consumptive couples; in about twenty direct contagion could be found. Still, if the experience of the thousands of general practitioners be consulted, the results will be different.

It has also been stated that nurses and employees in hospitals and State prisons, as long as they are not overworked and underfed, do not become tubercular; but other reports prove the danger to which nurses in consumptive wards are exposed. Cases of tuberculosis occurring in a hitherto healthy family after the return of a consumptive member are quite frequent. The increase of tuberculosis in proportion to the density of the population points in the same direction; and cases like that of Demme, who boarded a healthy baby with the family of a consumptive man, and acquired for it at the age of eight months ozæna, bacilli in the nasal mucus, and tubercular meningitis, are not at all exceptional.

Direct contagion is perhaps at no time more readily accomplished than immediately after birth, when the baby is kept in bed with the mother and exposed to the dangers of contact. What I stated as the result of Flügge's recent experiments and the facility of inhaling the finely distributed particles of contaminated sputum explains best the unfortunate situation of the newly born. Fröbelius, whose babies were transferred from the obstetrical wards to the foundling hospital after a number of weeks, had a mortality from tuberculosis of 0.4 per cent. of all his deaths in the first year (21.7 per cent. general mortality in 91,370 infants from 1874 to 1883). Epstein, who transferred immediately after birth, had none at all.

Direct *hereditary* transmission was discussed by me in vol. ii. pp. 166–168. There are still those who are convinced that there is no congenital tuberculosis in man;[1] those who believe in its direct transmission;[2] those who suspect its existence, though the malady exist in relatives only;[3] and those who think it but rare. That it occurs in some instances, at least, there can be no doubt. If there were but isolated cases like those reported by me (vol. ii. p. 167) and by Birch-Hirschfeld, who found bacilli in the liver of a calf extracted by Cæsarean section, the question would be decided affirmatively. Meanwhile the list of undoubted cases is longer.

Hereditariness of tuberculosis and hereditary disposition should not be considered equivalent, as has been shown elsewhere. It is true it is difficult to discriminate between the two in the case of a tubercular infant of a few weeks or months. For the younger the tissue, and the more copious its proliferation, the less is its resisting power. It is a well-established fact that pathological changes are most liable to take place during the period of most active physiological growth. Moreover, the formation of a tubercle need not take more than ten or fourteen days after the invasion of a bacillus. On the other hand, there is no reason why tuberculosis, like syphilis, could not be stowed away in a single organ, and remain latent, or dormant, for an indefinite period.

Hereditary transmission may be either placentar or germinative. The placenta, when healthy, is a perfect filter, but, in spite of this perfection, anthrax, malaria, variola, and so on, are known to pass it, and tubercle bacilli have been found in the blood of the umbilical vein, the liver, spleen, and kidneys of the fœtus. In connection with this subject I do not care to emphasize the finding by F. Lehmann of giant cells in the villi of the chorion, for giant cells are not exclusively found in tuberculosis, but also in sarcoma, syphilitic endarteritis, and other conditions. The possibility of germinative transmission is proved by Iahni and Weigert, who found bacilli in the juices of testes and prostates without tubercular alterations of those organs; by Spano, who found them in sperma; by Maffucci, Baumgarten, Pänder, and Gärtner, who injected bacilli into the external layer of the egg-albumin and caused the chick to be tubercular. It appears, however, that a certain number of bacilli are required to cause an infection. To have that effect, a bacillus culture diluted in a proportion of one to four hundred thousand was required in subcutaneous injections, one to one hundred thousand was demanded for inhalation, and one to eight for feeding. When but eight (up to forty) bacilli were injected, no infection took place. If, therefore, a certain condensation or number be required, it becomes doubtful whether one ovule or spermatozoon may be sufficiently saturated with them to cause during cohabitation an hereditary transmission. Besides, not all the bacilli are of equal efficacy. Still, as from forty-five to seventy-five millions have been calculated to dwell in a single cubic millimetre of sputum, the number of the microbes is so incalculable as to suggest their power to do harm wherever their presence, though in small number, is discovered.

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[1] Biedert, Handbook, p. 93.

[2] Liebermeister.

[3] Lorey.

Age.—There are not many additional facts concerning the occurrence of tuberculosis in early age since the data published in vol. ii. p. 671. It becomes, however, more and more evident that it is very frequent. J. Mackenzie states that of 1591 consumptives who died in London hospitals, only 65 were under fifteen years; but we do not learn to what extent children were admitted, as compared with adults. Among 3575 deaths from tuberculosis in one year, there were in Berlin 95 under one year, 89 from one to two, 91 from two to five, 38 from five to ten. Of Demme's 59,000 sick children, 2410 (4 per cent.) were tuberculous; 87 of them died of acute tuberculosis. Of all the deaths occurring in Tübingen under one year, there were, according to Dennig, of tuberculosis 25 per cent.; from one to two, 20; from two to three, 8.3; from three to four, 6.7; from four to five, 11.7; from five to six, 3.3; from six to seven, 6.7; from seven to eight, 3.3; from eight to nine, 6.7; from nine to twelve, none; from twelve to thirteen, 3.3; from thirteen to fourteen, none; from fourteen to fifteen, 5 per cent. Calculated in periods of five years each, the first yields, among all the causes of death, 71.7 per cent. due to tuberculosis, the second 20, the third 8.3 per cent. For the same periods, O. Müller furnishes 50, 26, and 23.3 per cent. Altogether, in 500 autopsies made at Munich, he found tuberculosis in 40 per cent. L. Emmet Holt has added valuable material, thus refuting for these last years my remark (vol. ii. p. 167) of ten years ago that New York institutions had failed to contribute to our statistical knowledge by publishing the results of 1045 autopsies made in the New York Foundling (Northrup) and the Babies' Hospitals. In the 1045 autopsies, tuberculosis was found in 14 per cent. in those of the Babies' Hospital (sick infants only admitted); in the Foundling Hospital (admission indiscriminate, sick or well), 10 per cent. Total number of Holt's cases, 119.

In these 119 cases the lungs were affected 117 times, the pleura 69, the bronchial lymph-nodes 108, the brain 40, the liver 77, the spleen 88, the kidneys 46, the stomach 5, the intestines 40, the mesentery 38, the peritoneum 10, the pericardium 7, the endocardium 1, the thymus 3, the adrenals 2, the pancreas 3.

Among the organs accused of being the direct cause of pulmonary tuberculosis is the *nose*. It contains cocci and bacilli of all sorts, for no organ is more accessible; indeed, no cavity is free of microbes, not even (according to Zaufal) the middle ear. On the Schneiderian membrane and all over the naso-pharynx they are met with in large numbers; adenoid growths are covered with them; some writers go so far as to claim them as positively tubercular. Dieulafoy inoculated sixty-one guinea-pigs with the secretion of healthy persons; eight of the animals became tubercular. That does not prove anything, however, but that the surfaces of our integuments may be and are covered with all sorts of noxious elements which become pathogenous only when causing ulcerations, or when carried, by accident or intentionally, into the circulation. In the same way several infectious diseases result from inoculations made from the scrapings of a healthy mouth.

Primary tubercular ulcerations of the nose are acknowledged to be quite rare. It should not be overlooked that, while bacilli are frequently found and catarrhal erosions are numerous, the latter should not be taken for tubercular. On the other hand, it is claimed that normal nasal mucus is bactericide. If that be true, as microbes are ubiquitous, most nasal discharges would no longer be "normal" if the microbes remained active enough to cause an infection. Still, a modern author utters the startling news that "retro-nasal catarrh is the main foundation of pulmonary tuberculosis."

According to others, this exclusive claim of the naso-pharynx seemed ill founded. Their attention was mostly fixed on the *tonsils*. Like the larynx, the tonsils were found to become infected by expectoration. When pulmonary tuberculosis was not very extensive, or the disease was confined to the bones, or the mucous membrane of the pharynx was either not much inflamed or was cicatrized, and the tonsils were small, hard, and pale, the latter were mostly found not to be much affected, and even when bacilli were found in or on the tonsils, in which no caseous degeneration was shown, the cervical lymph bodies would mostly be exempt. But, on the other hand, it has been claimed that in nearly all cases of pulmonary tuberculosis the disease is also in the tonsils;[1] that it begins in the superficial lacunæ, the morbid contents of which are forced into the tissue by the act of deglutition; and, finally, that while pulmonary tuberculosis is almost always accompanied by that of the tonsils, the latter is not only attended by but causes the former.

What I mean to insist upon is this: that undoubtedly bacilli are found frequently in the nose and in the pharynx. But their presence does not mean tuberculosis. Nor does it appear that tuberculosis of the lungs is (often) occasioned by their presence. Among the four hundred and sixteen autopsies of Fröbelius on tubercular infants the lungs were affected in every case, the pharynx in none. When the pharynx and naso-pharynx are tuberculous they are liable to infect the neighboring lymph-nodes first. Moreover, it appears that if intestinal and mesenteric tuberculosis is apt to be present with that of the pharynx, that may be so in consequence of a uniform morbid disposition on the part of the whole intestinal tract. This uniformity of morbid disposition is a well-established fact on other—for instance, the respiratory—tracts also.

IV. SYMPTOMATOLOGY.

The symptomatology of chronic pulmonary tuberculosis is amply discussed in vol. ii. pp. 676–678. The difficulty of diagnosis in many cases has not been lessened. Frequently it is still the totality of, in part, insufficient symptoms that is to be consulted. Percussion never yields a reliable result except when quite gentle; auscultation may be valuable, but in some cases it reveals nothing whatsoever. The normal puerile respiration may be a little coarser, generally or locally. The usual symptoms of bronchitis are sometimes all that can be found. Suddenly, now and then, a localized bronchial respiration can be heard corresponding with a local dulness, for infiltrations take place quite frequently. They may disappear again, or they may migrate; some or all of the deposits may either disappear or remain. Respiratory murmurs may change. A bronchial respiration which was of long standing may temporarily disappear because of the obstruction of a normal or the filling up of a dilated bronchus. The most characteristic auscultatory symptom is the *persistence*, in a given locality, not always near the hilus or in an apex, of a subcrepitant râle. Cavities are rare, and generally small. They are difficult to find, and often their diagnosis is deceptive,—more so than in the adult. Percussion that yielded dulness near a clavicle may not do so after a while; an infiltration may contract; the neighboring pulmonary tissue may in part cover the hardened spot, or may become emphysematous and yield a semi-tympanitic sound in place of the former dulness. This emphysematous change does not so readily take place in cases of extensive interstitial pneumonia (peribronchitis), which finally results in retraction of an extensive part of the pulmonary tissue, preferably in the upper lobes.

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[1] E. Schlesinger, Berliner klinische Wochenschrift, September, 1896.

A heart-murmur may accompany chronic tuberculosis. When it is not cardiac, it may, in rare cases, result from compression, by cicatrizing tissue, or by an infiltration, of the pulmonary vein.

In connection with what was said on p. 676, vol. ii., on the *fever* accompanying chronic tuberculosis of the lungs, it will be remembered that the temperature has no particular type. It is sometimes higher in the morning and at noon than in the evening; frequently it is only the expression of the collateral bronchitis and pneumonia, therefore in the course of time it may be high or low alternately. When suppuration sets in it may be hectic. In acute miliary tuberculosis of the lungs, which may occur without for some time being complicated with general tuberculosis, the temperature is generally higher,—104° F. and more, sometimes less. Miliary tuberculosis may be confined to a part of the lungs; then the temperature may be moderate. It may become normal after a while, to rise again when a new miliary infiltration takes place in the same or another portion of the lungs.

The condition of the *blood* is not materially changed. What alterations there are result from accompanying septicæmia or fever (red cells diminished), or from hæmoptysis, extensive infiltration, the presence of cavities, and from pyogenic fever (leucocytosis).[1] Some changes of the blood which have been noticed in (tuberculosis of) the nursling, such as the presence of eosinophile cells and of occasional nucleated red cells (normoblasts), are physiological and not pathological.[1]

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[1] R. C. Cabot, A Guide to the Clinical Examination of the Blood, 1897.

Nor is the condition of the *urine* conclusive of tuberculosis. It was believed to exhibit an undue amount of indican. The latter is found in proportion to the atrophy or the increased intestinal putrefaction which accompanies chronic tuberculosis, and to its complication with bronchopneumonia and the inflammation of serous membranes.

V. COMPLICATIONS.

Complications with tuberculosis of the *stomach*, the *intestines*, and the *mesenteric glands* are not so frequent in children as they are in adults. Indeed, the stomach is rarely affected. Primary tuberculosis of the intestine is very rare indeed, and but few trustworthy examples are found in the literature besides those furnished by Demme. The mesenteric glands are the seat of tuberculosis far less frequently than the bronchial glands (one to ten). Therefore the number of cases of tuberculosis acquired by inhalation exceeds immensely those depending on intestinal infection. In those children whose gastric juice is not sufficient, or is entirely absent, bacilli may pass the stomach and prove injurious. Tubercular mothers who (not an uncommon practice) chew their children's food before giving it to them may thus transmit their disease. In the milk of tuberculous cows bacilli are, as a rule, found only in the presence of general miliary tuberculosis and of tubercular mastitis. Thus direct transmission through milk is an exceptional occurrence. Still, it does occur, and therefore pasteurization or sterilization is demanded as a matter of safety. It should not be forgotten, however, that the successful inoculation of milk bacilli into guinea-pigs does not prove that the same danger attends the same milk when it is introduced into the human alimentary tract.

The *kidneys* of tuberculous young children suffer like those of tuberculous adults. Amyloid degeneration and chronic diffuse nephritis, toxic nephritis of variable severity, and congestive conditions in different stages depending on venous obstruction are not uncommon. Besides, transitory albuminuria, with or without an excess of phosphates or of urates, or of both, is frequently observed, mostly in very anæmic children and those in whom hereditary disposition to tuberculosis is well marked. That it is common in tuberculosis of the kidneys, which appears to complicate that of the lungs quite often, even before marked symptoms of nephritis are in evidence, is readily understood.

VI. DIAGNOSIS.

The surface alterations of scrofulous children are closely connected with the lymph-bodies and lymph-circulation, which is more active, as the vessels are larger, at an early age. To distinguish between the scrofulous and the tubercular nature of the swelling of the lymph-bodies we have no means besides their bacteriological examination. The main question is whether in the first period of scrofulous affections the tubercle bacillus can be found. Litten[1] reports the results of nineteen autopsies of scrofulous children. In one only a few bacilli were found in the lymph-bodies; in that case there were also the symptoms and pathological changes of pulmonary tuberculosis. Nor were other scrofulous symptoms identical with tuberculosis; no bacillus was found in one hundred and twenty-nine cases of dry or vesicular eczema. Of forty-two softened lymph-bodies of the neck and thirteen extirpated glandular conglomerates, three only had scanty bacilli; these three were complicated with extensive lesions in the glands and bones, one of them with such in the knee-joint. Twenty-three children with acute multiple suppurations of the subcutaneous tissue had no bacilli; the result was also negative in five cold abscesses with thin pus, but positive in seven with thick, caseous pus and proliferating membrane. There was no bacillus in the catarrhal secretions of the noses, the ears, and the eyes of one hundred and thirty-eight scrofulous children. Thus in the initial stages of scrofula there are no tubercle bacilli, therefore these cannot be the causes of scrofula, nor are they the sources of the peculiar vulnerability and the singular form of chronic inflammation characteristic of scrofula.

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[1] Hock and Schlesinger, Beitr. z. Kinderheilk., ii., 1892.

VII. PROGNOSIS.

The remarks on *prognosis* contained in vol. ii. p. 679 are correct as far as our present and past knowledge goes. To them I beg to refer the reader. Prognosis is mostly grave, in many instances doubtful. Pulmonary tuberculosis may practically heal, however; that is proved by the autopsies, which reveal the presence of caseous, indurated, even calcified tubercular infiltrations which either were or never were diagnosticated during life; but even in such bacilli retain their vitality a long time, and may be enabled any time to renew their virulence and proliferation by the occasional influence of bronchitis, pneumonia, Koch's tuberculin, or other irritants. Therefore, not to speak of the unfavorable prognosis furnished by extensive infiltration or cavities, a dormant or an apparently recovered case, in spite of ample nutrition, fresh air, exercise, and cold water, ought not to be pronounced cured without mental reservation.

VIII. PREVENTION AND TREATMENT.

The peculiar *scrofulous condition* of the tissues, mainly of the skin and mucous membranes, with their passive congestion, facile disintegration of the epithelia, and sluggish recovery after injury or disease, is the very soil for the invasion of tuberculosis. Identity of the two conditions does not exist except in an erroneous diagnosis.[2] The very efficacy in scrofula of remedies which are not borne at all in tuberculosis would prove the differences in the nature of the two. The treatment of scrofulous infants and children has the purpose of rendering the tissues more capable of resisting the invasion of infectious processes. Sea-bathing and the use of mineral springs containing iodine, such as St. Catherine's or Kreuznach, the systematic use of cold water, with friction of the surface, the occasional administration of diaphoretics, and the persistent use of iodides (of potassium, sodium, or iron) and of cod-liver oil, will be required in scrofula. Of all this medication the use of cold water to harden the skin and to stimulate and strengthen both the cutaneous and the general circulation, and of cod-liver oil, is the only treatment applicable both to scrofula and to established tuberculosis.

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[1] Berliner klinische Wochenschrift, 1897, N. 28.

[2] See article by Ashby, page 317 of this volume.

Among the *preventives* of pulmonary tuberculosis the *destruction of the tubercle bacillus*—easily accomplished outside the organism—before its entrance into the lung-tissue takes a high rank. No internal remedy, however, will prove effective, for no living tissue bears disinfectants strong enough to annihilate the microbe. Whenever lymph-bodies, cavities, sinuses, ulcerations, joints, or bones are affected with tuberculosis and the disease is almost certain to spread, timely surgical interference will prevent the extension of the morbid process. A tubercular gland must not be permitted to suppurate and burst, but should be enucleated in time. Tubercular *abscesses* and *fistulas* should be scraped out, the pyogenous membranes removed, and the surface kept disinfected until recovery is completed. Tuberculous joints and bones may require exsection, but in most cases repeated injections of *iodoform emulsions* (better than solutions), with the occasional removal of loose particles, will prove effective. Another method of conservative treatment has been introduced by A. Bier. Encouraged by the fact that lungs in a condition of passive hyperæmia resulting from cardiac disease or from kyphosis have a rather pronounced immunity from tuberculosis, he advised the production of a passive venous congestion of the tubercular joints by bandaging the limb below the affected joint and compressing it above with an india-rubber band. To secure a moderate amount of hyperæmia and local cyanosis only, the bandage should be loosened once or twice daily. Such a degree of passive hyperæmia is known to give rise to the new formation of connective tissue and to induration, which is expected to afford a certain degree of protection[1] against the proliferation and action of bacilli. That is what Landerer expected to accomplish by the local injection near the joint of *cinnamylic acid*, and Lannelongue by that of a ten per cent. solution of *chloride of zinc*. That is also the effect Koch meant to attain when he advised *tuberculin*. The remedy was expected to light up a sufficient amount of interstitial irritation and consecutive hyperplasia to encapsulate the bacilli and render them innocuous.

The bacilli should be destroyed in *milk* and *meat*. Pasteurization of the former and thorough heating of the latter are all that is required. Muscles are seldom tuberculous, more frequently the kidneys, spleen, and liver are; it is principally the latter which require attention. After all, tubercular infection from these two sources is not frequent, but ought to be, and may be, avoided altogether. (Vol. ii. pp. 172, 182.) When cow's milk is suspected, the buttermilk, butter, or cheese made of it should not be eaten. The milk of a tubercular mother or wet-nurse may be dangerous; what is more so is the direct contact of the baby with the tuberculous patient and the inhalation of bacilli.

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[1] See my Therapeutics of Infancy and Childhood, Philadelphia, 1896, p. 473.

As preventives and curatives, proper feeding, clothing, and the hygienic treatment of the *skin* (vol. ii. p. 682) are of the first importance. Nothing of any account could be added to former rules. As to the *climatic treatment* of pulmonary tuberculosis, I refer to vol. ii. p. 680. I think I have given these matters much attention then and since. The remarks I then made I could but repeat now; the briefness of this reference to what I consider most important should not be a temptation to think of bacilli first and last and of hygiene least. The most urgent indication is always to protect the organism against an invasion, and to fortify it and enhance its powers of resistance.

Among the *preventives*, as a means of diagnosticating tuberculosis in cattle I should mention tuberculin. After it failed as a healing agent, the attempts at reducing its danger and rendering it a positive remedy have not been given up. Tuberculoidin and *tuberculocidin* (Klebs) were obtained by clearing the tuberculin of its bacilli by treating it with alcohol, but the confidence these modifications commanded was not greater than that placed in a serum recommended by Maragliano, which seems to contain no antitoxin, or the *cantharidin* introduced by Liebreich. But lately Koch recommended a *new tuberculin*, obtained by disintegrating dry bacilli by a mechanical process (the latter is claimed by Buchner, a priority question we have nothing to do with). The new tuberculin, which contains the insoluble parts of the bacilli (while the old was said to contain those soluble in glycerin), is claimed to possess a positive immunizing power. It is sold in vials holding one cubic centimetre (=15 minims = 15 grains). One per cent. of it is the dry material of tubercle bacilli in a condition of mechanical disintegration. The first dose to be injected into the subcutaneous tissue of an adult should be one-five-hundredth of a milligramme of the dry substance (one-fifth milligramme of the fluid). To obtain this dose, one part (one minim) of the fluid tuberculin may be mixed with five thousand parts of a preserving fluid. One minim of this mixture contains the required dose of one-five-hundredth of a milligramme. The solvent is a six per mille salt-water solution containing twenty per cent. of pure glycerin. Salt water alone will not preserve. Injections should be made every other day, and the temperature should be watched. An elevation of more than ½° C. should be avoided, and no new injection be made until the temperature is again normal. Slowly the doses should or may be increased to twenty milligrammes of the dry substance (two cubic centimetres, two original bottles of the fluid). If there be no reaction, it is best to desist, or to inject at long intervals only. Before this amount is reached, the injections ought to be made once or twice in a week.

There are cases, however, without any reaction. A patient of mine, who died in Bellevue Hospital lately of pulmonary tuberculosis, received from me a daily injection of the new tuberculin. The first dose was one-tenth of a minim, which was carefully but persistently increased. Within a fortnight the dose reached three minims, with no effect on the temperature whatsoever.

The exaggerated claims of the old tuberculin are not repeated for the new. This is said by Koch to exert its influence in the very beginning of the morbid process, when there is no complication with streptococci or septicæmia, and when the temperature of the body does not exceed 38° C. (100.4° F.). It is readily seen that under these circumstances there will be but few cases of pulmonary tuberculosis in children in which, because of the difficulty of the diagnosis at that age and in that stage, the remedy could be administered with any show of justification.

Behring,[1] while claiming that Koch's new tuberculin is weaker than the old, announces that he has prepared a stronger one from very virulent dry cultures of tubercle bacilli. He claims to have a *tuberculin* twenty-five thousand times stronger than a dose fatal for one gramme of guinea-pig weight, or eighty times stronger than a dose fatal to a guinea-pig of ten ounces in weight. His mode of preparing his tuberculin is evidently similar to that of Koch, but his statements are made with his old intrepidity.

Immunization, either indirect, by employing the blood-serum of immune animals, or direct, by injecting small quantities of bacteric poison, virulent or attenuated, as the case may be, has less favorable results in tuberculosis than in some other bacteric maladies. Koch's tuberculin was a glycerin extract of pure cultures of tubercle bacilli containing a great many of the latter. Its remedial effect was, when the first universal enthusiasm had cooled down, either soon found to be *nil*, or, on the contrary, many chronic cases were rendered by it acute and speedily fatal. But it has retained a great power for good as a preventive, through its application for diagnosis. When injected into cattle, those affected with tuberculosis react speedily by some elevation of the temperature. If all the cattle of the land were subjected to that test, and those responding affirmatively were killed, the country would be cleared almost entirely of the tuberculous animals which supply milk and meat. It is true that the percentage of cases of tuberculosis contracted from either milk or meat, according to what has been stated before, is small; but, though small, it is too large if it is avoidable.

Medicinal treatment (vol. ii. p. 684) of pulmonary tuberculosis takes no low rank.

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[1] Fifteenth International Medical Congress, Berlin, Session of June 10, 1897.

Creosote was introduced into practice, both for inhalation and for internal administration, in 1877. No direct influence on bacilli should be looked for from it. What it can do is to better the condition of the patient. It will often improve appetite, combat putrefaction, thereby facilitate assimilation, and (sometimes) relieve diarrhœa. The doses vary. Almost incredible doses have been given,—from ten to fifteen cubic centimetres = from two to four drachms daily, and more, to adults. Probably from one to ten drops daily is a dose for children which, according to their ages, may be administered for a long time. The carbonate of creosote, almost tasteless, and easily borne, is a proper substitute in similar doses. Neither ought to be persisted in when the appetite does not improve within a reasonable time, or during a pulmonary hemorrhage, or when the urine, which requires frequent examination, contains, or is beginning to contain, albumin.

These last seven years I have replaced creosote by *guaiacol*, which forms nearly sixty per cent. of the very best creosote in the market, recommended by Schüller, Sahli, and others. A child will readily take from six to fifteen drops daily (according to age) in from three to four doses. It is best taken after meals, in sugar-water, in milk, or in cod-liver oil. There are but few who object to it. Those who do may take one of its salts,—the benzoate (benzozol), salicylate, cinnamylate, or carbonate. Of these I have employed mostly the last, occasionally the first. They are (almost) tasteless, and are readily taken in doses of as many (or more) grains as the fluid guaiacol contains drops. With guaiacol I have been less disappointed than with any other internal remedy administered in pulmonary tuberculosis, cod-liver oil not excepted. It is a good stomachic, appetite and digestion improve under its use, the cough gradually becomes looser, less purulent, and the râles more mucous, and the body-weight is apt to increase. While creosote is not well tolerated in the stage of cavities and hectic fever, guaiacol is not only borne, but appears to exert its beneficial influence even in that condition. There are few patients who do not derive some benefit from its internal use. Externally, it has been recommended to subdue hectic fevers. For that purpose the chest and abdomen are painted with the pure guaiacol several times daily. It has the advantage over creosote of not being contra-indicated either in hemorrhage or in renal complication.[1]

I do not fear that it will be replaced by *ichthyol* (sulpho-ichthyolate of ammonium), which has been eulogized by Cohn, Scarpa, Le Tanneur, H. Fraenkel, and others. Adults (children in proportion) are expected to take from one-fourth of a grain to four grains in a capsule before every meal, or from twenty to forty drops four times a day of a solution in equal parts of distilled water. In spite of the admixture of aromatic oil, it has a bad taste, and will be administered with difficulty.

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[1] See my paper in the International Medical Magazine, November, 1892, and Transactions of the Climatic Association, 1892.

The favorable influence of *cod-liver oil* in the chronic pulmonary tuberculosis of children is an established fact. Its effect is probably not due to its minute percentage of potassium, sulphur, iodine, bromine, phosphorus, and iron. Perhaps the large number of peculiar organic bases (aselline, morrhuine, etc.) contained in it, particularly in the dark varieties, has more to do with its effects on metamorphosis and nutrition. At all events, the free fat acids, of which there is one-half of one per cent. in the light, five per cent. in some dark specimens, appear to control digestion. Its wholesome effect cannot be due to its fat, for cream or some other fat, in daily doses of from two to eight teaspoonfuls, could not replace cod-liver oil. It is quite possible that we have not yet found out the exact nature of the remedy. Perhaps its action is due to some glandular substance which works similarly to the thyroid extracts.

Arsenic in small doses (vol. ii. p. 682) still justifies in my experience its reputation as a cell-growth stimulant and general nutritive when administered either by itself or with digitalis. Fowler's solution should be given, well diluted, after meals, in three daily doses, for weeks and months, or until (which is rare) gastric or intestinal irritation or local palpebral œdema makes its appearance. The daily dose depends on the age of the child, and should vary between one and five drops. If Fowler's solution be not well tolerated, its equivalent in arsenous acid will act equally; indeed, the latter is often tolerated for a much longer time than the former.

The preparations of *digitalis* are the same that were formerly recommended. What we now know of digitoxin and digitalinum verum, which have similar effects, does not commend them for general practice. A good tincture of the English leaves, the fluid extract, and the solid extract are best fitted for children's use. When complaints are heard of their inefficiency, the fault lies generally with the insufficiency of the dose. As soon as digitalis begins to cause arrhythmia of the pulse it is advisable to reduce the dose and combine it with strophanthus, or adonis, or caffeine, for its most favorable effect is obtained before the pulse becomes irregular. In the first stages of its effect it lowers the pulse and increases the blood-pressure, mainly of the left ventricle; and thus, while it stimulates the whole circulation, it relieves the lungs, they being supplied by the right ventricle, which is not so stimulated by the drug.

Inhalations of different kinds were discussed in vol. ii. p. 684. In addition (and partly in repetition) it ought to be remembered that they are (or were) intended to destroy bacilli, to act on the products of inflammation or of disintegration, or to influence cough or expectoration. No bacillus can be reached except, if at all, in the tissue which harbors it, and inhalations take effect on the surface only. Neither medicines nor *hot air* have any other effect. Oxygen, nitrogen, sulphide of hydrogen, hydrofluoric acid, iodoform, carbolic acid, creosote, have been employed in vain. *Turpentine* inhalations are frequently beneficial by loosening, in some cases diminishing, expectoration from suppurating surfaces, and, mixed or not with eucalyptol or other disinfectants, by relieving the fetor of pulmonary gangrene. The inhalations of *compressed air* will prove advantageous in chronic processes where the object is to expand the contracted lung-tissue.

Operative procedures are less indicated in pulmonary tuberculosis of children than in that of advanced age. The opening of a superficial large and copiously secreting abscess is a rare indication, for the latter seldom occurs except in the semi-adolescent, and, if it does, the prognosis is at any rate absolutely fatal. Besides, the dissemination of the tubercular process is so general in the lungs of the young that not more than a slight temporary improvement can be expected of an operation.

The symptomatic treatment of *cough* is one of the urgent indications of pulmonary consumption. When it is mild it requires no attention. Severe spells of coughing, however, may injure the pulmonary tissue by the rapid alternation of expiratory pressure and spasmodic inspiration; indeed, they may lead to emphysema of the hitherto healthy parts. They may force muco-pus with bacilli into alveoli not yet affected, and thus spread the morbid process. Docile children should be taught to suppress cough, no matter from what source, for cough begets cough. When the irritation is pharyngeal, frequent drinking of water, or of an alkaline water, or of milk, or the sucking of a liquorice lozenge, or of a part of the officinal trochiscus of ipecac and morphine (one-fortieth grain in each), from time to time, is indicated. The latter, or a part of it, will render good service in this also, that it may prevent vomiting when taken from five to fifteen minutes before meals. A drop of Magendie's solution on the tongue, not diluted, will have the same effect. Sprays with alkalies or turpentine, the inhalation of steam, whether pure or medicated with aromatics or disinfectants, and, finally, the long list of expectorants, the indications of which every one is familiar with, may or ought to be used according to indications, and a dose of Dover's powder, or codeine or some other opiate, administered at bedtime if required. Sulphonal is credited with diminishing exuberant expectoration, while acting as an hypnotic. Docile children should be taught how to expectorate. When the acid secretion of the stomach which destroys bacilli is deficient, the appetite poor, and the mucous membrane of the intestine catarrhal or ulcerated, the ingested bacilli are capable of lighting up intestinal, mesenteric, or peritoneal tuberculosis.

In the management of the *fever* which attends pulmonary tuberculosis (vol. ii. p. 684) we should remember that it is the effect of various agents. The invasion of bacilli into new territories, the proliferation of the microbes of suppuration and putrefaction, and the progress of inflammatory changes are equally concerned. The question whether it is proper to interfere with every rise of temperature is a very nice one. It is true that it increases the disintegration of albuminoids, and ought not to go on for an indefinite time, but many patients feel better with a moderate rise than with a normal (or perhaps subnormal) temperature. High hectic fevers are better influenced by a combination of quinine with a coal-tar preparation than with either of the two. The latter has a quieting and soporific effect not possessed by the former; still, in every individual case the indications may vary. Whenever acetanilid, or phenacetine, or antipyrin acts unfavorably on the heart, it ought to be combined with caffeine, or strychnine, or camphor in appropriate doses. When they cause an undue amount of perspiration, the remedies recommended (vol. ii. p. 687) for night-sweats will be aptly combined with them. Atropine, agaric acid, camphoric acid (0.25–0.5 gramme, four to eight daily), may then be given in fractional doses, while full doses would be administered for night-sweats at bedtime.

Former remarks on pulmonary *hemorrhage* (vol. ii. p. 686 *et seq.*) are still valid. The most powerful remedy is absolute rest, which should continue for days after its cessation. The applications of ice and morphine sufficient to enforce rest, both physical and mental, are indicated. The popular and frequently effective dose of a teaspoonful or more of table-salt in a little water, with or without the addition of vinegar, may cut short an incipient bleeding. Drinking should be reduced to a minimum, to avoid unnecessary blood-pressure. The doses of acetate of lead, in order to be effective, should be "large." From four to twelve grains a day may be given a child of ten years to advantage. It should be remembered that they are demanded a few days only, and no poisonous effect need be feared. Some opiate, the extract of opium or codeia, should be continued, so as to keep up the quieting effect of the first doses of morphine. Coughing, sneezing,—indeed, efforts of any kind,—are dangerous. That is why inhalations advised for the (very questionable) local effect of astringents should be omitted. Tying the extremities to stop bleeding should not be continued long; the ligatures ought to be loosened after twenty or thirty minutes, and should never be so tight as to constrict arteries as well as veins. Our knowledge of the action of ergot in hemorrhages of the lungs (or other organs) has neither increased nor been refuted during the last decade. Some attribute its effects to the diminution of blood-pressure owing to the presence of ergotinic acid, others to the artery-contracting action of the cornutine. At all events, as long as the chemical and physiological researches concerning the drug are not finished, clinical experience ought to be valued most highly. It is favorable, but the subcutaneous effect is marred by the local irritation apt to be produced by the fluid extract. Sclerotinic acid, besides being painful, is liable to be decomposed by microbes, which are rarely absent from ergot preparations, and will hardly fulfil the expectations in regard to it.

The *complication with intestinal tuberculosis* is not frequent; less so in children than in adults, perhaps because the former do not suffer as long as the latter. It is infrequent (in comparison with the large number of cases of pulmonary tuberculosis) because of the facility with which bacilli are either destroyed in the acid secretion of the stomach or swept through the whole length of the intestinal tract. That is why the presence of tubercle bacilli in the fæces is not a conclusive evidence of intestinal tuberculosis. Nor is it permissible to claim diarrhœa as the conclusive proof of tuberculous enteritis. A simple catarrh, or the presence of hardened fæces which require castor oil or enemata, may be expected during the course of tuberculosis as under ordinary circumstances. When the suspicion of tubercular colitis is justified, large enemata (the hips being raised), containing from one to five per cent. of subnitrate of bismuth, and possibly some disinfectant like thymol (1 : 2000 of warm water or of starch decoction) are indicated. They should be given daily, or several times a day. Internally, the remedies recommended formerly (vol. ii. p. 686) will act well. What I said of bismuth, opium, lead, naphthalin, and resorcin still holds good. Corrosive sublimate I do not value any more highly at present than formerly. Salol may be added to bismuth, from eight to thirty grains (0.5–2.0) daily. Warm fomentations (water or poultices) over the abdomen have a gratifying effect when there is colic or persistent sensitiveness pointing to the presence of a tubercular peritonitis.

FIBROID DISEASE OF THE LUNG.

BY FREDERICK A. PACKARD, M.D.

THE terms "fibroid disease of the lung," "fibroid phthisis," "pulmonary cirrhosis," "chronic interstitial pneumonia," and "fibroid degeneration of the lung" are all extremely unsatisfactory, and it is doubtful if any one title can be found that will entirely satisfy the conditions necessary in defining a disease of the lung having such a wide variety of causations and such different clinical manifestations as are presented by the condition of excessive fibroid change in the pulmonary structures. The title at the head of this article is chosen because it has the widest application and involves no etiological restriction, so that by prefixing an adjective the variety, etiologically speaking, can be designated.

An exact definition is out of the question, but the statement can be made that fibroid disease of the lung is a condition either of development of fibrous tissue in excess of that required for the inclusion of irritating material in the pulmonary tissue, or of riotous excess of formation of fibrous tissue in the chronic inflammatory diseases of the ultimate air-passages. At what point the conservative and curative fibrous tissue formation around an inflammatory focus is exceeded it is impossible definitely to determine. Nevertheless, some imaginary line must be drawn. Conservative fibrous tissue formation may be likened to a scar, while the growth of pulmonary connective tissue in excess of that necessary for repair resembles keloid in its exuberance.

Fibroid overgrowth is undoubtedly most frequently initiated by the presence of the tubercle bacillus, but any irritating organic or inorganic material acting for a sufficient length of time, and with an activity insufficient to cause destructive inflammatory or degenerative changes, is capable of producing the process.

One cause of proliferative connective tissue change in the lungs of adults is practically absent in children; that is, the inhalation of inorganic materials in large quantity, such as occurs in siderosis and anthracosis in the adult. A frequent precursor of the condition—in fact, the most frequent—is the catarrhal pneumonia that often complicates measles and pertussis. Of course this pneumonia often becomes the seat of tuberculous infection, which, in turn, may lead to fibroid change; yet in children tuberculous infection of a lung in a condition of inflammation in the course of or after the two diseases mentioned is usually too active to permit of much connective tissue increase before the death of the patient. Certain it is that in a large majority of cases of apparently non-tuberculous pulmonary fibrosis in children the immediate cause has been a catarrhal pneumonia complicating or following measles or pertussis. It would seem as though these two diseases in some way almost predisposed to connective tissue overgrowth in the lung. Why this should be the case is not certainly known, but the following explanation may be satisfactory. In both of these diseases there is marked involvement of the tracheo-bronchial glands, which may remain for a time so altered as to interfere with the proper flow of lymph from the lungs. If such be the case, the excessive connective tissue formation may with propriety be ascribed to this factor common to the above-mentioned diseases, and more frequent in them than in any other general diseases or conditions.

While the great majority of cases of pulmonary fibrosis may be traced to tuberculous infection and to catarrhal pneumonia following measles or pertussis, a small percentage may follow the peculiar form of fibroid or proliferative lobar pneumonia described by Wagner, Kohn, Aldinger, Auld, Herbig, Delafield, and others. This probably plays a *rôle* even less important in children than in adults.

Malarial cirrhosis of the lung has been described, but the etiological *rôle* of malaria is very doubtful.

Excessive fibrous tissue overgrowth in the lung is a frequent result of chronic bronchitis, especially in cases where the chronic process follows frequent attacks of subacute or acute bronchial inflammation. The fibroid invasion is particularly marked in the cases where bronchial dilatation has occurred. The mutual relationship of fibroid disease of the lung and bronchiectasis will be mentioned at greater length below.

Finally, in a certain proportion of cases, the connective tissue growth is inaugurated in the subendothelial layer of the pleuræ, following either repeated slight attacks of pleural inflammation or one severe attack of pleurisy with serous or purulent exudate. The statement that tuberculosis is always at the bottom of such pleurisies would seem to be too sweeping, inasmuch as the post-mortem evidence of tuberculous infection may be entirely wanting.

It is impossible to state definitely the reason for the occurrence of this process in some cases and its absence in others. The view that a "fibroid diathesis" is present in some and not in others is not satisfactory, for while in a few cases simultaneous fibroid change is found in other organs (such as the liver and kidneys), these are rarely of such a nature that they cannot be attributed to the secondary effects of the pulmonary disease through circulatory and cardiac disturbance. There is no evidence that the negro, who has such capacity for fibrous tissue overgrowth in other regions, is proportionately more often the subject of fibrous overgrowth in the lung than is the Caucasian, although this may possibly be accounted for by the rapid course of tuberculous disease in the negro, which carries him off before the fibrous tissue has had an opportunity to form.

Sir Andrew Clark, to whose writings much of our knowledge of fibroid disease of the lungs is due, makes the following divisions: 1. Pure fibroid phthisis, by which is understood a condition wholly independent of tuberculosis. 2. Tuberculo-fibroid disease, a condition primarily tuberculous, but in which the tuberculous element is much less marked than is the fibrous. 3. Fibro-tuberculous disease, in which the condition was primarily one of simple fibrosis, the infection with tuberculosis being a secondary affair.

Before the revolutionizing discovery of the causative agency of the tubercle bacillus by Koch, the clinical and even the pathological separation of the various forms was impossible, so that a study of the cases published prior to Koch's discovery is of comparatively little value. Even now the determination of the primary condition in the last two divisions of Sir Andrew Clark's classification is impossible unless the case has been under observation from the outset.

The earlier symptoms and signs of fibroid disease of the lung vary with the primary cause of the excessive growth of connective tissue. In the cases following pneumonia, whether this be of tuberculous or non-tuberculous causation, the general symptoms continue to be present in a modified degree for a varying length of time; slight fever persists, the cough does not cease, expectoration lessens or may entirely disappear, some shortness of breath on exertion remains, and the flesh is but slowly regained, the previous weight relative to stature being seldom attained. The cough changes its character somewhat, being harsher, more harassing, and even paroxysmal. A fair degree of general health may be obtained, particularly if a change of climate has been tried, but the patient seldom feels perfectly well or appears as robust as before the inaugurating illness. If the process of fibroid overgrowth be the result of tuberculous infection, the regaining of even a fair degree of health will be a slow process, and it is probable that recovery is never so nearly complete in this as in the non-tuberculous cases.

The morbid physical signs primarily present during the stage of active pulmonary infection never entirely disappear, but some impairment of resonance, whiffing, broncho-vesicular, or bronchial breath-sounds continue to replace the normal vesicular murmur; some degree of increase of vocal resonance and tactile fremitus continues, until, finally, the physical signs of partial or complete fibroid change of the diseased part are found to have developed.

In the fibroid change of bronchial origin the lesion is usually so gradually established that the patient is seldom seen during the forming stage. Ordinarily, after frequently recurring attacks of bronchitis, the patient is found, upon examination during an acute attack, possibly preceded by a long interval of freedom, to present the signs of fibroid disease that were not present at the former examination. The intervening steps of the process are, therefore, seldom seen by the physician. On inquiry, it will usually be found that after the preceding acute attack the cough never entirely ceased, but had continued as an annoying, rather paroxysmal, relic of the severer acute cough with free expectoration.

In the pleurogenous form also the intervening steps from the acute pleural inflammation to fibroid infiltration of the pulmonary parenchyma are seldom witnessed, examination during an acute exacerbation or some independent illness usually revealing the condition.

After the development of fibroid overgrowth of whatever origin the cases run a fairly uniform course, varying but little in the different forms because of the fact that usually by the very overgrowth of fibrous tissue the original lesion is rendered quiescent in the case of mechanical and bacterial irritants, and in all forms this fibrous proliferation and its results overshadow other and primary conditions.

The general physical condition may be apparently but little impaired, and the superficial fat may be fairly well retained, while muscular strength may suffer but little. The patient can seldom endure prolonged or vigorous exertion, but this is usually because of limited respiratory capacity rather than lack of muscular power. The most troublesome symptoms are paroxysmal cough and shortness of breath on exertion. Frequently these are the only symptoms of which complaint is made. The cough is often extremely annoying, and in cases with bronchial dilatation is usually worse at definite intervals, on change of posture (as from one side to the other), and is often relieved only by the evacuation of a large quantity of sputum. Efforts to bring up secretion frequently induce vomiting. The excitation of cough by change of posture should always suggest the presence of bronchial dilatation, from the fact that the mucous membrane at the seat of dilatation seems to become insensitive to the irritation of large amounts of fetid secretion, and cough is excited only when a change of posture brings this material in contact with a portion of more normal mucous membrane. The expectoration may be extremely scanty and be composed of a small quantity of glairy, transparent mucus, or may be excessive in quantity and of fetid odor in cases with bronchiectasis. In the latter class the sputum separates into three layers on standing, and contains mucus filaments, pus-cells, fatty acid crystals, and sometimes elastic tissue without alveolar arrangement. In the cases of tuberculous origin tubercle bacilli may be found. The sputum is frequently tinged with blood, and in a few of the reported cases fatal hæmoptysis has occurred.

The aspect of the patient shows nothing definite, but in a certain number of cases the thick lips and heavy features usually associated with cardiac disease are seen.

The conformation of the chest in well-marked cases is quite characteristic. The shoulder of the diseased side is lower than its fellow, the scapula stands out prominently, the spinal column is curved in the dorsal region towards the diseased side, with a compensatory curvature in the opposite direction in the lower dorsal and lumbar regions. The diseased half of the thorax shows general or partial diminution in size, with diminished respiratory excursion and retraction of the intercostal spaces.

Groups of dilated venous radicles may be seen beneath the skin, but these seem to be produced in children less readily than in adults. The apex-beat of the heart is usually more plainly visible than normal when the left lung is the seat of disease, owing to the uncovering of the heart by its retraction, whereas if the right lung be affected the compensatory emphysema of the left lung renders the impulse obscure. Cardiac displacement is present in a degree commensurate with and to the side of the disease.

The signs elicited by palpation vary much in accordance with the physical condition of the underlying lung. The lack of respiratory movement is often appreciated better by the hand than by the eye. Tactile fremitus is diminished in well-marked examples, and, where the pleura is much thickened, cannot be perceived.

The percussion-note varies from impairment of resonance with slight increase of resistance in the slight cases up to a wooden, dull note with great increase of resistance in the cases of advanced lesion with great pleural thickening. Dull tympany in a localized area may be at times elicited in evidence of a bronchiectatic, gangrenous, or tuberculous cavity.

The breath-sounds are feeble and distant, with broncho-vesicular character in the greater number of cases; but in cases of marked bronchiectasis cavernous breathing may be heard, or pleural thickening may be of such extent as absolutely to prevent the transmission of any murmur. Râles are frequently entirely absent, save during catarrhal exacerbations and overdilated bronchi. In the latter case they are frequently large, bubbling, and metallic. "Cirrhotic crackling," described by Juergensen as possessing characters midway between those of pleural friction and the crepitant râle, may be heard, while Sir Andrew Clark draws attention to a peculiar "sucking sound" heard in some cases.

On the healthy side of the chest and over uninvolved portions of the diseased lung the signs of compensatory emphysema are present.

On the side of the heart, often the only sign, except for displacement, is accentuation of the second pulmonary sound. In some cases of shrinkage of the left lung there are to be felt in the second left interspace a systolic impulse and a diastolic shock owing to uncovering and possibly distortion of the pulmonary artery. In long-standing cases enlargement of the right side of the heart can be determined by percussion.

Some degree of hepatic enlargement is present in many cases in the later stages.

Œdema exists in a fair proportion of cases, most marked in the legs and feet. The occurrence of ascites is affirmed by some, denied by others. It is by no means a frequent form of dropsy, and doubtless is dependent upon the secondary changes in the liver and peritoneum.

Clubbing of the fingers is very frequent, it being noted in over seventy per cent. of the cases collected by Clark, Hadley, and Chapin.

The amount of fever present varies from entire absence to quite marked intermittent temperature. In the latter case the rise of temperature is due to bronchial ulceration or the accumulation of septic materials in tuberculous or gangrenous cavities.

The pulse is usually of fair force, and it is only in the late stages of the disease, where the right heart is beginning to suffer from prolonged strain and to fail in furnishing a proper quota of blood to the left side of the heart, or when general asthenia is present, that the pulse shows abnormality.

The mode of termination is very varied. In a certain proportion of cases, and that by no means small, the disease does not materially shorten life, and the patient may be said to have recovered although the fibroid induration is still present. The great danger in all cases is the occurrence of tuberculous infection or the relighting of tuberculous activity in those primarily caused by that infection. In some cases the circulatory disturbance produces sufficient trouble to destroy life by hepatic cirrhosis (of cardiac form), renal cirrhosis and congestion, or chronic proliferative peritonitis. The occasional occurrence of metastatic cerebral abscess in cases of bronchiectasis must be borne in mind.

The pathological changes vary greatly with the cause of the condition. In all cases of marked extent the diaphragm is found to be drawn upward on the diseased side, the mediastinum is more or less deviated from its normal position, and the heart is correspondingly dislocated. The liver, kidneys, spleen, and other viscera may present pathological evidence of venous obstruction.

The heart usually shows enlargement of the right ventricle and auricle from hypertrophy or dilatation or a combination of these two processes.

In all forms, of whatever causation, the lungs show certain characteristics, while in each variety there are minor differences that depend chiefly upon the point of departure of the sclerotic process. The lobe or lung involved is small, shrunken, heavy, and usually darker than normal. It is invariably more or less adherent to the parietes by old pleural adhesions or by a firm, dense layer of sclerotic tissue requiring separation by the knife.

On section it is found that the lung cuts with much resistance, and in marked cases there is decided grating. The cut surface shows a varied picture according to the point of origin of the overgrowth, whether from interlobular connective tissue, from peribronchial tissues, or from the pleura. In all forms, except in cases of very slight degree, the cut bronchi stand out plainly, and many of them form veritable cavities. In any of the forms other cavities may be seen with necrotic walls and containing broken-down tissue,—gangrenous cavities. In the primarily or secondarily tuberculous cases other cavities are present, with cheesy or mucopurulent contents and with grayish walls of ragged granulation tissue.

In the pleural form the shrunken lung or lobe is encapsulated in a thick yellowish-white envelope, and from this strands of connective tissue course inward for a varying distance. In the peribronchial form the connective tissue is most abundant near the root, and coarse bundles of fibroid tissue accompany the larger tubes, from which proceed bands of connective tissue coursing through the interlobular tissue. In the post-pneumonic (tuberculous or non-tuberculous) forms the connective tissue is of more uniform distribution in the portion of lung or lobe affected, and the diminished alveolar spaces show as small pits in the midst of the grayish-white area. In some cases following tuberculous pneumonia a dense white, yellowish-white, or gray scar is seen, with ray-like processes spreading from it on all sides.

In the portions of the lung not affected, and in the lung of the opposite side, emphysema is found showing the ordinary appearances of that condition.

The cause of bronchiectasis in pulmonary fibrosis has been the subject of much dispute. The three most plausible explanations for its occurrence are expiratory strain, negative pressure upon the bronchial walls from without by the diminution in size of the surrounding pulmonary tissue, and mechanical dilatation from contraction of the tissues between the tubes. It is probable that all three factors are at work, yet the last named would seem to have the greatest weight. It has been urged that contraction of the tissues surrounding a bronchus should narrow its calibre, and undoubtedly such would be the case were there only the two materials, a bronchus and surrounding contracting material; but where there are two bronchial tubes with intervening contractile tissue the contraction would tend to draw them together or, in other words, to dilate their calibre. It can, therefore, readily be seen why bronchial dilatation is most marked in cases of bronchial origin, two factors being at work,—a dense formation of connective tissue immediately around the bronchus, and a bronchial wall whose vitality is impaired by repeated inflammatory attacks.

Microscopically there is found great increase of connective tissue cells with infrequent nuclei. In the cases resulting from a primarily fibrous pneumonia the changes produced have been carefully studied, especially in recent years. In these there have been found all stages of the disease, from the primary fibrinous exudate in the alveoli to the development of fibrous connective tissue plugs in the alveoli and infundibula united to the wall of the air-space by a pedicle which is continuous with the increased interstitial connective tissue of the lung. The derivation of the new connective tissue cells is still a matter of dispute. The microscopic changes in the bronchi concern chiefly the external coat, which is thickened by connective tissue growth. The mucous membrane shows the appearances of chronic bronchitis in the cases of bronchial origin, while in the specimens of bronchiectasis the mucosa is thinned, and in places has lost its columnar epithelium.

The prognosis is a question of great difficulty. In all forms tuberculous infection or a relighting of quiescent tuberculosis is to be dreaded. In the pleural form the outlook is by no means unfavorable; in the cases of bronchial origin the prognosis is dependent upon the possibility of avoiding fresh catarrhal attacks; while in the tuberculous variety the outlook is less favorable, although far better than in any other form of tuberculous pulmonary invasion.

Prophylactic treatment can be carried out only by an exhaustive effort to thoroughly restore the tissues after every attack of pulmonary, bronchial, or pleural inflammation, and by seeking to prevent recurrence of repeated attacks of bronchitis and pleurisy. In those subject to bronchial catarrhs or recurring dry pleurisy much can be done by hygienic measures. In cases where fibroid change has already taken place,—*i.e.*, where there is a formation of connective tissue in excess of that required for the repair of damage or for inclusion of irritants,—much can be done to prevent increase of the trouble. The avoidance of anything conducive to intercurrent inflammatory attacks by hygienic means is of the first importance in all forms of the trouble. Change of climate may be necessary to accomplish this. The patient should, if possible, live in an equable dry climate. Elevation of itself is of no advantage, inasmuch as we cannot hope to remove the physical condition present, but may be able to prevent additions to it and to maintain the patient's general condition. Temperature alone has by no means so much to do with choice of climate as has equability of temperature and moisture.

Unfortunately, especially in the case of children, the seeking of a favorable place of residence is often entirely out of the question. Much, however, can be done to avoid the harmful effects of a climate which is not all that could be desired. An abundance of fresh air in all but very inclement weather is desirable. Protection of the skin by varying weights of silk or woollen underwear, in order to prevent chilling of the surface and consequent internal congestion, is extremely important. The danger of the evil effect of unavoidable exposure to varying temperature can be much lessened by the employment of the daily cool bath with friction, a measure that probably more than any other is capable of preventing recurring catarrhal attacks. Even quite young children can be habituated to the cool bath with nothing but favorable result, provided only that the water be of such a temperature that the child can readily react. Where the vitality is much lowered, an "alcohol sponge" is the best substitute.

Pulmonary gymnastics are of great value, not only in preventive treatment, but in obviating the evil effects of the developed condition. The increase in respiratory capacity and chest-measurement in children after a course of mild pulmonary gymnastics is often marked. The practising of forced inspiration and slow prolonged expiration, and the use of the calisthenic wand, of light dumb-bells, or of Indian clubs, are the best means of reaching this result, because in using the apparatus mentioned general as well as pulmonary exercise is attained. In some cases the use of forced expiration into a spirometer is of advantage in order to, so to speak, stretch the healthy portions of lung. A home-made apparatus composed of a gallon jar fitted with corks, in which are two tubes (one reaching from the patient's mouth to the shoulder of the jar, the other running from the bottom of the jar to a second bottle), answers every purpose. In spite of the existence of emphysema and of bronchiectasis, such forced expiration is not contra-indicated, the emphysema being purely compensatory and the bronchial dilatation being chiefly produced by causes external to the lumen of the tubes.

We possess no medicinal agents that can be said to have any specific effect in lessening the existing overgrowth of connective tissue. Iodide of potassium, which has such a well-earned repute as an absorbefacient, is probably entirely incapable of removing already formed dense connective tissue, such as is found in this disease. It is of value in some cases as an addition to expectorant mixtures towards the close of an acute attack of intercurrent bronchitis, and on such an occasion may possibly prevent the formation of new fibrous growth.

Every effort should be made to keep the general condition of the patient at the highest possible point. For this purpose the various tonics will be often needed, and especially cod-liver oil and arsenic. The latter drug is of especial value in this class of cases, improving the general nutrition probably better than any other single drug. It has an advantage over cod-liver oil in that, in proper doses, it tends to aid rather than impair digestion. Intercurrent attacks of pleurisy are best met by the use of salicylate of strontium, in doses suited to the age of the child, followed by a short course of small quantities of iodide of sodium or potassium, and this again by arsenic in tonic doses. It is in these cases especially that arsenic is of great value.

Intercurrent attacks of bronchitis should be cut short as rapidly as possible, due care being taken that in treating the acute disease we do not impair digestion and assimilation by the too liberal use of expectorants. The cotton jacket, and rubbing of the chest with a mixture of equal parts of olive oil and turpentine or oil of amber, may be sufficient treatment, or we may have to use some such expectorant as chloride of ammonium; but the use of nauseating compounds of many drugs with reputed expectorant qualities is to be sedulously avoided. After such an attack it is well to resort again to small quantities of the iodides for a week or ten days.

For the chronic bronchitis that is so frequently a cause of and an attendant upon the condition it is best to employ topical remedies by the use of inhalants or the steam spray. One remedy that is of great value is the compound tincture of benzoin, used on the ordinary Yeo respirator or in the globe nebulizer. With it may be combined one of the volatile oils, especially oil of eucalyptus. By these means alone much improvement may be obtained without risk of derangement of digestion. Iodide of sodium, chloride or carbonate of ammonium, and oil of eucalyptus, in frequent small doses, may be required in addition, but they should be used with care, and only after failure in the use of topical remedies.

For the bronchiectasis, or rather for the accumulation of sputum in the bronchial cavities, we may have to employ remedies to obviate the fetor. This is best done by the use of creosote, iodoform, oil of eucalyptus, and alcohol on the inhaler, which can be worn for long periods at a time by older children, or by saturating a piece of lint and laying it on the pillow beside those too young to be instructed. Of internal remedies, creosote and oil of eucalyptus are of most value, both being excreted by the lungs.

Where paroxysmal cough without a corresponding amount of expectoration is present, a sedative inhalation of iodoform, chloroform, and alcohol may be required. On no account should opium be used unless absolutely necessary, in which case it is best to give its alkaloid codeia with a small quantity of hyoscyamus, in proportion to the age of the child.

Cardiac failure, hepatic or renal congestion, ascites, and other symptoms of secondary involvement of organs aside from the lungs require treatment on general principles, as in the same conditions from other causes.

External remedies applied to the chest are often of great value. The application of tincture of iodine over the affected lung after any acute intercurrent inflammatory attack is of value in almost all cases, not to cause absorption of the old fibrous tissue, but as a counter-irritant against any recent addition to that material. A plain hot-water or turpentine stupe will frequently relieve the paroxysmal cough.

Surgical treatment of the bronchiectatic or tuberculous cavities is at the present time not warranted. This is especially true of children. Whether the technique of thoracic surgery will ever reach a point to permit of such treatment is still a matter of doubt. The only opportunity for surgical interference at present permissible in this disease is the drainage of a metastatic cerebral abscess, which, in the few cases where it does occur, should be treated as is cerebral abscess from other causes.

PLEURISY.

BY HERMAN F. VICKERY, M.D.

Definition.—Pleurisy, or pleuritis, is an inflammation of the pleura, in children more often secondary than primary. It is accompanied by an exudation upon the serous surface of the pleura or into the pleural cavity, which may be either fibrinous, in dry pleurisy, sero-fibrinous, in pleurisy with effusion, or more or less purulent, in suppurative pleurisy or empyema.

Etiology.—Pleurisy is more common in infancy and childhood than was formerly believed. A greater proportion of cases are suppurative than in adults. The greatest number of cases occurs below the age of five years, the next greatest number between five and ten years. It is commonest in the spring and winter, attacks more boys than girls, chooses the poor and feeble rather than the well-to-do and robust, is almost always unilateral, and is somewhat more apt to affect the left side than the right.

Whether any cases are primary is an unsettled point. In a great majority of instances pleurisy in children is secondary. It is seen in the new-born as the result of septicæmia, due to infection from the navel or to the poison of puerperal fever absorbed before birth. Traumatism, even without a lesion of the surface, may apparently provoke an attack.

Of many of the causes enumerated, other than pulmonary, it is probable that they act not directly upon the pleura, but mediately through the lungs, setting up a pneumonia which, in its turn, occasions the pleurisy.

A bilateral pleurisy is rare, as already stated. If not due to bronchopneumonia, it suggests sepsis or tuberculosis.

The most important advances in our knowledge of pleurisy of late years have been due to bacteriological research. It is desirable that every pleuritic effusion should be examined for micro-organisms, as aiding both the diagnosis and the prognosis of the case. So far as the present writer is aware, Koplik is the only author whose bacteriological investigations of pleural effusions have been made in children. He states, however, that, with certain exceptions, his results "bring the pleurisies of children much into line with those of the adult as to causation."

For the organisms found in purulent effusions, the reader is referred to the article upon Empyema. In serous effusions there may be found:

(1) No germs whatever; (2) tubercle bacilli; (3) pneumococci; (4)

staphylococci; (5) streptococci; (6) pneumococci together with streptococci or staphylococci.

The apparently sterile effusions sometimes occasion tuberculosis if injected into animals, and an important proportion of the cases with germ-free effusions are believed to be tuberculous. In this connection it may be mentioned that Netter reports that thirteen out of fifteen cases of pleurisy reacted to tuberculin, and that Dr. Franklin White, of Boston, has injected tuberculin in eight cases of serous pleurisy with a positive result in every case. Tubercle bacilli, when found at all in the effusion, are scanty. Three different observers have reported single cases of a serous effusion containing the typhoid bacillus.

The inflamed pleura speedily loses the normal lustre of its serous surface. Its tissues present dilated blood-vessels and lymph-channels, with more or less escape of leucocytes into the parenchyma, but, except in case of infiltrating tuberculosis or malignant growth, the important products of the inflammation are found either within the pleural cavity or upon its lining membrane. These products are fibrin, serum, pus, and blood. Blood is rarely macroscopically evident, except in scurvy, purpura hæmorrhagica, or tubercular or cancerous new growths. Fibrin is the chief product in dry pleurisy (pleuritis sicca). It may be present on either the pulmonary or the costal surface, or more frequently on both, and it often extends in bands from one surface to the other. These bands rapidly develop new blood-vessels and become organized, binding the lung to the ribs and thus more or less obliterating the pleural cavity.

Dry pleurisy may be an insidious process. On the other hand, it is the chief cause of pain in lobar or lobular pneumonia or pulmonary tuberculosis, in which diseases it appears in localized areas corresponding to the position of the pulmonary lesions.

Pleurisy with effusion begins in much the same way as dry pleurisy, but in a few hours or a few days a considerable quantity of fluid exudes into the pleural cavity. Fluid is often demonstrable in twenty-four hours, and the thoracic cavity may be full at the end of two or three days. It is exceptional for a serous exudate to become purulent.

Fatal syncope may be the penalty of oversight or unwarranted delay when the effusion is very large and the heart displaced.

As fluid accumulates in the pleural cavity the corresponding lung, by virtue of its own elasticity, retracts upward and inward. If the amount of fluid becomes large enough to exert actual pressure upon the lung, that organ may become a small, almost solid, and almost bloodless mass in front of the scapula. In case the fluid is removed by nature or art within two or three weeks, the lung usually re-expands to its normal dimensions, but if the fluid remains too long, the lung may be permanently tied down by adhesions, and the patient will practically have but one lung left to breathe with.

Exceptionally, it is said that after a pleurisy has run its course the fibrinous new formations extend inward and invade the interlobular spaces of the lung itself, thus more or less crippling it. The present writer has never met an instance of this kind.

Symptoms.—The classical symptoms are fully detailed on page 691, vol. ii. Where the careful consideration of all the symptoms and signs still leaves the physician in doubt whether there is an effusion or not, and where the condition of the patient would be benefited by the withdrawal of fluid, if present, recourse should be had to an exploratory puncture. For this many recommend a hypodermic syringe. To the writer it has seemed wiser to use an instrument of larger calibre, because thick fluid might not flow through a fine needle, and the failure of a puncture made with it might consequently lead one into error. Another consideration is, that if fluid be found by puncture, whether serous or purulent, it will be desirable to proceed at once to its removal, which could not be done by means of a hypodermic syringe.

Râles may rarely be heard over fluid; and the writer, as well as other observers, has known fluid to be withdrawn through a needle inserted where moist râles were audible. A similar statement has been made in regard to a friction-rub.

Further details as to aspiration will be given under Treatment.

Pleuritic effusions may be sacculated. This is more frequent in purulent than in serous pleurisy. Such collections are more apt to occur in the back. The signs are as already described (vol. ii.), only localized.

Diagnosis.—At first the complaint of abdominal pain, combined, perhaps, with vomiting or diarrhœa, may lead one to suppose that the trouble is a digestive one, until a physical examination discloses the true state of affairs.

Sometimes, but less often than in pneumonia, the cerebral symptoms may so predominate that meningitis is strongly suspected. Henoch relates that early in his own career he treated for two weeks a boy afflicted with pleurisy as a case of meningitis. The rule he then formed would be good for all to follow,—in no febrile disease omit the examination of the chest, even if there be no thoracic symptoms. Such a course would also protect the physician from the mistake of regarding pleurisy as malaria or typhoid.

The diagnosis from pneumonia has been considered in vol. ii. It must be remembered that undoubted pneumonia may be complicated or followed by pleurisy with effusion.

Consolidation due to tuberculosis differs from pleurisy with effusion in its physical signs much as does pneumonia. The condition of the other lung may aid in diagnosis, and also the history of the case. The dulness, harsh respiration, moist râles, and dulness or muffled tympany under the clavicle, when there is a large effusion, have sometimes suggested tuberculous infiltration, or even a cavity; but, in any case, the clearing up of these signs upon removal of the fluid will dispel our doubts.

Hydrothorax is afebrile, usually bilateral, and seldom occurs except in cardiac or renal disease.

Prognosis.—Dry pleurisy is rare in younger children, except in connection with lobar or lobular pneumonia or tuberculosis. It may increase the suffering, but not materially the dangers, of these diseases. Occurring by itself, it usually subsides in a week or two, and always terminates favorably.

The prognosis of serous and sero-fibrinous effusions is very favorable. Antisepsis and asepsis have greatly improved the prognosis of operative cases. In rare instances the volume of fluid may so embarrass the heart as to cause sudden death; but usually cases begin to improve in the second or third week, the effusion is absorbed, and the lung resumes its normal place and functions. Complete recovery may occupy several months. If a large effusion has been allowed to remain for many weeks, the lung may, as already stated, be permanently crippled, being too firmly bound down ever to re-expand.

With regard to bacteriological examination of the fluid, if aspirated, something has already been said under Etiology. Most serous effusions are sterile. That a certain proportion of these owe their origin to tuberculosis is certain, but it is going too far to say that they all do so. Occasionally tubercle bacilli are found, or the fluid inoculated into animals occasions tuberculosis. In such instances the ultimate prognosis is that of incipient pulmonary tuberculosis. The immediate prognosis with regard to the pleurisy is good.

Of other germs found in serous effusions, the pneumococcus is the most frequent and most favorable; next comes the staphylococcus; and least favorable and least frequent is the streptococcus. With these three, in the order named, there is increasing liability to pus-formation; but yet, under aseptic precautions, it is rare to have a serous effusion transformed into an empyema. In most instances empyema, if it occurs at all, begins as empyema.

Treatment.—There has been no material change in the method of treating this disease since the original article was written. The treatment is fully considered in vol. ii. p. 699.

Some authors have praised salicylic acid and salicylate of sodium as useful both to abort the pleuritic process and to absorb serous effusions. In the limited experience of the writer with these they have proved disappointing. Moreover, their effect upon the stomach is not desirable.

The general trend of professional opinion seems to be in favor of earlier aspiration than was formerly practised. The presence of a considerable amount of fluid and the subsidence of active febrile disturbance are sufficient to justify the operation, even at an earlier period than named above. It is not, however, desirable to empty the chest too early, unless dyspnœa is becoming extreme, for the inflamed pleural surfaces may be irritated by rubbing against each other, and where very early aspiration is necessary, it is wise to remove a part rather than the whole of the fluid.

With regard to aspiration, a few practical hints may not be out of place.

A trocar and canula are better than a sharp-pointed needle, because the needle may scratch the lung as it expands. The calibre of the trocar should be large enough to allow pus to flow. An aspirating apparatus is preferable to a syringe, because with it we can not only confirm our diagnosis, but evacuate the fluid if present. It should be made an invariable rule to test the aspirating power of the apparatus before inserting the trocar, lest some unsuspected occlusion render the whole effort valueless or misleading. If the pump connected with the aspirator is, unfortunately, so contrived that it can force air into the chest as well as aspirate fluid out of the chest, the operator must be sure that his assistant can be trusted. It is better to have an instrument that can do nothing but aspirate.

The place for the insertion of the trocar is often chosen too low. Good localities are the seventh space, just outside the lower angle of the scapula, and the sixth space in the axilla, in front of the latissimus dorsi. Antiseptic precautions must be thorough. The instrument should be kept close to the upper edge of a rib, and after the operator feels that he has evaded the bone, the final thrust should be rather quick, so as to pierce any false membranes which may exist, rather than merely to push them forward. Great care should be exercised lest the trocar be driven in too far. This is a vital matter. As a rule, a penetration of one inch (2.54 centimetres) in a young child is sufficient. Perhaps the maximum may be set at one and a half inches (4 centimetres).

If fluid is found, it should be withdrawn gradually, and the aspiration should cease as soon as the child has much cough or seems distressed. The removal of a very small amount often suffices to bring about the rapid absorption of the rest. It is preferable to have the patient in a sitting posture during the operation, so that in case of any cardiac weakness we may have the advantage of a change to the horizontal position.

In rare instances serous effusions obstinately reaccumulate. West reports one which was tapped thirty-seven times. These demand thoracotomy and drainage, as described under Empyema.

EMPYEMA.

BY A. T. CABOT, M.D., AND F. G. BALCH, M.D.

THE most important recent advances in our knowledge about empyema have been in the matter of etiology. The additional light shed on the subject from this source aids us greatly in forming a correct prognosis, and somewhat in directing our treatment. Points in diagnosis and treatment to which little space is given in the present article will be found more fully elucidated in the article in vol. ii. p. 709 of this Cyclopædia.

Definition.—Empyema, or purulent pleurisy, is the name given to a collection of pus or sero-pus in the pleural cavity. Such a pleural effusion is especially common in children, Mackey finding forty per cent. of all effusions in the pleural cavity in children to be purulent, as against five per cent. in adults.

Etiology.—As we extend our knowledge of the subject it becomes more evident that every case of empyema is due to bacterial infection. Occasionally it is impossible to prove this, but it is probable that the occasional failure is due to imperfect methods of investigation.

The micro-organisms usually concerned in the process are the pneumococcus, streptococcus, staphylococcus, tubercle bacillus, and, rarely, the typhoid bacillus. The avenues of infection are many. Netter groups the conditions which may lead to empyema as follows:

1. Broncho-pneumonia, especially the form seen in influenza, pneumonia, tuberculosis, bronchiectasis, gangrene, cancer, and pyæmic abscesses.

2. *Diseases of the Mediastinum.*—Pericarditis, cancer or other diseases of the œsophagus, and abscess.

3. *Abdominal Diseases.*—Purulent peritonitis and subphrenic suppuration.

4. *Infectious Diseases.*—Scarlet fever, diphtheria, erysipelas, puerperal fever, and typhoid.

Infection from aspiration, bullet wounds, stab wounds, and the like, must also be considered.

The bacteriology of empyema in children and adults is very different, and on this account the prognosis and treatment also vary. The only figures at present published on the bacteriology of empyema in children are those of Netter and Koplik. Netter gives the following table, showing the relative frequency of the different micro-organisms in adults and in children:

CHILDREN.	PER CENT.
Pneumococcus	53.6
Pneumococcus and streptococcus	3.6
Putrid	10.7
Tubercular	14.3
Streptococcus	17.6

ADULTS.	PER CENT.
Pneumococcus	17.3
Pneumococcus and streptococcus	2.5
Staphylococcus	1.2
Tubercular and putrid	25.0
Streptococcus	53.0

Koplik in a smaller number of cases found a pure pneumococcus infection in sixty per cent. Holt publishes nineteen cases with pneumococcus alone in fourteen, streptococcus alone in three, pneumococcus and streptococcus in one, and staphylococcus alone in one.

The metapneumonic empyema—that is, the form following or complicating pneumonia—is far the most common in children. At times we find numerous pneumococci in a serous effusion which never becomes purulent, while in others the effusion will be purulent from the start, and there may be little or no trouble found in the lung itself.

In many cases where the micro-organism is the streptococcus or staphylococcus either alone or mixed, the infection is through the lung, but in the majority of this class of cases the point of entry can be found in some other part of the body. Thus, a streptococcus infection of the throat or a slight septic wound may give rise to a streptococcus empyema, and it is necessary to consider carefully these other sources before deciding upon the lung as the point of entrance.

The tubercular form of empyema is much less frequent in children than in adults. Netter places it at fourteen per cent. of the cases, but other statistics show that that is probably a high estimate. In the adult, on the other hand, the number of tubercular pleurisies is variously estimated at from 43⅓ to 68.5 per cent. The tubercle bacillus is not easy to find in the fluid, and it is often only by inoculation that we can prove the nature of the trouble. The bacillus is at times found associated with the streptococcus or staphylococcus. A sterile sero-fibrinous fluid strongly suggests the probability that we have to deal with a case of tubercular empyema.

A pneumococcus or streptococcus empyema may become fetid or putrid in case of gangrene of the lung or if communication is opened with the air-passages. After operation a tubercular empyema may become fetid. In these cases the fetor is due to a mixed infection, but the exact nature of the organism is not yet determined.

While the direct causative agent of empyema is probably always a micro-organism, there are predisposing causes, such as exposure to cold, or external violence, which prepare the tissues for bacterial invasion.

Pathology.—Except in the cancerous or tubercular form, the pathological process confines itself to the surface of the pleura. The membrane itself is often but little changed. In the metapneumonic cases there is often a large amount of fibrin present. The lung is rarely injured by the compression in acute cases, though where the fluid has been present for a long time it is frequently reduced to a small mass with hardly a trace of alveolar structure. In the tubercular form there is often great thickening of the pleura, especially of the parietal pleura, where the lesions are usually most advanced. The whole pleural wall looks like that of a cold abscess. Adhesions often bind the two pleural surfaces together, and in this way a collection of pus may become encapsulated.

Empyema is usually confined to one side, but both may be involved, either simultaneously or one a short time after the other.

The pus is usually thick, yellow, and without odor except in the putrid form. The pus from a tubercular empyema is nearly always rich in fat-globules, and is often slightly colored by blood.

As in other cases of long suppuration, amyloid changes in other organs may supervene upon an old empyema.

Symptomatology.—The symptoms of empyema closely resemble those of pleurisy, but in the acute infections the fever is likely to be higher and more sustained than in non-infected serous effusions. A tubercular empyema often comes on insidiously and with little elevation of temperature.

When an empyema develops early in the course of a pneumonia, it may be suggested only by a marked increase in the respiratory difficulty. If it comes on later in the course of the illness, after the pneumonia has begun to improve, its onset may be marked by a rise of temperature and a return of cough and dyspnœa. A primary empyema may have an extremely rapid onset, with a clinical picture not unlike that of pneumonia. The chest may be found filled with pus on the third day. When the empyema complicates one of the infectious diseases or follows a septic process in another part of the body, cough, shortness of breath, and a stitch in the side are the first signs which draw attention to the chest. A slight increase in the number of respirations is often the only symptom noticed at first.

Diagnosis.—The symptoms resemble those of a simple pleurisy so closely that without aspirating a diagnosis is usually impossible. The constitutional symptoms are apt to be more severe in empyema, but this is by no means an invariable rule. Emaciation is often marked, even after only two weeks. The absence of transmission of the whispered voice is not a positive point in diagnosis, though it often is of value. The age of the child is a guide in making the diagnosis, as in a child under three the effusion is almost invariably purulent. In the first ten years pus is much more frequent than serum.

In aspirating we have a positive means of diagnosis. The greatest care should be used in this operation, so as not to infect the fluid if a serous effusion be found, or introduce more virulent bacteria into a fluid already infected by one of the milder organisms. Needles should be boiled before use, the chest-wall carefully cleaned with soap and water, ether, and corrosive sublimate, and the same precautions taken for clean hands that would be taken for a more formidable operation. If the arm be raised above the head the ribs are more widely separated, and there is less danger of striking one. Too small a needle may be obstructed by a mass of fibrin. Too short a needle or too deep a puncture may also lead to error, the former by not reaching the fluid, the latter by crossing it and perforating the lung or diaphragm. Puncture of the lung is not liable to occur except when, through fear of striking the diaphragm, the puncture is made too high, where the layer of pus between the chest-wall and the lung is thin. The puncture is best made in the sixth interspace if the effusion is on the left, or in the fifth if it is on the right. The fluid withdrawn should be examined bacteriologically, as upon the organisms present may depend the treatment of the case, and we are often greatly aided by this examination in giving our prognosis.

The most frequent error in diagnosis is mistaking an empyema for an unresolved pneumonia, and the aspirating-needle is often the only means of reaching a positive opinion. In an unresolved pneumonia there is dulness over only one lobe, as a rule, and we hear râles and, perhaps, friction-rubs. Moreover, the heart is never displaced. In an empyema the area of flatness is greater; we have no râles, and the heart may be displaced, especially if the fluid be in the left side. An encapsulated empyema is usually, though not always, behind and at the base.

At times the displacement of the heart is the only physical sign by which we can distinguish a solidification of the lung from fluid, and this cannot be considered a positive sign, as the heart may be forced out of place by a new growth. There is usually marked leucocytosis in empyema, though statistics are as yet rather meagre on this point.

Prognosis.—The prognosis depends upon the cause, the age of the patient, and the duration of the disease before coming to operation. The most favorable cases are those following a pneumonia where the pneumococcus is the organism present. Where the streptococcus is found associated with the pneumococcus the result is not affected. In those cases Netter places the mortality at 2.3 per cent. in adults. In children the mortality is probably somewhat higher, especially in those under one year, but we have not yet definite figures on any number of cases. In older children the result is almost always good.

In the streptococcus and staphylococcus empyemata so many factors have to be considered that it is impossible to give a prognosis except in an individual case. Coming on in the course of scarlet fever, abscess of the tonsil, or septic process in some part of the body, we must consider the source of the empyema in giving a prognosis. Tuberculosis is a rare cause of empyema in children, and the prognosis is generally bad. Occasionally, however, cases recover. As a rule, fistulæ remain; often operations for their relief fail, and the child finally dies of exhaustion or from tuberculosis in other parts of the body.

The duration of the disease before coming to operation is another important factor. In cases where it has existed for a long time the lung is much more compressed and far more liable to be bound up by adhesions than in cases operated upon in the first month or six weeks. Treatment makes more difference than anything else in the final result of empyema. Occasionally a case recovers without operation, either through rupture into a bronchus or externally, and, very rarely, by absorption of the fluid. In cases where the patient comes to early operation the prognosis is much better.

The secondary abscesses, which are quite common, such as otitis media, periostitis, or abscess of scalp, are very serious, and often sufficient to turn the tide against the patient.

Treatment.—As soon as it is determined that pus is present, it should be treated as any other collection of pus would be,—that is, by open incision and drainage. In the cases where the streptococcus, staphylococcus, or pneumococcus is present, this treatment should always be adopted. In tuberculous cases, in view of the frequently unfavorable result of operation, aspiration may be more seriously considered. Undoubtedly certain cases of empyema are cured by aspiration. Holt has collected one hundred and thirty-nine cases treated by this method. Of these, twenty-five were cured, eight by a single aspiration; thirteen died, and the remaining one hundred and one were afterwards subjected to other treatment. As a temporary measure it is often useful, but even here it is a question whether the shock is much less than from an open incision.

A method which has been used by some with good success is to make a puncture with a large trocar and pass a small drainage-tube through it. The trocar is then removed and the tube left in. The operation is hardly less serious than open incision. Where this method is used, carrying the tube into a bottle filled with antiseptic fluid is a useless precaution. The longer tube gives more chance for infection, and a large, sterile, absorbent dressing is safer, and far more convenient, especially with children.

Where open incision is decided upon,—and it always should be, except perhaps in tubercular cases,—a preliminary aspiration is to be advised only in cases of double empyema. The removal of a part of the fluid before operation causes the lung to expand less thoroughly, and on the thorough expansion of the lung the success of the operation depends.

The chest-wall should be cleaned as for any other operation. The incision should not be less than two inches long, in the posterior axillary line, and not lower than the sixth interspace, especially if the empyema be on the right side. Usually resection of a rib is not necessary. Two drainage-tubes should be used as described in the original article on page 712 of vol. ii. of this Cyclopædia.

On the question of irrigation there is considerable difference of opinion, and many believe that it is dangerous and delays recovery. Irrigation with sterile water at a temperature of 125° F. can have no bad effect, and certainly aids greatly in the removal of clotted fibrin. After the first washing out, irrigation is unnecessary except in putrid cases. Nothing stronger than a two per cent. solution of boric acid or a one to twenty solution of chlorinated soda should be used. The valvular form of dressing described in the original article is useful when it keeps in place, but the majority now use simply a large sterile, absorbent dressing.

Unlike abscess-cavities, empyemata do not heal by granulation, but by expansion of the lung, rising of the diaphragm, and contraction of the chest-wall. If, after freely opening the cavity, the lung does not tend to expand, it can often be made to by exercises such as the one devised by James, where the child, by blowing, forces a colored fluid from one bottle into another set on a slightly higher level, by massage and passive movements of the chest, or by blowing on a wind instrument.

If these fail, we can try to make the chest-wall adapt itself to the smaller size of the lung. There has been a tendency to resection of the ribs in recent cases, but it gives a certain amount of deformity, adds to the danger of the operation, and should be reserved for old cases where the lung does not expand. In Estlander's operation, where considerable portions of the ribs are resected, the mortality is high in children, and in those under two years of age it is a very fatal operation.

In bilateral empyema several days should elapse before the second side is operated upon.

FRACTURE OF THE CHEST-WALL.

In some of the doubtful cases, where we suspect fracture, but are unable to find it, the X-rays are a help. Children especially are very easy to examine in this way, as the chest-wall can be photographed or examined with the fluoroscope very readily. This subject is fully considered in vol. ii. p. 716 of this Cyclopædia.

THE DISEASES OF THE BLOOD.

By ALONZO ENGLEBERT TAYLOR, M.D.

ANÆMIA.

For convenience' sake the anæmiæ will be considered as primary and secondary. The secondary anæmiæ fall under the consideration of this section only as they are of importance in the study of the essential blood-diseases, and since in such cases symptoms which are due to the anæmia and not to the primary condition are in the main the same as those presented by the primary anæmiæ, only the blood-changes themselves in the secondary anæmiæ will be briefly summarized, in order that they may be compared with those to be described as existing in the primary anæmiæ. It must be borne in mind that the blood of children is less resistant than that of adults,—that it reverts more easily to the embryonal type and regenerates less quickly. In secondary anæmiæ both quantitative and qualitative changes are more marked than in similar conditions in adults. This is as true of the white as of the red cells. Slight causes produce marked leucocytosis in infants. In infants fed every three hours leucocytosis is almost constant. The lymphocytes are normally abundant in infants' blood, so that an excess of them does not have the meaning it would in an adult. As a large number of cases of infantile secondary anæmia are accompanied by a leucocytosis, so a large number of them are accompanied by splenic enlargement. The classification of secondary infantile anæmiæ of Monti is as follows:

- 1. Congenital . . . .
    - Syphilis.
    - Tuberculosis.
    - Other infections.
- 2. Acquired . . . . . .
    - Hemorrhagic . .
        - From navel, etc.
        - The purpuric conditions.
    - General . . . .
        - Visceral disease.
        - Tuberculosis.
        - Syphilis.
        - Rickets.
        - Gastro-intestinal disease.
        - Sepsis.
        - Post-infections.
        - Subnutrition.
        - Poor hygiene.
        - Nephritis.
        - Neoplasms.

The anæmiæ due to syphilis, tuberculosis, sepsis, rickets, and gastro-intestinal disease are usually accompanied by splenic enlargement.

Conditions of Life and of the Individual Constitution, Independent of any Distinguishable Disease.—Here are to be included age and sex, improper hygiene, malnutrition, subnutrition, overwork, social and psychic conditions, inherited hæmic or other systemic or developmental weakness. Careful study, by demonstrating definite causal diseases, will greatly reduce the cases in this category, yet some cases will remain which must be grouped here. Children under the best of conditions seem inclined to anæmia, and female children more than male. Often patients slightly anæmic recover spontaneously when removed from any of the unfavorable conditions. Immigrants often show a pseudo-chlorosis, which passes away with acclimation. In some families all the members have poor blood; their blood is never up to the standard of their conditions; it is disproportionately reduced by disease and recovers slowly. That developmental conditions have an influence seems probable. Sailer and the author found that the average children in the Pennsylvania Institution for the Feeble-Minded, living under excellent conditions, were notably anæmic. It is doubtful whether climate *per se* is of influence; the notion is based upon appearances of complexion more than upon blood-study.

Infectious Diseases.—These all produce anæmia, which may, however, be less apparent on blood-examination during than directly following the attack. In a general way the severity of the attack determines the degree of the anæmia, especially in hemorrhagic cases, but there are notable exceptions to this.

Measles.—Uncomplicated cases rarely reduce the hæmoglobin or the red cells more than ten per cent. There is no leucocytosis, and qualitative changes are not known to occur. The same applies to Rötheln.

Scarlet Fever.—There is a marked reduction of the red cells in many cases, especially the nephritic; when the fever falls they are reduced to or below 3,000,000, the color index being disproportionately reduced. There is leucocytosis, from 12,000 to 20,000 per cubic millimetre, often with an excess of oxyphilic cells. Myelocytes have been seen.

Small-Pox.—Marked oligocythæmia and disproportionate oligochromæmia seem to be the result of this infection. Leucocytosis is absent (or mild) during the period of initial fever and eruption; when pustulation occurs it is constantly present. It would seem that in typical vaccination there is no leucocytosis.

Varicella.—Leucocytosis was not present in the few cases that I have examined.

Pertussis.—Leucocytosis is present during the paroxysmal period.

Influenza.—The hæmoglobin and red cells are not much affected by ordinary cases. There is, as a rule, no leucocytosis, nor do the complications, as pneumonia, gastritis, and meningitis, seem to produce it unless due to mixed infection.

Diphtheria.—In most cases the hæmoglobin and red cells are markedly reduced. There is leucocytosis from the first appearance of the membrane, often up to 40,000 per cubic millimetre. The extent of the membrane seems to influence the height of leucocytosis, which is also increased in the cases of mixed infection. In fulminating cases it may not occur. Antitoxin exerts no hæmolytic action; on the contrary, competent observers report that under its use the anæmia is less marked and the subsequent regeneration more rapid. Myelocytes are present in bad cases.

Cholera.—Owing to the profuse discharges, the blood is oligohydræmic, and the red cells and hæmoglobin are above normal, but fall below normal during convalescence. Moderate leucocytosis is present.

Acute Rheumatism.—In this condition there is usually a rapid and marked destruction of the red cells, with a reduction in the color index. Moderate leucocytosis is present, but is not exaggerated by endocarditis.

Typhoid Fever.—The red corpuscles are moderately reduced; the color index is low. Leucocytosis is not present; indeed, leucopena is usually present towards the close of the febrile period. The mononuclear cells are increased. Complications may or may not produce leucocytosis. I have seen extensive pneumonia without it. On the contrary, hemorrhage, perforation, and suppuration usually provoke it. Possibly those complications due to the typhoid bacillus do not cause it, while it accompanies those due to other bacteria. During convalescence the anæmia may be marked. Widal's blood-reaction for typhoid fever is based on the phenomena of agglutination and degeneration as described by Pfeiffer, occurring after intra-peritoneal injections of bacilli into immune animals. The essential idea is that the blood of typhoidal patients checks the motility and induces agglutination of the specific microbe. The test is simple. One part of blood is mixed with at least twenty parts of a bouillon culture or an emulsion of an agar culture, and a drop examined in the hanging drop; motility should cease and clumping of the bacilli occur within thirty minutes. Pseudo or partial reactions should be guarded against: these occur often in non-typhoidal blood, especially where the dilution is low, and also in typhoidal cases before or after the appearance of a complete reaction. A clear, positive reaction may be regarded as almost indisputably diagnostic. The reaction generally appears early in the disease, and lasts a variable time; in rare instances it persists for years; often it disappears during convalescence.

Of *typhus* it is known only that it is accompanied by leucocytosis.

Alterations in the red cells have been noted in all these infections; they have not the importance often ascribed to them. Alterations in the degree of alkalinity have not been quoted, because, as a rule, the methods have not been trustworthy. Generally the specific gravity follows the red cells and hæmoglobin. A hypalbuminosis of the plasma in the native volume of blood has not, to my knowledge, been shown.

Conditions of Sepsis.—It may be stated as a general rule that, either by pyogenic bacteria or by the reactions between them and the organism, substances are evolved which are markedly hæmolytic. It is not known whether hæmolysis is greater in infections caused by certain bacteria than in those caused by others. To some observers the streptococcus pyogenes has seemed especially active; certain it is that there are wide ranges in degrees of virulence of the same bacterium. The virulence of the individual case on the one hand, and the tissue affected and the extent of absorption from the diseased area on the other, may serve to explain the greater or less hæmolysis in different cases. Wide-spread areas of infection, as in cellulitis, the lack of local defensive reaction, with free absorption, and a natural impossibility of evacuation of pus, favor septic hæmolysis. Suppuration of serous membranes is usually severe. There are, however, anomalous cases, and there may be less hæmolysis in wide-spread peritonitis than in autopsy infections whose local lesions seem very trivial. As a rule, there is marked and rapid destruction of red cells, and to a certain extent this runs parallel to the severity of infection. This is especially true of septic arthritis, peritonitis (except gonorrhœal), pericarditis, pleuritis (less often), meningitis, and visceral abscesses. Erysipelas, suppuration of mucous membranes, and peripheral abscesses are, as a rule, only mildly hæmolytic. The red cells often show polychromatophilia, but distortions are not marked. The reduction of hæmoglobin is commonly greater than that of the cells, and hæmoglobinæmia is not uncommon. In some cases the blood clots very rapidly, in others very slowly; why, we do not know. Albumoses may be present in the plasma, and the alkalinity of the latter is held to be reduced. As a rule, there is leucocytosis, due to an increase in the polymorphous cells. This is usually up to 20,000, and may reach 75,000 per cubic millimetre; the degree usually corresponds to the severity of infection, yet some of the fulminating cases show none. Such a leucocytosis is independent of fever, and is associated with sepsis; the absorption of hemorrhagic or other aseptic exudates will not produce it. It is of obvious importance in the differentiation of septic from tuberculous bone- and joint-disease, of appendicitis from typhoid fever or enteritis, or of meningeal or cerebral abscess from non-septic conditions with similar signs. Cultures should be made by drawing into an empty sterile tube a few cubic centimetres of blood obtained by venepuncture and then treated according to the general methods prescribed for the various bacteria.

In the specific chronic inflammations (the granulomata) the hæmolytic action is less marked than in sepsis. In mild cases of tuberculosis the number of red cells may not be reduced, but the hæmoglobin usually is. More advanced cases present a pseudo-chlorosis, slight or moderate oligocythæmia, disproportionate oligochromæmia. Severe cases may cause a marked reduction of both down to or below thirty per cent. Poikilocytosis is usually only moderate. Nucleated red cells are unusual. The degree of fever seems to make little difference. As a rule, there is no leucocytosis in tuberculosis unless complications exist, such as hemorrhage or septic infection, as in lungs or kidneys. There are, however, a few cases on record of meningeal and bone tuberculosis with leucocytosis without discoverable complication. When mixed infection occurs, not only is leucocytosis produced, but hæmolysis is exaggerated. There is no evidence that any leucocytic changes are characteristic of tuberculosis, nor are any qualitative alterations constant. Myelocytes may be present.

Syphilis.—There are no changes during the primary stage. During the secondary stage there is a pseudo-chlorosis, which is more marked during the third stage, and finally the number of cells may be much reduced. It is in hereditary syphilis in children that many of the most anæmic cases are found, often with splenic and lymphatic enlargement. They have often been classed as instances of splenic anæmia or Hodgkin's disease, or, in cases with marked leucocytosis, as leukæmia. Loos has described a polychromatophilia held by him to be quite peculiar to the blood of inherited syphilis. In the only case under direct observation I have failed to confirm this. Leucocytosis is absent in the primary stage, present in the eruptive stage, and usually absent in the tertiary stages, also in hereditary syphilis, but may be present in the anæmia gravis of syphilis. In the eruptive leucocytosis many oxyphilic cells are present; in the tertiary and post-tertiary stages the lymphocytes may be increased. It has been held that in syphilis the immediate effect of the action of mercury is a sudden fall in the hæmoglobin (Justus's reaction). It is the general experience that mercurial treatment rapidly amends the anæmia.

Glanders in the acute form is known to present a leucocytosis, in the chronic form none. *Actinomycosis* also produces leucocytosis.

Respiratory Diseases.—Acute inflammations of the upper passages and bronchi may cause a mild leucocytosis. Croupous pneumonia produces a moderate or marked hæmolysis, apparent after the crisis. There is, except in fulminating cases or in cases of low typhoidal type, a marked leucocytosis which bears no constant relation to the degree of consolidation and drops with the true crisis. The leucocytosis of catarrhal pneumonia is more irregular in appearance and degree. Abscess of the lung, gangrene, and tumors all cause a leucocytosis. Simple pleurisy shows little leucocytosis, septic pleurisy much more, and tubercular pleurisy generally none. In asthma there is often an excess of oxyphilic cells.

Circulatory Diseases.—*Acute Simple Endocarditis.*—There are no blood-changes known to be due to this, and, since it so often complicates other acute diseases, alterations are difficult to judge. It is known that it can cause a moderate leucocytosis. Ulcerative endocarditis affects the blood like sepsis. The most important elements in the examination are the determination of a high leucocytosis, and the cultures from venous blood. The exact effects of broken cardiac compensation in chronic *valvular disease* are not known, nor is it probable that finger-blood bears constant relations to vessel-blood. In some cases of acute failure of compensation the red cells and hæmoglobin are above and in other cases below normal, in many cases quite normal. The same is true of chronic broken compensation, and it is evident that the strength of the heart-beats and the degree of fluidity of the tissues are not the only factors which affect the estimations. In children high figures are more the rule, especially in the blood of the new-born with congenital lesions, where the count is usually much above the normal. Following a period of broken compensation a moderate pseudo-chlorosis may be present. Leucocytosis is not produced by broken compensation, but is always to be looked upon as suggesting endocardial inflammation. Hemorrhage, as a matter of course, produces anæmia in a measure directly in proportion to the loss of blood. Children bear bleeding less well than adults, and young infants may become very anæmic from trivial bleeding, such as from the navel, bowels, or following slight operations; and in them, too, leucocytosis seems to be produced by small hemorrhages, but is of the same type as hemorrhagic leucocytosis in adults.

Diseases of the Genito-Urinary System.—In nephritis there is considerable hæmolysis, no doubt toxic in nature. This is especially marked in acute parenchymatous nephritis, when the red cells and hæmoglobin may fall to below half the normal. In chronic parenchymatous nephritis the hæmolysis is usually less marked, while the oligochromæmia is often more disproportionate. In chronic interstitial nephritis the reductions are of mild degree. I have not seen the high counts described in extensive polysarcia. Leucocytosis is quite constant in acute cases and occurs very often in chronic cases, and, as far as I have seen it, it was of the ordinary type, increase of the polymorphous cells. There is no evidence that the albuminuria causes a hypalbuminæmia. In neoplasms, calculus, and suppuration the results depend upon the amount of sepsis or hemorrhage, and the same is true of similar vesical conditions. A floating kidney causes no leucocytosis. Hemorrhage from the kidney or the bladder may almost exsanguinate a subject. The immense leucocytic reserve of the body is well illustrated by some severe chronic cystitides, in which the leucocytic count may be entirely normal, while there are daily voided twenty-five times as many pus-cells as there are leucocytes in the circulating blood. In gonorrhœa there may be a leucocytosis with increase in the oxyphilic cells; both are inconstant.

Diseases of the Alimentary Tract.—Intoxication and malnutrition play varying *rôles* here, and probably account for inconstant conditions. The acute and chronic gastro-enteric diseases of children affect their blood profoundly. Where profuse vomiting or serous discharges dehydrate the tissues the blood-figures may be as high as in true cholera, and leucocytosis is then usually present. It is in chronic entero-colitis that we find perhaps the most marked secondary anæmiæ of children. The red cells may be reduced to below 2,000,000, the hæmoglobin to twenty-five per cent., or both lower. The red cells are then commonly quite distorted, and nucleated cells are common. Leucocytosis is usually present, and may be marked, with a noteworthy increase in the mononuclear forms, which, with the quite frequent association of an enlarged spleen, has led to the diagnosis of both leukæmia and anæmia infantum pseudo-leukæmia. It is probable that such a condition may become either a leukæmia or a pernicious anæmia, certainly as far as the blood-examination is concerned. In gastric ulcer marked anæmia is the rule, of the pseudo-chlorotic type, and not dependent on, though often exaggerated by, the hemorrhages; unless complicated, leucocytosis is usually not present. The same applies to duodenal ulcers. Intestinal ulcers often cause no anæmia. In gastritis, acute or chronic, anæmia is always produced, but vomiting and diminished absorption may so inspissate the blood that the counts remain high. Nervous dyspepsia with hyperacidity usually does not produce such anæmia; but there are exceptions. Leucocytosis is absent in chronic gastric and enteric inflammation, and it seems that digestion leucocytosis may also be absent, —a point which requires further study. In acute obstruction, however produced, leucocytosis may be present. There are few definite changes in the blood in the non-septic *hepatic* diseases. The cirrhoses and degenerations cause a moderate reduction of red cells and hæmoglobin, which is much modified by the gastro-intestinal conditions. Leucocytosis is, as a rule, not present except when the bile-ducts are especially involved. Acute yellow atrophy and phosphorus poisoning both present leucocytosis, as do abscess, hydatid cysts, and colangitis, while gall-stones do not.

Cutaneous Diseases.—The most important fact is that in many of the skin diseases there is a marked excess of the oxyphilic leucocytes. This may be present during the stage of eruption of the infectious fevers, but is uncommon and less marked, and may be of value in differential diagnosis.

Nervous Diseases.—Children with congenital defects of nervous development, or with hereditary nervous vices, generally present a moderate simple anæmia. As a rule, the blood is influenced only by associated conditions. Burr has shown that the finger-blood is normal in red cells and hæmoglobin in the acute chorea of children. Nervous disease is much more often a result than a cause of anæmia. In the insane, anæmia is often marked. Neuritis generally presents a leucocytosis, while neuralgia and neurasthenia do not. It is improbable that brain-abscess can be diagnosed from brain-tumor by the blood-count, but tertiary syphilis might be excluded.

Constitutional and Unclassified Diseases.—*Rickets* is usually accompanied by a notable anæmia. In nearly all cases the red cells fall below 3,500,000 and the hæmoglobin to fifty per cent.; often the red cells descend to below 2,000,000, and the hæmoglobin proportionately. Poikilocytosis is the rule, and nucleated red cells are common. A leucocytosis is generally held to be present; often there is an excess of lymphocytes, and myelocytes may be present. The spleen is often markedly enlarged, and a large number of cases of splenic anæmia and pseudo-leukæmia infantum belong here. In *myxœdema* the sparse studies indicate that there is little anæmia and no leucocytosis. In some cases the red cells have been described as of very large size. In *scleroderma* I have been able to find in five cases no changes beyond moderate simple anæmia and an excess of large mononuclear leucocytes; leucocytosis was never present. In *gout*, as a rule, there is little anæmia. Neusser has described perinuclear basophilic granulations in the leucocytes. In *diabetes*, when the question of water-absorption and elimination is set aside, there will usually be found to be a moderate pseudo-chlorosis without leucocytosis, and late in the disease rather marked anæmia. Fat, glycogen, and sugar are increased in the blood. The blood generally presents the diabetes reaction of Bremer. In many of the reported studies of the blood in *exophthalmic goitre* it has been given as nearly normal. I have never seen a case without a moderate or a marked degree of chlor-anæmia without leucocytosis.

Malignant Neoplasms.—Benign neoplasms affect the blood only through hemorrhage or mechanical effects; malignant tumors affect it also by intoxication. Carcinoma produces an anæmia severe according to the tissue involved, especially severe in visceral cancer. Early in the cases the condition is that of a pseudo-chlorosis; later the reduction of red cells and hæmoglobin may be quite as low as in pernicious anæmia. Cancer of the œsophagus, or otherwise so situated as to obstruct the ingestion or absorption of water, gives high blood-counts. Poikilocytosis is quite usual, and nucleated cells are not uncommon. Leucocytosis is present in most cases, highest when complicated by metastasis or hemorrhages; it is absent in œsophageal cancer. The percentage of the large mononuclear cells is increased, and myelocytes may be present. Sugar has been described as in excess in the plasma, and in carcinoma ventriculi an absence of digestion-leucocytosis. In sarcomata, especially in children, the anæmia is usually marked, and the leucocytosis (especially the lymphocytes) is higher than in cancer. In these cases the spleen may be enlarged. For both cancer and sarcoma rapid regeneration with disappearance of the leucocytosis has been reported following removal by operation, and a recurrence of the leucocytosis noted as an early sign of recurrence.

Parasites.—The parasites which may cause anæmia are the plasmodium malariæ, the filaria sanguinis hominis, the tæniæ, the distomæ pulmonalis and hæmatobium, the ankylostomum duodenale, the bothriocephalus latus, the anguillula intestinalis, psorosperms, the eustrongylus gigas, the ascaris lumbricoides, and the oxyuris vermicularis. Parasites produce anæmia by direct hæmolysis (as in malaria), by the abstraction of blood, or by intoxication. The worst cases are produced by malaria, the ankylostomum, and the bothriocephalus latus, in which the red cells may be reduced to 1,000,000 and the hæmoglobin to below twenty-five per cent. Parasitic anæmiæ are uncommon in children, and intestinal worms cause less anæmia than the appearances of the subjects would indicate.

CHLOROSIS.

Chlorosis is an essential blood-disease of obscure origin. A great many factors have been believed to contribute to its occurrence. It is most often seen in girls between the ages of twelve and nineteen years, usually appearing a couple of years after the establishment of menstruation, often apparently determined by it, and notably frequent in girls of irregular adolescence. It is not uncommon in children below twelve years and in women over twenty; it is very rare in men and in the aged. It occurs independently of climate or season, although it may be aggravated in the winter. It seems to be a Caucasian disease; at least, the negroes in this country are rarely chlorotic, and this accords with the experience of Stengel and Thayer. It is not more frequent in cities than in the country, but is more common now than in the last generation. Subnutrition, malnutrition, indigestion, and constipation have been often incriminated. Poor hygiene, with all which that implies, especially a miserable in-door life, with no exercise except drudgery, and constant or excessive physical or nervous exhaustion, are important conditions. The constitution often seems of influence; delicate, highly strung, nervous girls with no reserve power seem especially liable. Mental and emotional conditions, nervous shock, sorrow, mental degeneracies, and social conditions are sometimes of possible influence. Some cases are directly hereditary, many occur in tuberculous, syphilitic, and neurotic families, and the French writers insist that it also occurs in the "arthritic diathesis." Change of residence may appear to produce it, as in emigrants. It has in various ways been connected with the menstrual function, while pregnancy and lactation are held to cause it, especially in those who were previously afflicted. Some cases begin during convalescence from acute or chronic disease of various kinds. The truth is that the etiology is obscure. Of the above-named factors some are merely associated conditions, some are results, while the few which are of causal influence cannot be held to be the fundamental basis. It seems most probable that there is some condition of defective hæmatogenesis inferred as resident in the hæmatopoietic organs. It is probably not toxic, and there are no evidences of excessive hæmolysis. Animals have been made quite chlorotic by iron-starvation from birth, but there is no evidence that this is the cause of chlorosis, nor has it ever been shown that chlorotics absorb and contain less iron than is physiologically necessary. It has been claimed that abnormal substances hold the iron and render it unavailable for hæmoglobin. The disease is quite certainly not of gastro-intestinal origin; it cannot be a simple condition of malassimilation; there is no excessive hæmolysis; there are no chemical or metabolic evidences of excessive intestinal decomposition, or that the iron of the diet is rendered unabsorbable by abnormal chemical conditions in the stomach or intestines. Nor has it been shown that in chlorosis melæna is constant or even common. Forchheimer has claimed that the intestinal tract is the chief site of the formation of hæmoglobin, and that thus intestinal disease causes chlorosis; the primary statement, however, is not proved. The theory of Virchow was that an arterial hypoplasia was the organic cause. There are, however, no physiological reasons why arterial hypoplasia should produce the peculiar condition of chlorosis, or any anæmia, for that matter; for, apart from the capillaries of the bone-marrow, the arterial system has after infancy no known relations to blood-development or blood-destruction; and, as a matter of fact, many chlorotics lack the arterial hypoplasia, while non-chlorotics possess it. The same is true of genital hypoplasia, of which the theory is that it causes a reflex sympathetic neurosis which in some way depresses hæmatogenesis; but, apart from menstrual disturbances, chlorotic girls fulfil their sexual functions quite as well as other women. A poor but ancient hypothesis is that it is a menstrual auto-intoxication by substances normally eliminated in the menses. Von Noorden has recently suggested that the ovaries may possess an internal secretion, disturbances in which may be the ulterior cause of the defective hæmatogenesis. The suggestion of Bunge that the reason why girls are subject to chlorosis may lie in the fact that the adolescent female is supposed to be storing up iron to be used in endowing the livers of her offspring does not lie within the range of demonstration, though it is known that an increase of iron in the mammæ and uterus occurs during adolescence. Many French clinicians transfer the causation to the nervous system, to organic or functional disturbances, which have, however, never been particularly defined for either. The same defect holds here. Not only do we know nothing of the relations of the nervous system to hæmatogenesis, or of its pathological states to the blood, but practically no clinical relations obtain. It is easy to say that an irritable or degenerated cord alters the blood-nutrition, that a splanchnoptosis or a chronically impacted colon disturbs the splanchnic sympathetic system, and that an irritated vaso-motor system alters both the physical and the chemical behavior of the blood-plasma; but that in no wise explains the causation of chlorosis. By exclusion, most hæmatologists have thus come to accept as the cause some defect of hæmatogenesis. For this view there is admittedly no direct proof; but, since we consider the deficiency in hæmoglobin as the cardinal feature in chlorosis, it follows that, since there are no evidences of hæmolysis, and since the elements (including iron) needed in the molecule of hæmoglobin are in the body in plentiful amounts, there must be some fault in the functions which manufacture hæmoglobin and place it in the red cells; or the formation of the red cells themselves is defective, so that they are not able to hold the normal amount of hæmoglobin; and thus we have come to believe that the reason the red cells have so little hæmoglobin is because abnormal hæmatopoietic organs did not fulfil their function properly. Once such an indefinite hæmatopoietic weakness exists, any one of the aforenamed conditions can promptly bring it into full activity with the production of chlorosis. Clinically there are many cases termed "transitory" chlorosis, all of which are probably secondary anæmiæ. True chlorosis is of two types: the one class have real chlorosis usually only once; the second class (a small number) are habitually chlorotic.

Pathology.—The patients often present a peculiar greenish-yellow *color* of the skin, varied according to the individual complexion, the pigmentary changes of which have not been well studied. Chlorotics, as a rule, are fat, but not in excess of the best of health for the subject affected; a few cases become lean. The fat is not pathological; there is no evidence that it is due to suboxidation or other metabolic disturbance. The *muscular* system is soft and flaccid during life, but no abnormal conditions except under-development have been noted post mortem. There may rarely be petechiæ into the skin, subcutaneous tissues, and muscles. No alterations have been noted in the *bones*, except splenification of the marrow, which has been uncommon and not marked. The skeleton may be undersized. The *thyroid* gland may be enlarged, probably a vascular rather than a parenchymatous or a fibrous enlargement. The eyeballs may bulge outward; this has not been noted as constantly associated with thyroid enlargement. In a number of cases there is a hypoplasia of the *heart* and large blood-vessels. Of the few cases which have come to autopsy some had entire symmetry and undoubted physiological balance; in others the right ventricle was enlarged, less often the left or both; in other cases there has been hypertrophy of one or both ventricles. The auricles may be dilated, and lately attention has been directed to dilatation of the conus arteriosus. The endocardial lining is often very pale; old endocarditis is not uncommon, most often on the mitral valve. The heart-muscle has usually been pale, with moderate uniform fatty change or with large areas or strands of degeneration. The aorta has often been small, its endothelium and muscularis affected like the heart, and of abnormal elasticity. In some cases there have been many arterial anomalies, especially of the intercostal branches. Arterial thrombosis has been very rare, usually cranial. Venous thrombosis has been often noted, most often in the femoral veins and within the skull. The blood-pressure has not been carefully studied; owing to vaso-motor conditions, it is probably lowered. Hemorrhages are uncommon, most often from the nose, bronchi, stomach, and uterus, rarely into the tissues. Lesions of the *pulmonary system* have been rare, œdema is uncommon, and venous thrombosis is more rare. The O-income and CO_2-output have been found normal or above normal. In many cases the diaphragm is abnormally high. Alterations in the *alimentary tissues* are common. Chlorotics often have bad teeth, but whether peculiarly frequently is not known. The gastric and intestinal epithelium has been seen in fatty degeneration in a number of cases; ulcerations have been found in both, more often in the stomach, to which hæmatemesis is usually to be attributed. Gastrectasis is quite common, moderate in degree. Gastroptosis is not infrequent, sometimes accompanied by enteroptosis or even splanchnoptosis. The HCl acidity is often normal, often excessive, less often defective. For pepsin and lab-ferment no diminution has been shown. Gastric motility is usually quite normal, unless there be dilatation. Of intestinal digestion little is known; the stools give no evidence of faulty assimilation, except that in some cases the fats have not been well absorbed, and the stools had an acholic appearance. The colon may become impacted, and may present mucous colitis. Careful studies of the food, the stools, and the urine seem to show that the digestion, absorption, and nutrition of chlorotic subjects are normal. The liver may be rarely enlarged, and has been seen moderately, even excessively, fatty. The pancreas has been noted as fatty. In a few cases the *kidneys* have presented signs of vascular nephritis; more often they have been normal. Movable kidney may be present, especially in cases with gastroptosis. The urine is usually normal in color, specific gravity, and quantity, but polyuria is often present. The coloring matters, sulphates, phosphates, and chlorides are usually within normal limits; the chlorides, however, may be deficient. The nitrogen compounds have rarely been studied under properly controlled conditions; there is no conclusive evidence that nitrogen-elimination is excessive, or that any particular compounds are morbidly altered. A trace of serum-albumin and globulin may be present; in one case I have seen a mild glycosuria. A few hyaline casts, or, rather, cylindroids, are occasionally observed. Changes in the *genitalia* are commonly described, usually a hypoplasia of the ovaries and uterus, with persistence of the infantile type and of the infantile pelvis, accompanied sometimes by mammary hypoplasia; in exceptional cases, however, the breasts have become indurated. In many cases the genitalia are normal; in some cases hyperplasia of the ovaries has been noted. Uterine displacement is often seen, more often anteriorly. Ulcerations are sometimes seen upon the vaginal cervix, less often upon the vaginal mucosa. Leucorrhœa is common. Scanty menstruation is the most common condition; complete suppression for months or years may occur; sometimes the function is regular and normal, or may even be excessive. Of the *lymphatic system* little is known. The spleen is often enlarged, but its substance has been found little altered. It has been claimed that it shows signs of excessive hæmoglobin disintegration, but this has not been confirmed. In the lymph-glands, which may be enlarged, increased pigmentation has been noted, a frequent occurrence under other conditions. Slight degenerations have been noted in the abdominal and cervical glands, in the splanchnici and the vagi, and in the cord and brain; they seem to be secondary. Hemorrhagic retinitis, optic neuritis, neuro-retinitis, retinal degenerations, and scleritis are uncommon conditions. The eye-grounds are usually pale, and the vessels may pulsate.

The Blood.—We have no way of determining the total quantity of the blood. In chlorosis bleeding is natural, or often uncommonly free, but that can be better explained by vaso-motor conditions than by an assumption of plethora vera. The reaction has not been sufficiently studied by accurate methods to permit of a definite statement concerning it; it is probably normal. The specific gravity is decreased, due, probably, entirely to the fact that many corpuscles are absent and their place taken by water, and that the weight of the red cells is very much reduced; it may fall below 1025, usually not below 1035; there is a proportionate reduction in the dried residue. The quantity of fibrin has not been carefully studied. Hypalbuminosis is constant, most likely of cellular origin. The sodium is in excess, the potassium is decreased, the chlorides and phosphates increased, the calcium normal; and, unless it can be held that the chlorotic plasma contains an excess of iron, the decrease in the iron, denied by Biernacki, has been proved in the blood of animals rendered poor in hæmoglobin by iron-starvation. Chlorotic plasma has been shown to be hæmolytic, whether by chemical or by toxic action is not known. In a few cases the red cells are within the normal limits. In the majority of cases the number varies from 2,500,000 to 4,000,000. In not a few severe cases it falls under 3,000,000, and rarely below 2,000,000. The pallor of the cells is the most striking feature of stained preparations; polychromatophilia may be present. Poikilocytosis is common in the severe cases, and microcytes and macrocytes are often likewise present. Nucleated red cells (which may appear in crises) are common, usually normoblasts; microblasts and gigantoblasts are rare. As regards shape, size, and nucleated forms of the red cells, in severe chlorosis conditions may be as pronounced as in pernicious anæmia. The reduction in hæmoglobin is the most marked feature of the blood of chlorosis. In no other condition is the oligochromæmia so marked, constant, and persistent. In mild cases, where the red cells are not reduced below 4,000,000, the hæmoglobin is often below fifty per cent., while in severe cases it falls below thirty per cent. The disproportion is not so striking when the oligocythæmia is marked; then the globular value is higher. It has been held that the hæmoglobin itself is altered in chlorosis, since an especial slowness of reduction has been described in this disease. The resistance of the red cells has been found diminished. Leucocytosis is not present in uncomplicated chlorosis, nor are any constant qualitative changes to be determined, though in very bad cases there may be an excess of lymphocytes. Degenerated leucocytes are rare. Myelocytes have been seen. The blood-plaques have been described as excessive; plaque-technique is, however, so difficult and faulty that little reliance can be placed upon it.

Symptoms and Complications.—The onset is usually gradual, rarely abrupt. The first symptoms may be general, or may be referable to the nervous, circulatory, or alimentary system, or to the menstrual function, while in rare cases the subjects complain of no symptoms at a time when the skin and blood are obviously chlorotic. Of general symptoms, apathy, inability, and disinclination to perform any mental or physical functions are almost invariable.

Circulatory Symptoms.—The pulse is usually rapid, soft, and weak, of soft tension, and it may be dicrotic, although in some cases curious vasomotor storms render the pulse hard and wiry. Variations in frequency are common; abnormal arterial pulsations (except the capillary pulse) are uncommon except in attacks of violent palpitation, and are best seen in the neck. In some cases the abdominal aorta pulsates inordinately. Systolic and double murmurs have been described in the arteries, especially the femorals. Venous pulsation is frequent, most marked in the jugulars (negative venous pulsation, not a regurgitation). There may be a faint thrill, and very often a peculiar continuous murmur, usually heard only, and always best, at the root of the jugular veins. This venous hum is exaggerated by the erect posture and by deep inspiration. It can be obliterated by peripheral pressure on the vein. It may be heard in other veins. Arterial thrombosis is very rare; venous thromboses are not uncommon (exercise seems to favor them); most often seen in the femoral veins, the intra-cranial sinuses, and various external and internal veins, and add the symptoms of the complication according to its site; death has been due to pulmonary embolism from venous thrombosis. Hemorrhage is an infrequent complication. Epistaxis, menorrhagia, and hæmatemesis are the most common, in the order given, and they are often local in origin. There are always *cardiac* symptoms. In bad cases all the time, and in other cases upon slight provocation, the heart's action is accelerated. It may be irregular, and spasms of violent palpitation are common; they may be of hæmic or of nervous origin. Upon percussion the area of dulness is most often normal, sometimes enlarged, especially to the right, rarely decreased. The apex-beat is usually close to the normal area; it may be strong or weak, and is often widely diffused. The most marked venous pulsations are synchronous with the apex-beat. Murmurs are present in over eighty per cent. of cases. A basal systolic murmur is the most common: loudest over the pulmonary area, usually soft and blowing, rarely harsh, not transmitted, and very rarely accompanied by a thrill; this probably originates at the mitral valve. A systolic apical murmur is next in frequency; soft, often transmitted to the left or right, rarely with a thrill. Either of these is rarely loud enough to be heard all over the chest, or even in the back. Diastolic murmurs do not occur at the pulmonary or mitral areas unless the result of complication. Less frequent are systolic murmurs at the aortic and tricuspid areas. At the aortic area diastolic murmurs are sometimes heard. The first sound of the heart may lose its muscular quality. With the advent of right ventricular hypertrophy the second pulmonary sound becomes accentuated. Myocardial and blood changes are held to be mainly responsible for these murmurs. More rarely there is evidence of increased intra-cardiac pressure or of relative valvular incompetency. Acute endocarditis is an uncommon complication, affecting the valves of the left heart, usually the mitral. It is apt to be simple, rarely malignant.

Respiratory Symptoms.—A slight chronic laryngitis is not uncommon, the secretions are scanty, and rhinitis is thus also frequent. Functional aphonia has been rarely noted. Dyspnœa upon slight exertion is seen in all cases, dyspnœa independent of exertion in bad cases. That the dyspnœa fully compensates for the deficiency in hæmoglobin is shown by the fact that the O-income and CO_2-output have always been found normal. The respiratory rhythm may be altered; the sounds are usually weak. A dry cough is common. Bronchitis is an occasional complication. Tuberculosis develops in a suspicious number of chlorotics.

Alimentary Symptoms.—The buccal secretions are usually scant, the tongue coated and furred; the breath may be fetid. Attacks of salivation (vaso-motor?) have been noted. Hamill found the saliva normal in six cases. Simple ulcers of the mucous membrane are not uncommon; thrush is rare. Variations of appetite from anorexia to bulimia are common; not rare are pica and geophagia. Nausea is common, vomiting less so. Digestive distress is almost constant, most often after eating, but quite typical gastralgic attacks may occur on an empty stomach. The gastric distress may provoke cardiac palpitation or violent emesis. The epigastrium may be tender, and examination will reveal dilatation or gastroptosis in a surprising number of cases. The spleen may be enlarged; the liver is not. Corresponding to the normal acidity or hyperacidity and to the normal secretion of digestive ferments, test meals are usually well digested, and it has become well demonstrated that digestion is quite normal in chlorosis. Constipation is present in the majority of cases, usually moderate. Diarrhœa is present at times, in some cases constantly. Fecal impaction is rare. Gastric ulcer is an occasional complication; most of such cases are the secondary anæmia of ulcus ventriculi.

Renal Symptoms.—Apart from nephritis as a complication, which is rare, there are no symptoms. Œdema is common in chlorosis, but should always incite a careful urinalysis.

Menstrual Symptoms.—In their order of frequency the menstrual conditions may be summarized thus: scanty, irregular, suppression of, normal menstruation, and metrorrhagia. Pain may be present, and the period may be prolonged. In cases of amenorrhœa there is usually no pain at the time of expected menstruation. Chlorosis may follow the first menstruation. Ovarian tenderness may be present.

Neuro-Muscular Symptoms.—Muscular weakness is the rule; the muscles seem soft and flabby, but react normally. In the rare cases of extreme paresis the reflexes are normal or exaggerated. Headache is very common, constant or paroxysmal, moderate or severe, but not proportionate to the anæmia. Neuralgiæ are often seen, most often trifacial and pelvic. Especial attention has been called to the corset-pains in the ribs of chlorotic girls. Insomnia is seen in some cases, somnolence in others. The disposition of the subjects may be changed; in a few cases pathological melancholia or mania has developed. There is no doubt that chlorotics not infrequently have fully developed hysteria, but the anæsthesiæ, hyperæsthesiæ, and paræsthesiæ occasionally noted, as well as paralyses and convulsions, need not always be deemed hysterical, nor the sensitiveness to bright light and loud sounds sometimes seen. Chorea minor is a rare complication. The rare deafness and the occasional amaurosis not connected with optic changes are probably nervous. Ocular pain, weakness of accommodation, and concentric retraction of the fields are common.

Metabolic Symptoms.—As stated, with the exception of the chemical blood-alterations there are few signs of metabolic derangements. The temperature is usually normal, rarely subnormal; seldom is there moderate fever of regular or irregular type, or the typus inversus.

Cutaneous Symptoms.—Chlorotic patients are usually fat and soft, and a sudden loss of fat should suggest organic disease. Slight œdema, usually gravitatory, is often seen; marked dropsy is rare. The typical color of the skin is a greenish yellow, less often an alabaster white, rarely a decided flushed pink most marked about the face. The mucous membranes, especially of the lips and mouth, show most often a yellowish pallor which is striking; the conjunctivæ may be a pearly white. A few patients look almost jaundiced, especially in the conjunctivæ. Patches of dark pigmentation or of loss of pigment have been described. The skin is usually dry, often itches, and eruptions, especially acne, may be noted. The hair may become lighter in color, and the nails appear pure white. There are various symptoms which ought to be classed as *vaso-motor symptoms.* Attacks of generalized or local sweating, local flushing, or the sensation of cold areas or extremities, or shivering in the entire body, vague anginal pains, polyuria, and the enlargement of the thyroid gland are such. Not rare are prolonged attacks of angiospasm, which produce the digitus semimortuus, or angioparesis so marked that the hands present the appearance of erythromelalgia. Regarding the thyroid, what can be termed a pseudo-Graves's disease develops in some cases and passes away with treatment. In these cases a thyroid murmur is not present; true exophthalmic goitre, however, has been described.

Diagnosis.—Chlorosis must be distinguished from the pseudo-chlorosis of tuberculosis, neoplasms, syphilis, rickets, gastro-intestinal disease, intoxications, parasites, and from the other essential anæmiæ. The appearance of the patient, the examination of the organs and their functions and of the excretions, together with the results of the blood-examination as contrasted with the blood-conditions detailed under symptomatic anæmiæ, usually lead to a diagnosis.

Prognosis and Treatment.—Considered as a condition of defective hæmatogenesis, it is obvious that, although some of the clinical causes may have been the occasion of that latent weakness becoming active, the later removal of those clinical causes will not effect a cure, and that is the most general experience. Nor is there any definite tendency to self-limitation. Nevertheless, as adjuncts to direct treatment, hygienic conditions are most important. Rest in bed is advisable in all severe cases. Massage is usually of marked value. Later an out-door life and proper exercise are necessary. A proteid diet is best for the early stages of treatment. Bunge used to insist upon the value of eggs, because of the hæmatogen (a form of hæmoglobin containing an atom of phosphorus) in the yolk, and certainly forced feeding with milk, eggs, rare meats, pure white bread, and a little of plain starches or cereals often makes rapid improvement, nor is constipation any contra-indication to such a diet. Later cooked fruits and green vegetables may be advantageously added. But these will rarely cure typical chlorosis; that requires, in addition, a blood-tonic. Iron is the one best adapted to chlorosis. The form of iron is of secondary importance; apart from mucous corrosion or ulceration, it is probably always absorbed as an organic combination. It must be insisted upon that iron is not indicated for the supply of the atoms of iron needed in the hæmoglobin molecule; the iron in ordinary diet is more than sufficient for that. There is less than a drachm of iron in the adult body; and although the amount absorbed is very minute, it is vastly in excess of the amount required for the hæmoglobin molecule. The truth is that, like arsenic and to a lesser degree mercury, iron acts in some unknown way as a hæmatopoietic stimulant. From three to ten grains (calculation for adults) of any non-irritating organic or inorganic combination should be the initial dose, and this should be rapidly pushed until marked improvement is effected, unless contra-indicated by signs of ill effect. Gastric symptoms rarely contra-indicate iron. In some cases hypodermic injections are necessary; the best preparation is the ferrous manganese citrate, from three to six grains (Da Costa). In a few cases iron is not tolerated in the doses mentioned, and must then be administered in minute doses or in natural waters. Usually the cell-regeneration is more rapid than the hæmoglobin-regeneration. Arsenic is of value in nearly all cases, and may be used alone when iron is not tolerated. It, too, should be pushed. Minute doses of mercury (one-one-hundred-and-twentieth of a grain of corrosive chloride) act distinctly as a blood-tonic (Pepper); sulphur, copper, and silver have been recommended. Marked constipation calls for the mildest effective laxative, usually salines or the mild vegetable laxatives. Cases with œdema do well upon a dry diet with diaphoresis. The now popular use of intestinal antiseptics is not based upon any therapeutic indications, but upon the disproved theory that chlorosis is a copræmia, and has not proved a success. The same is true of hydrotherapy, except for bathing as a hygienic measure. Recently small repeated venesections have been extolled; one theory for their action is that hemorrhage causes lymphagosis which is followed by rapid blood-regeneration; another is that they relieve a plethora vera. The results have not been harmonious, and I have no experience with the treatment; but I have not been able to confirm the statement that venesection is followed by a lymphagogic augmentation of the blood-volume, nor has it, on the contrary, been shown that a relative plethora exists in chlorosis, although a polyplasmia seems to have been shown. Chlorotics seem predisposed to tuberculosis, a fact which must be borne in mind during the treatment. In some cases cod-liver oil and iodine preparations appear to be of marked benefit. Treatment must be continued long after restoration of the blood to the normal.

PERNICIOUS ANÆMIA.

An essential anæmia of toxic origin, whose chief pathologic condition is excessive hæmolysis, the results of which form with toxæmia the basis of its most distinctive signs and symptoms.

Etiology.—The study of the blood and tissues in cases of pernicious anæmia and of the blood and tissues in experimental anæmiæ of various kinds has made the fundamental pathological processes so clear that pernicious anæmia ought now to be classed as an intoxication. Anæmiæ may be produced in animals by iron-starvation, carbohydrate-starvation, proteid-starvation, sepsis, organic mutilation, etc.; but none of these resembles pernicious anæmia. The anæmiæ, however, which can be readily produced by the use of various hæmolytic substances are so strikingly like pernicious anæmia in symptoms, signs, and pathological conditions that it is made quite certain that we are dealing with a disease of toxic hæmolysis. Every circumstance which has been held to point to the theory that it is a condition of defective hæmatogenesis—splenification of the marrow, the appearance in the circulating blood of nucleated and juvenile erythrocytes, and their deformation—has been seen in severe secondary anæmia and in the experimental hæmolytic anæmiæ, but cannot be regularly reproduced in experiments at defective hæmatogenesis. The nucleated red cells can and do escape from the bone-marrow physiologically, nor can it be longer held that an excess of them in the marrow is evidence of defective hæmatogenesis because of their not having extruded their nuclei, since it has been shown that there is no evidence that they normally lose their nuclei by extrusion. On the one side are the evidences in the urine, blood, and tissues of increased hæmolysis; on the other side the evidences of a compensatory hypertrophy in the blood-making organs,—splenification of marrow, excess of nucleated red cells in marrow and in the blood and active cell-division in them, excessive karyokinesis in lymphatic glands, and an excess of lymphocytes in the blood,—conditions which resemble those in the blood and blood-tissues in the new-born. That these alterations, which I have termed a compensatory hæmatopoietic hypertrophy, are absent from some cases of pernicious anæmia only strengthens the belief in a toxic hæmolysis; and, lastly, there are other evidences of toxæmia which are not found in simple anæmia nor in chlorosis. Pernicious anæmia occurs most often from the thirtieth to the fiftieth year. There are on record a considerable number before the age of sixteen years, the youngest at six months; it seems more severe in children. It seems equally divided between the sexes. The majority of cases occur in poorly nourished subjects living under poor hygienic conditions; there is often a syphilitic history. The majority of the cases in women have followed pregnancy and lactation, but only a small minority of the severe anæmiæ in pregnant or nursing women are true pernicious anæmia. A great many cases have a history of neurotic constitution or nervous shock, but causal relations have not been made out as a matter of fact nor reasoned out as a matter of theory, since, were it admitted that sympathetic irritation might alter the functions of the spleen, we should still be no nearer to pernicious anæmia. Parasitic anæmiæ—ankylostomum and bothriocephalus, at least—appear to be toxic and not hemorrhagic; the pathogenesis seems quite analogous to that in pernicious anæmia. Gastro-intestinal disease has often seemed to antedate it, and post-mortem lesions are constant, yet gastro-intestinal disease in the ordinary sense does not cause anything like pernicious anæmia; there is, however, considerable evidence that the alimentary tract is the origin of the unknown poison of whose hæmolysis pernicious anæmia is the result. The fact that the disease is especially prevalent in certain places and at certain times has led to the suggestion of an infectious origin. A number of cases have followed infectious diseases. A large number of bacteria have been described in the blood and the alimentary tract, but for none of them has any definite relation been established, nor are there any positive indications that the poison is bacterial rather than metabolic. Season seems of no influence, except that ameliorated cases are liable to relapse in the spring.

Pathology.—The color of the *skin* is usually a deep yellow, not always marked; the appearance may be seen in very severe secondary anæmiæ. The subcutaneous tissues are usually similarly stained; the visceral fat, the fasciæ, and even the muscles may have the same tinge. The pigmentation has not been thoroughly studied; it is probably a deposition in altered shape of the hæmoglobin set free by the destruction of the cells. Fat is abundant, both superficially and internally. Fatty infiltration and degeneration affect all tissues liable to them, and also those least liable, as peripheral or involuntary muscles in the diaphragm or intestines. This is probably of toxic origin, not due to suboxidation, the O-income and CO_2-output having been repeatedly found normal. There are rare cases with extreme emaciation. Cutaneous eruptions are rare, as is pigmentation. Œdema is quite constantly present: early in the case it is usually transitory and may be marked about the face; later it may become general and invade the serous cavities. The muscles are soft, dry, and fatty, but usually not atrophied.

The Circulatory System.—The heart may be of normal size or may be enlarged; it is always of low specific gravity. The chambers are usually dilated and contain little blood, the valves capacious. The entire organ is very fatty, usually of a clean yellow color, occasionally mottled. The arteries present hyaline and fatty changes, even into the fine arterioles, and this fact may in part explain the capillary hemorrhages. Actual endocarditis is rare, as is pericardial effusion. Thrombosis is very rare, in striking contrast to chlorosis. Hemorrhage is common,—petechiæ, ecchymoses, ecchymomata and vibices in the skin and beneath it, ecchymosis into the tissues, oozing from the mucous membranes into the retina, even into the inner ear, usually minute in quantity. It cannot be held that they are the cause of the disease, as has been assumed by some English writers; hemorrhagic anæmia presents an entirely different picture.

The Respiratory System.—Laryngeal œdema has been described. Pulmonary œdema and infarcts are very rare. Pleural effusion is not uncommon.

The Renal System.—Fatty degeneration is always present, often very marked; interstitial overgrowth is rare, as are infarcts. There is an excess of iron and pigment, and this is made comprehensible by the now well-known fact that the renal cells possess to some degree the same function as the hepatic cells in relation to the reduction of hæmoglobin.

The Urine.—Unless influenced by ingestion of too much or too little fluid, the quantity is normal. Albumin is common, but not proportionate to the renal degeneration; so are hyaline casts. Epithelial casts and hæmaturia are rare. In some cases there is an excess of both normal and pathological urobilin. The total sulphur, and especially the ethereal sulphates, are usually in excess. Although few cases have been studied upon a constant diet, it has been shown in some cases that the nitrogen-elimination was excessive, and that the urea, uric acid, and xanthine bases all shared in the increase. Albumosuria is rare, and peptonuria has not been conclusively demonstrated. Leucine and tyrosine, lactic, diacetic, and fatty acids, and an excess of ammonia and of iron have been described. Although there is a saline excess in the blood, the urinary chlorides are normal. The phosphates are usually in excess. The toxicity of the urine has been found normal. There may be subrenal mucous hemorrhage.

The Alimentary Tract.—I know of no accurate studies of the saliva. Stomatitis is not uncommon, nor hemorrhagic gingivitis; parageusiæ are common. Marked atrophy of the gastric mucosa has been very often described, "anadenia." In some cases the mucous membrane is thin and smooth, the gastric epithelium is entirely absent and its place taken by epithelium of the common intestinal type, the muscularis is thinned, there is no fibrosis. In other cases fibrous hyperplasia is marked, the tubules are literally choked out of existence by it, the thickness of the wall is increased. Ulcers are very rare. A slight degree of dilatation is common, marked dilatation is rare. Free HCl, pepsin, and lab-ferment are much reduced, free acid often absent. The motor power is also reduced, and absorption is slow. Cancer has been several times found in the stomach of patients dead of apparently typical pernicious anæmia. Epithelial atrophy is often seen in the small intestine, also œdema of the mucosa and follicular hypertrophy; ulcerations are rare. There is a marked excess of iron in the mucosa, especially of the colon, which is evidence of an excessive activity of a normal intestinal function, the excretion of iron. The nitrogen-assimilation has usually been found quite normal, the fat-assimilation often defective. The liver may be of normal size or enlarged, has been noted atrophied, and may present a rusty color. It is usually very fatty and friable, a condition of parenchymatous degeneration without attempt at definite cirrhosis. In the middle and outer zones of the hepatic lobules there is a great excess of iron and pigment in the liver-cells and leucocytes and free between the cells. In some cases the biliary epithelium and the cells of Küpfer have contained free iron,—probably simply loaded upon them by leucocytes, and not an evidence of an attempt at a vicarious excretion.

Globuliferous and sideriferous leucocytes may be seen just outside the capillary walls, and the blood of the hepatic veins contains a disproportionate number of these. The bile is usually not in excess; it contains only the normal traces of iron phosphate. Careful studies of the quantity of leucine and tyrosine and of the pigments in the bile have not been made. The pancreas is occasionally fatty, rarely indurated or hemorrhagic, and often shows an excess of iron. That it functionates properly is indicated by the stools.

The Nervous System.—Œdema of the membranes is common, ecchymoses and pigmentation are rare. Hemorrhages into the brain and cord have been often described, and nuclear degenerations are probably caused in this way. A quite common condition is a sclerosis affecting the posterior cord, to a less degree the posterior lateral columns, and slightly and irregularly the antero-lateral and anterior columns; the cervical cord is most affected; softening of the cord is rare; the nerve-roots and the gray matter are quite normal, except as a result of hemorrhage. Degeneration and atrophy of the abdominal sympathetic ganglia and of the sympathetic trunks and the plexuses of Meissner and Auerbach have been seen, also of the peripheral nerves.

Special Senses.—These concern the eyes chiefly. Retinal hemorrhages are common, usually radiating from the optic disk. Neuro-retinitis may be present, likewise yellow spots, and venous congestion with œdema. The vessels may be ectatic.

The Hæmatopoietic System.—The spleen is, as a rule, enlarged, and also some or even most of the lymph-glands. Both are hard, and may contain hemorrhages. The enlargement of the spleen is due partly to fibrous overgrowth and partly to an excess of pulp-tissue. Nucleated red cells are often present, probably brought there from the marrow and caught in the pulp. Globuliferous and sideriferous cells are in excess; the organ contains an excess of iron. The spleen has been often incriminated as the seat of the disease. It may, however, be atrophic. The lymph-glands are frequently hyperæmic, with dilatation of the lymph-channels, which contain bloody lymph. They present an excess of iron. The bone-marrow is usually "splenified,"—that is, it has returned to the embryonal state. Usually in the long bones, often in all bones, either *en masse* or interruptedly, the fat has been replaced by red marrow-tissue. Microscopically, the conditions are not identical with those in fœtal marrow. The number of nucleated red corpuscles is excessive in proportion to the marrow-cells, the nucleated red cells are of all sizes and irregular shapes, they do not stain properly, and attempts at cell-division often look more like karyolysis than karyokinesis. Siderosis is usually present, and hemorrhages may be. In a few cases these changes have been absent. They may be seen in severe secondary anæmiæ of many kinds, can be produced experimentally, and are to be looked upon as a compensatory hæmatopoietic hypertrophy, whereby the system attempts to replace the red blood-cells which are being constantly destroyed.

The Blood.—Post mortem the vessels and tissues contain little blood (the total quantity has been found diminished); the color is then, as during life, quite pale, and the clear plasma quickly separates from the cells. The specific gravity is decreased in proportion to the diminution of cells and salts, often below 1030. In a few cases I have seen hypercoagulability, but hypocoagulability is the rule; the quantity of fibrin has not been determined. The chlorides are increased, phosphates and iron decreased; there is, of course, hypalbuminosis, but less in proportion than the oligocythæmia; the dried residue is much diminished, the alkalinity is variable. Albumoses have been noted; in a number of cases I have failed to find them. The nuclein bases have been found excessive. The plasma is distinctly hæmolytic, and contains hæmoglobin. Marked oligocythæmia is the more striking feature of the disease. Usually the reduction is below 2,000,000, often below 1,200,000, sometimes below 1,000,000, and towards the fatal end the number may fall to or below 500,000. The cells may seem well colored or may be pale. Deeply stained microcytes and irregularly or poorly colored macrocytes are present in notable quantities. In many cases the average diameter is greater than normal, in other cases less. Poikilocytosis is usually present in extreme degree; not only are many of the cells very distorted, but few of the total number may have preserved the perfect outline. The red cells sometimes display active movement, especially the small poikilocytes, and tiny rod-shaped cells have been mistaken for parasites. The staining reactions are irregular; the poikilocytes and macrocytes are especially polychromatophilic; the hæmoglobin may be separated from the stroma. Nucleated corpuscles are common in all the forms of cells; thus we have normoblasts, microblasts, gigantoblasts, and poikiloblasts, no form being constantly in excess. Often the nucleus is round and stains almost solidly, the location may be central or peripheral; in other cases, particularly in large nuclei, chromatic figures are well stained; budded and double nuclei and distorted forms are not rare; true karyokinetic figures are not uncommon, and there are occasional nuclei with karyolysis (hypochromatosis). "Crises" of simple and of nucleated cells have been described; shadow cells occur occasionally. The hæmoglobin is proportionately diminished. It has often been described as in comparative excess,—that is, a high "globular richness." The range of error with the clinical hæmoglobinometer is fully ten per cent.; the counts of the cells are especially inaccurate in this disease; there may be, furthermore, hæmoglobinæmia, all factors which must be estimated, and in the cases I have seen in which the relations were carefully studied (eight in number) no comparative excess of hæmoglobin beyond the range of errors could be established. As mentioned, methæmoglobin may be present in the plasma. Little can be said of the plaques, except that the French school contends that they are always reduced, and that this constitutes the origin of the disease, since they assume the plaque to have been the progenitor of the red cell. Protoplasmic granules may be seen free in the plasma, and cell-detritus is not rare,—whether of extra-vascular origin, however, is difficult to determine. Numerous fungoid bodies have been described, probably of accidental presence. The leucocytes are usually normal or subnormal in number; leucocytosis has been considered a favorable sign; an agonal leucocytosis is common, and may be of extreme degree. Generally the percentage of lymphocytes is increased, and myelocytes may be present. The leucocytes have been described as microcytic. There seems to be no doubt that the condition has been converted into a leukæmia, and *vice versa*.

Symptoms and Complications.—The onset is usually gradual, but may be rapid and terminate in a few weeks. The patient becomes gradually weaker, the pallor grows more marked, the inability and disinclination to work are very pronounced, and the several symptoms of the different systems are then added to produce a general clinical picture which varies considerably in different cases. The complexion often has an icteroid hue; the patient complains of a dry, irritable skin, though excessive perspiration is not rare. The agonal perspiration has been noted as cadaverous. The nutrition of the hair and nails may be impaired. Petechiæ may often be seen, eruptions and pigmentation rarely. The muscles or bones may be tender on pressure. Fever is a quite constant symptom, indicating an intoxication; it is usually not high and is irregular; subnormal temperature has been rarely noted; the agonal temperature may be very high or very low.

Alimentary Symptoms.—The tongue may be densely coated; the breath is often fetid; simple stomatitis is common, as are ecchymoses or even free bleeding. Both an excess and a deficiency of salivary secretion have been described. In some cases there is anorexia; in others bulimia or pica; in others an intense thirst. Thrush has been seen. Distress after eating, acrid and gaseous eructations, and gastric distention disturb many; nor is vomiting rare, and it may bring up blood. The gastric area may be very tender, and a sense of resistance may lead to the suspicion of neoplasm. In the majority of cases diarrhœa alternates with constipation. The diarrhœa is more often mucous than serous, and may be excessive. Flatus is often excessive; melæna is rare. The enlarged liver may be tender.

The Circulatory System.—The heart's action is usually rapid, and attacks of palpitation occur frequently. Stenocardia may be very severe; syncope may be seen in the later stages, and marked cardiac asthenia dominates some cases. The heart's action is often irregular; the pulse is quick, soft, and of low tension. On inspection the apex-beat may be seen to be a little displaced to the left, and to be very feeble, in marked contrast to the pulsations which may be seen in the neck. These are most often venous, but arterial throbbing in the neck, abdomen, and extremities may be present, and a capillary pulse is not infrequent; there may be an arterial thrill. The heart seems enlarged, due to dilatation; the sounds are often not strong, and the first sound may frequently resemble the second. A basal systolic murmur, often extending into the neck, is common; a mitral systolic murmur means, as a rule, a dilatation-regurgitation; diastolic murmurs are rare. A venous hum is usually present in the jugulars, less often in other large veins; loudest during inspiration. Peripheral arterial murmurs may be present. The hemorrhages usually occur well along in the disease in the various sites mentioned, commonly trivial in amount. Pericardial effusions rarely cause symptoms.

Respiratory Symptoms.—Dyspnœa is constant, and often extreme. It may be constant or spasmodic, or of a heavy, stertorous type. It is exaggerated by pulmonary œdema, hydrothorax, or the rare pneumonia. A slight cough is quite constant; expectoration is scanty in the absence of œdema. Hæmoptysis is very rare; epistaxis is oftener seen. Apart from the urinary signs, there are, as a rule, no *renal* symptoms. It is often noteworthy that the markedly degenerated kidneys could have functionated so long.

Symptoms of Special Senses.—Amaurosis may come on gradually or suddenly; it may be due to the organic changes mentioned, or may be accompanied by no ocular signs. Amblyopia and scotoma have been noted. Partial or total deafness, anosmia, and anageusia may occur.

The Nervous System.—Headache, vertigo, tinnitus, insomnia or somnolence, paræsthesiæ, and tremors are seen in most cases. There may be mental hebetude, loss of memory, and a peculiar slowness of speech; delirium is not uncommon, wild mania is rare, agonal coma common. Convulsions may occur, and local or systemic paralysis, which in most but not all of the cases have been due to cerebral hemorrhage. A pseudo-tabes develops in many cases, the knee-jerk is diminished or lost, there are marked anæsthesia and paræsthesia of the legs and feet; there is some incoördination, the gait is altered, and there may be a pseudo girdle sensation, vesical paralysis, and even loss of the pupillary reactions. Symptoms resembling those of lateral or disseminated sclerosis have been very rarely described.

Special complications have been in rare instances the appearances of cancer and sarcoma in subjects suffering from true pernicious anæmia.

Diagnosis and Prognosis.—Pernicious anæmia may be confused with severe chlorosis and with secondary anæmiæ, especially of cancer and gastro-intestinal disease. The history of the case, the physical and clinical examinations, and the different conditions in the blood, as already described, usually allow of differential diagnosis. In rare cases of chlorosis and cancer it may be impossible, and only the subsequent course or an autopsy will complete the diagnosis. The *prognosis* is to-day almost absolutely fatal, except in the parasitic and puerperal cases. In some cases the disease may be held in abeyance for years with arsenic, but sooner or later the relapse comes, and although a second pseudo-restoration may be attained, it is uncommon and at best transient. In a few cases no relapse has occurred. The diagnosis, however, must always be rigidly scrutinized.

Treatment.—Arsenic has given the best results. It should be pushed to the point of toleration, and should signs of intoxication arise it should be withdrawn for a short time. Care should be taken to avoid arsenical neuritis. Fowler's solution is usually the least irritating. A child of ten can, as a rule, be advanced to thirty drops per day. If advisable, it may be given per rectum or hypodermatically. The use of arsenic must be continued long after any apparent recovery. Iron is of great value when, under the influence of arsenic, improvement has become established; large doses are necessary. During convalescence small doses of bichloride of mercury may prove of decided value. Bone-marrow is the most recent treatment. There is no known reason why it should do good here; it must be markedly altered during digestion, and from what has been said it seems obvious that the subject already has marrow enough of his own; yet in a certain number of cases good and even striking results have been achieved. Stengel recommends a plain jam composed of equal parts of red marrow of calf or lamb and glycerin, well mixed. Such a jam is very slightly irritating. Should diarrhœa follow, the proportion of glycerin should be reduced. As a matter of fact, it is with regret that the profession must realize that bone-marrow has not justified the hopes aroused by Fraser. The administration of spleen has also been suggested. The transfusion or injection of defibrinated blood or plasma has been proposed and done. Little of direct action can, however, be expected from them except to increase the intra-vascular circulation, which can be better accomplished by injections of a nine-tenths per cent. NaCl solution into a vein or subcutaneously in large quantities. Marked temporary relief may follow. Lavage of the stomach and colon has given brilliant results in some cases, and should be employed in all cases. It should be thorough, and following the lavage the colon should be again filled with saline solution, which will be absorbed. Further medication is confined to the digestive tract. Pepsin and HCl are usually called for, or the food may be predigested. Strychnine is often of distinct value. Intestinal antiseptics should be given a thorough trial. While they do not render the alimentary tract aseptic, under their use digestion is often improved. The diet should consist largely of proteids and simple carbohydrates. Whenever possible, feeding should be forced. Rest in bed should be the rule until the blood is well upon the road to recovery, and rest from work for months after apparently complete recovery. Massage is of great value, and should never be neglected. Throughout the treatment the blood should be regularly examined and the treatment controlled by it. "Relapses" are much more intractable than the first "attack."

LEUKÆMIA.

A disease of obscure origin, characterized by an excessive hypertrophy of lymphatic elements in the blood and tissues, with marked anæmia and symptoms of intoxication. There are acute and chronic forms, but the two seem scarcely distinct. Like chlorosis, it seems to depend somewhat on conditions of life, previous disease, poor hygiene, especially overwork, etc. (the majority of cases come from the laboring classes), but often appears independent of them; males are most often affected. While it is a disease of middle life, there is in children more leukæmia than pernicious anæmia,—in fact, one-sixth of the cases occur in children. Of the cases in children the majority are of the mixed type, but of the total number of lymphatic and acute leukæmiæ the majority are in children. In children, malaria, syphilis, rickets, tuberculosis, chronic enteritis, and the infectious diseases seem to have antedated an undue number of the cases. Cases of direct heredity have been reported (two congenital cases have been described), and traumatism has also been noticed as the apparent cause. The evident toxic nature of many symptoms has led to the view of a possible infectious process, and many microörganisms have been described, for none of which any etiological relation has been shown. There is one case of quite clear contagion, and very recently it has been stated that animals have been successfully inoculated. As before mentioned, it has interchanged with pernicious anæmia, while with Hodgkin's disease the relations seem still more intimate, and several instances of transition have been described. It has been seen following splenectomy in man, but cannot be produced by that operation in animals. The history of the study of leukæmia reveals very little of the nature of the disease and a great deal of the site of the lesions. The occurrence of alterations not only in the hæmatopoietic organs, but also in the lymphatic tissues elsewhere,—evidences of an exaggeration in the production of lymphatic cells,—and the presence in the circulating blood of marrow-cells, have been the chief reasons for the view that the conditions in the circulating blood are secondary to those in the solid organs. The contrary view is that the lesion is in the circulating leucocyte, that the increase is due partly to the abnormally protracted cell life of the leucocytes and partly to cell multiplication while in the circulation (in the broadest sense of the term); that the anatomical alterations in the hæmatopoietic and other tissues are the result of the deposition there of the excessive leucocytes of the circulating blood, and that the so-called marrow-cells are really abnormal mononuclear leucocytes. In truth, demonstration either way is difficult. As to the lymphatic tissue, there are no anatomical or physiologic criteria to distinguish resident cells from deposited cells. In answer to the statement of the organic theorist that karyokinetic figures are in excess in the lymphatic tissues, the humoral theorist can reply that until the karyokinesis is shown to be in resident cells, and not in deposited cells (for lymphatic deposition and inflammatory round-cell infiltration are conceded by all), that statement proves nothing. True karyokinesis (apart from the problematical division by granular fissure) does not exist in the circulating blood (as examined) in quantities sufficient to explain the great excess. Yet it does occur in excess of the normal, and if in defence of the organic theory it can be pleaded, in those cases where cell-division in the organs has not been found, that leucocytic production occurs paroxysmally and in local areas, why cannot the same excuse be offered for the paucity of karyokinesis in the blood, when we consider how infinitely small a portion of the circulating blood is studied? Nor can the alteration in the red cells of the marrow be used to support the organic theory, since the same changes occur in other conditions, and since the derivation of white from red or red from white cells is entirely improbable. The two chief arguments in favor of the identity as wandering marrow-cells of the myelocytes of Ehrlich are their lack of amœboid movements and the presence of neutrophile granulations. As to the amœboid movements, they are, indeed, absent from marrow-cells and present, though slightly, in normal mononuclear cells. The distinction is of little avail in leukæmia, since here the non-granulated mononuclear cells have, as a rule, no amœboid movement. The neutrophile granulations of human marrow-cells and the occurrence of such cells in the circulating blood of most cases of leukæmia are arguments in favor of the organic school which have not been successfully met. The chief fault of the humoral theory is that we have no physiological or pathological analogues for the postulated delayed development and destruction of white cells; prolongation of the life of individual cells by their own disease would be unique. And, furthermore, the evidences of cell-degenerations (which follow the general types of cell-degeneration) in the leucocytes in leukæmia are usually present in excess, which disagrees with the humoral theory. It is, therefore, less because the organic theory is proved than because the humoral theory is incomprehensible that the former is generally accepted. The more recent theory that leukæmia is due to some gastro-intestinal infection, in that we have a constant absorption of some chemotactic substance which arouses an abnormal leucocytic activity, is highly suggestive. As yet, however, we know of no chemotactic leucocytosis which in any way resembles leukæmia. The analogous view that the peptones of digestion are not reconverted to coagulable proteids, but are absorbed as such and produce hyperleucocytosis, lacks physiological sanction and experimental confirmation. In no way has the use of albumoses or peptone, either in normal or in desplenized animals (who are subject to leukæmia), been able to produce anything like leukæmia. The ulterior question of the *cause* is not only obscure, but almost unstudied. Whether a condition of chronic nucleinic intoxication (Horsbaczewski), the result of an ulterior toxæmia, could account for the phenomena is at present doubtful.

Pathology.—The *skin* in leukæmia is usually of a pale yellow; in some cases this has become deepened into a dusky brown, and patches of dark pigmentation are not rare. A few cases are of alabaster whiteness, many are icteroid. Rare cases have a normal color. As a rule, the mucous membranes are shaded like the skin. Lesions in the skin and subcutaneous tissues are of two kinds,—lymphatic and coincidental. In a few cases the lymphatic deposits have been extensive, in the form either of nodules or of flat plates, hard, usually painless, and surrounded by eczematous inflammation; not rarely the depositions ulcerate. They seem to proceed from the vessels of the glands, and are most marked in the deeper layers of the chorium. A peculiar, hard œdema may surround the lesions. While such lesions occur in leukæmia, not all cases of lymphodermia perniciosa or mycosis fungoides are leukæmic; in the most typical cases I have seen the blood was normal except for an increase in the oxyphilic cells, so often seen in skin-diseases. Accidental cutaneous lesions are erythema, prurigo, eczema, acne, and a persistent furunculosis. Cutaneous ecchymoses are not rare, and may leave spots of pigmentation. The fat is less well preserved than in any primary anæmia. Apart from œdema, most subjects are distinctly emaciated. Œdema is common, and is of four types: general (anæmic); cardiac œdema; local œdema, due to venous stasis; and a hard, lymphatic œdema, due, it is held, to pressure upon the lymph-channels.

The Circulatory System.—The heart is often dilated. Moderate fatty change is common, extreme degeneration rare. Endocarditis affecting the valves of the left heart is not rare. Between the strands of muscle are lymphatic depositions of various sizes, and myocardial ecchymoses are common: the membranes may be similarly affected. The pericardium may be thickened and be distended with effusion. The great vessels often show a hyaline degeneration, and lymphatic collections may lie between the coats; the arterioles and capillaries are usually degenerated. Hemorrhages are very frequent in leukæmia, either spontaneous or excited by slight injuries; they may occur anywhere in the body.

The Respiratory System.—Lymphatic depositions occur throughout the tract, in the epiglottis, larynx, and trachea (not rarely producing obstruction), and often to a wide-spread extent in the lungs. The collections must be distinguished from tubercles, which they closely resemble. Those in the air-passages may ulcerate, those in the lungs may break down and form cavities, while in other cases pressure on a bronchial tube will produce an area of atelectasis. Epistaxis is common, hæmoptysis rare. The pleura is often thickened and studded with the lymphatic tubercles; effusion is common, and may rarely be bloody. The cervical, bronchial, or mediastinal glands may be much enlarged and press upon and displace the organs. The diaphragm may be heavily infiltrated. The O-income and CO_2-output have been found normal.

The Alimentary Tract.—Stomatitis, gingivitis, and pharyngitis are often seen, sometimes ulcerative or hemorrhagic. The lymphatic glands beneath the tongue and at its base, the tonsils, and the pharyngeal tonsils may be hugely enlarged. The salivary glands have been rarely infiltrated and enlarged. In the stomach and intestines the same lymphatic collections are noted, usually just beneath the mucosa which is elevated by them, often of large extent, and obviously enlargements of previously existing follicles and patches, though heteroplastic infiltrations occur. Ulceration is more common in the intestines than in the stomach, and the ulcers seem to be in some cases the site of terminal infection. In a few cases the lymphatic deposits in the intestinal tract have been so extensive and in the other parts of the body so sparse that the term "intestinal leukæmia" has been coined. While such a class of cases cannot yet be conceded, it must be insisted upon that the rich collections of lymphatic tissue in the intestinal tract are to be considered as physiological lymphatic tissue and not as a barrier against infection, and this applies also to the appendix. The HCl acidity and gastric ferments have been found diminished and absorption incomplete. The liver is nearly always symmetrically enlarged, often excessively, and the glands in the fissure also. Its tissue is hard and of light color, often mottled by areas of lighter tissue or pigmentation. The lymphatic deposits occur about the portal vessels, along the connective tissue, and as winding streams between the liver-cells, especially in the peripheral zone of the lobules. The collections may press upon small portal veins and obstruct them; dilated lymph-channels suggest a similar obstruction to them. Peribiliary deposits are rarer, but do occur. The liver-cells are usually fatty. In a few cases small areas of liver have seemed to have returned to the embryonal condition, with evidences of plentiful cell-division. There may be an excess of iron in the liver, in irregular distribution. Chemical studies of the liver and bile have given contradictory results; they serve only to show that the increase in xanthin bodies does not occur in the liver and that abnormal organic acids may occur there. The pancreas may be enlarged and hard, and will then present lymphatic deposits between the acini. Proteids and fats are poorly assimilated in leukæmia.

The Genito-Urinary Tract.—The kidneys are enlarged, hard, light in color. Lymphatic infiltration is most marked in the cortex; it is grouped about the tufts and the vessels, and is irregularly insinuated between the tubules. Large nodules may occur. The parenchyma is usually fatty; the connective tissue has in rare cases become amyloid. Uric acid or hemorrhagic infarcts are rare.

The Urine.—The amount is usually normal, not rarely excessive; it is hyperacid, due to acid phosphates. The urea and phosphates are usually in excess of the diet, but the proportion of urea-N to total N is diminished. The total N is in excess of the diet,—an excessive tissue-disintegration. The sulphur is in excess, especially the neutral sulphur and the ethereal sulphates. The ammonia is normal; there may be traces of fatty acid, but not lactic acid. The uric acid is markedly increased, while the amount of normal xanthin bodies is enormously exaggerated, and with these are nucleinic bases not normally excreted. Albumin is unusual; albumoses and an excess of nucleo-albumin may be present, likewise histon. Tube-casts are unusual; leucocytes in the urine should suggest, not renal infiltration, but vesical ulceration. Hæmaturia is not rare, and hæmoglobinuria has been described. The diazo-reaction may be present.

The adrenal bodies may be infiltrated and enlarged, and have caused death by rupture. Submucous collections may exist in the pelvis and bladder, and ulceration has been seen. Stone is rare. The testes, epididymes, and ovaries may be infiltrated, and also the corpora cavernosa, in several cases the cause of priapism. The peritoneum may be studded with lymphatic tubercles, and may contain a large effusion.

The Nervous System.—Lesions are not common. There may be lymphatic collections on the membranes, about the blood- and lymph-channels of the brain and cord, or small ecchymoses; in only one case were brain-tumors present. Degenerative changes in the posterior columns and also in the medulla have been described. Local muscular degenerations have been seen as the result of perineural hemorrhage.

Special Tissues.—Enlargement of the orbital lymphatic tissues may be marked. Hemorrhages and infiltration of the lens, iris, and choroid coat have been rare, but the retina is almost constantly affected. The collections about the dilated vessels may be very prominent, small vessels may be obstructed, and irregular areas of pale sclerosis and fatty degeneration follow. True hemorrhage is common. Deposits into the eyelids and tear-glands have been noted, while cataract is not rare. Deposits and hemorrhage have been described in the internal ear. The thyroid gland is not rarely enlarged, due most often to venous engorgement, rarely to lymphatic infiltration. The thymus gland is often enlarged, especially in children, and, indeed, in a few instances the first sign of leukæmia has been thymic enlargement.

The Hæmatopoietic Organs.—The lymphatic glands, in part, may be enlarged in any case of leukæmia, but it is in connection with lymphatic leukæmia that the enlargement is most marked. The superficial glands are commonly affected, and the cervical, mediastinal, axillary, and retro-peritoneal groups may constitute veritable tumors. They are usually soft, but may be hard. The color may be white, yellow, hemorrhagic, or rarely greenish. The enlargement is dependent upon cellular overgrowth; fibrosis is rarely present. The cellular excess is composed of several elements: small cells, quite like normal lymphocytes, large mononuclear lymphatic cells, cells with horseshoe nuclei without the rich, tightly woven chromatin of the polymorphous leucocytes, small and large cells with small central nuclei and large protoplasm, probably hyperplastic endothelial cells, oxyphilic and basophilic cells. Large globuliferous cells, as recently described by Musser and Sailer, are a new entity in leukæmic glands. Proliferative arteritis may occur; also small areas of softening and necrosis. The lymph-channels are patulous. There is little tendency to grow through the capsule, but the enlargements often follow the line of lymphatic currents, so that direct metastasis seems demonstrated. In mixed leukæmia the collections in the glands are apt to be more irregular in distribution and polymorphous in type than in lymphatic leukæmia; oxyphilic and basophilic granulations are also much more frequent. Karyokinesis is irregularly observed; sometimes no signs of it can be found, while again it may be abundant. Evidences of nuclear degeneration, hyperchromatosis, and hypochromatosis are often seen. Tiny hemorrhages are common. In the acute lymphatic leukæmiæ the type of cells has generally been more uniform, and usually of the lymphocytic type. It cannot be held that changes in the lymphatic glands are especially characteristic of lymphatic leukæmia; in fact, while some cases of lymphatic leukæmia show only the glandular enlargements, other cases have spleen and marrow changes in preponderance. By lymphatic leukæmia is meant one whose excess of cells is of the lymphatic type, not that it has originated in lymphatic glands necessarily, for the same tissue is in the spleen, marrow, and intestinal tract. The *spleen* is enlarged moderately in lymphatic and markedly in mixed leukæmia; in several cases it has been reported as normal. The capsule is thickened and very hard, and the fibrous trabeculæ are overgrown. The pulp itself is soft; it may be anæmic or hyperanæmic, according to which the color varies. It may become attached to other viscera, and has produced pressure-necrosis and intestinal perforation. Hemorrhagic infarcts are not rare, nor are areas of degeneration and softening; and rupture has occurred in both acute and chronic cases. The cellular excess usually corresponds to the depositions in the lymphatic glands with the addition of signs of red-cell destruction, pigmentation, globuliferous and sideriferous cells. Wandering myelocytes may be seen in either spleen or lymph-glands. Chemical studies are in accord only in the finding of xanthin bases and fatty acids in excess, and, in a few cases, of albumoses. The alterations in the *bone-marrow* are twofold. One consists in the same compensatory hyperplasia seen in pernicious anæmia. This change is constant. It occurs in the long bones especially, either alone or in association with the lymphatic change. The essential alteration is the lymphatic overgrowth known as the pyoid marrow. This consists partly of a hyperplasia of marrow-cells and of resident lymphocytes, and partly of collections of the types of cells noted in the infiltrations elsewhere in the body. The proportion of these classes varies. In some cases the myelocytes make up almost the entirety of the increase; in some the lymphocytosis is marked, in others the leucocytes are present in large numbers. Karyokinetic figures may be seen, and also degenerative changes. The capillary walls are often very fragmentary, and suggest one reason for the circulatory myelocytosis. Hemorrhage may be present, or areas of softening or necrosis. In rare cases the marrow changes have been entirely wanting so far as studied; but in no case has the study been complete enough to warrant the statement that no marrow changes existed. In several cases the marrow changes were the sole alterations noted. Here again, however, the body-tissues were not so completely studied as to warrant the diagnosis of a purely myelogenous leukæmia. Since in all probability the lesions are always threefold, —hyperplasia of resident tissue, infiltration with circulating cells, and inflammatory round-cell proliferation,—some alterations occur in the various tissues in all cases. Marked marrow changes may be present in acute cases, in lymphatic cases, and with exceptional splenic involvement. On the other hand, they may be very sparse in cases in which myelocytes were in excess in the blood. In some cases the brunt of the organic changes falls upon other than the hæmatopoietic organs,—for instance, the skin or the intestinal tract. At present, therefore, the types of leukæmia are not strictly related to different organic changes, but rather to the type of lymphatic cells most notably involved, it being clear that, apart from the myelocytes, the origin of the various lymphatic cells is not restricted to certain organs, they are probably derived from lymphatic tissue universally. In the marrow, spleen, and lymphatic glands various cocci, bacilli, and coccidial structures have been described. At present the evidence for them is not strong. In these same organs the Charcot-Leyden crystals may sometimes be seen in excess post mortem. They are not peculiar to leukæmia, but have been held to indicate medullary involvement. Osteomalacia and osteosclerosis have been rarely observed.

The Blood.—This is usually a pale pink. In none of the cases I have seen, and in several of which the ratio was higher than one to four, were the often-described puriform or chocolate appearances present. The pale color of the clots testifies to the leucocytic excess. Coagulation may be slow and incomplete; but which factor of coagulation may be at fault is not known. It does not seem likely that it is due to albumoses. The specific gravity is reduced, but never so low as in pernicious anæmia. The alkalinity is generally stated to be diminished. In two cases (one just before death and one in the full swing of the disease), in which I estimated the reaction by von Limbeck's method, the alkalinity was normal. There is, however, no doubt that the basic capacity is increased, and various fatty acids have been isolated from the plasma. The presence of peptone has never been demonstrated; albumosæmia is uncertain. The proportions and the quantities of the plasma proteids have not been well studied. Uric acid, the xanthin bodies, and leucine have often been found in excess; also lecithin and nucleinic phosphoric acid. The chlorides are in excess, due to the sodium salt; the phosphates and sulphur are in excess; potassium is diminished; glycogen may be in excess, both in the cells and free in the plasma. Fat may also be in excess. Oligocythæmia is the rule: in advanced cases generally between 2,500,000 and 4,000,000; in severe cases below 1,000,000. The cells are usually pale, and present a varying degree of poikilocytosis and of polychromatophilia. Nucleated erythrocytes, although rare or perhaps absent in the purest lymphatic and in the acute cases, in the mixed type are often present in numbers in excess of those in pernicious anæmia. Normoblasts are most common, but in my experience microblasts, poikiloblasts, and gigantoblasts have been as frequent as in pernicious anæmia. While they probably cannot be held to indicate that the myelogenous elements of the marrow are especially involved, they do indicate secondary changes with the production of a red marrow. Amœboid movements have been seen in the red cells. Karyokinetic figures are rare, but nuclear degenerations are common. The hæmoglobin is usually much below the percentage of cells; the lower the cell-count the higher the color-index, as a rule. Our knowledge of the plaques is as incomplete as are our methods of studying them.

Leucocytes.—Nearly all cases not under treatment have over 100,000 per cubic millimetre; from 150,000 to 300,000 is the usual number. Not uncommonly the number may rise to 700,000; above that figure the cases are rare, but 1,400,000 have been noted. The average proportion varies from one to twenty to one to four; rarely the white cells may equal or exceed the red. The number varies; daily variations of 50,000 in the finger-blood are not rare. Intercurrent diseases, especially infectious processes, may markedly reduce the number, but, as in the cases improved by treatment, the qualitative changes persist. According to the blood, there are two types of leukæmia,—the lymphatic and the mixed or leucocytic. Either of these may be acute, but the majority of acute cases are of the lymphatic type. In the lymphatic type the increase consists largely of lymphocytes, which are by no means of normal appearance, but vary very much in size, in the relations of nucleus to protoplasm, and in the appearance of the chromatin; they are always ungranulated. In acute leukæmia the large and aberrant lymphatic forms are usually in marked excess. Karyokinetic figures are rare, and degenerations are less common than in the mixed type. Lymphæmia is commonly connected with a preponderance of involvement of the lymph-glands, but need not be. Granulations of normal type are rare,—that is, present in normal numbers. Mast-cells and myelocytes are rare, often absent. In mixed leukæmia the chief characteristics are the irregularity of increase and the polymorphous character of the various cells. In rare cases the relative percentages of the various cells remain within the normal limits. In most cases the lymphocytes are markedly reduced in percentage (their number may remain normal, but is more often excessive), while the excess consists of the larger cells. In some cases the mononuclear and transitional leucocytes are disproportionately increased, in other cases the polymorphous leucocytes constitute the bulk of the excess, while in other cases the myelocytes may be the excessive factor. In rare cases the myelocytes are absent; usually they form from fifteen to thirty-five per cent of the total white cells, in some cases more than half. Here, again, polymorphism is the distinguishing feature. In the same case the relative proportions of the different cells may shift widely from time to time. The polymorphous cells are of varying size; their nuclei are of all sizes and shapes and have partly a tight and partly a loose chromatin net-work; their neutrophilic granules may be sparse or irregularly clumped, their amœboid activity below normal. The mononuclear and transitional leucocytes are likewise irregular in their morphological attributes; in leukæmia they have less amœboid activity than normally. The myelocytes vary from seven to thirty micro-millimetres in diameter, their nuclei are quite large and usually concentrically placed, the chromatin is diffuse and faintly stained, and the protoplasm contains neutrophilic granulations; a few contain typical oxyphilous granulations. Oxyphilic cells may be in marked excess up to ten per cent., or they may be present only in the normal number; they point to no tissue of origin, but are very irregular in form. The fine basophilic granulations may be increased; quite often some granulations are amphoteric. Mast-cells occur notably in some cases and are absent in others. The neutrophilic granulations may be quite oxyphilic. Degenerations are common, and may reach ten per cent. of the total: they consist of hypochromatosis, hyperchromatosis, and vacuolization, with loss of nuclear membrane and dispersion of the chromatin, and extrusion of the nuclei. Fatty degeneration has also been described, and some cells contain pigment and cell-remains.

Symptoms and Complications.—*Acute leukæmia* occurs most often in children. It begins with general pains, vertigo, headache, and feelings of extreme weakness and malaise. A chill, abdominal pain, or a hemorrhage may be the first symptom. The fever is irregular and often high. The lymph-glands and spleen swell rapidly, but not to a marked extent, and are tender. There is ulceration in the mouth and in the intestines; diarrhœa is frequent. The hemorrhages are both visceral and superficial, may be very extensive, and in the skin come out in rapid crops. The patients are markedly toxic, and usually die within a fortnight, though some cases ameliorate after the first severe onset and last a couple of months. The blood shows a moderate leukæmia; the excess of cells consists of small and large non-granulated mononuclear cells,—all classed as lymphocytes by some, but this is denied by others. Myelocytes and granules are rare. In some cases the white cells in the blood fall to normal before death. It is obvious that the subject needs study. Some of the cases seem rather to have been purpuræ, while others have probably been acute exacerbations in chronic leukæmia.

Chronic Leukæmia.—The onset is generally gradual, but acute onset is not rare. Pallor, pain in the left hypochondrium, dyspnœa, glandular enlargements, and circulatory symptoms are usually the first symptoms. Hemorrhage, priapism, or fever may be the initial symptom.

General Symptoms.—The asthenia, the changes in the mental condition, and the symptoms of intoxication are not so marked as in pernicious anæmia. Œdema may be excessive. Fever is quite constant, usually irregular, and the termination of an undiagnosed case may simulate an acute infection. Attacks of excessive perspiration are common.

Circulatory Symptoms.—The heart's action is rapid, easily excited, perhaps irregular. Palpitation is common, anginoid attacks are not rare. The pulse is soft; a capillary pulse may be present. Jugular pulsation is common, arterial pulsation much less so. The heart is not often noticeably enlarged; it may be displaced by splenic, aortic, or mediastinal pressure. A systolic murmur often exists at the base, rarely at the apex; it may be continued into the larger arteries; the venous hum is common. The hemorrhages come on spontaneously, as a rule.

Genito-Urinary.—Symptoms are rare; the most marked renal degeneration and infiltration cause uræmic symptoms rarely. Priapism has been referred to. In connection with parturition, it need only be stated here that while pregnancy intensifies the disease, the babies of leukæmic women seem healthy at birth, but do not do well upon the breast-milk, in which fact there is nothing remarkable. Menstruation is less influenced than in chlorosis, and excessive more common than deficient menstruation.

Respiratory.—Trying symptoms in some cases are produced by the laryngeal alterations, and partial or complete aphonia, which may be due to local infiltration or to nerve-pressure, may be present. Stenosis may require tracheotomy. Dyspnœa is constant; there is usually a slight cough, with little expectoration. The pressure of enlarged glands upon bronchial branches may accentuate the dyspnœa and give the physical signs of bronchial stenosis, and some of the strange spasms of dyspnœa may be due to pressure upon the pulmonary nerves. While substernal dulness is common, sternal projection from mediastinal pressure is rare. Frank pneumonia is rare; a low type without tendency to crisis is common, and may be the agonal complication. Marked œdema may occur, or atelectasis, abscess, or gangrene. Pleurisy is rare; hydrothorax common.

Alimentary.—Ulcerative stomatitis and tonsillar enlargement are often distressing. The appetite may be preserved, but is oftener lost; thirst may be excessive. Dysphagia, due to pressure of enlarged glands, is not common. There is usually pain or discomfort after eating, with acid and gaseous eructations. Vomiting may be severe. The stomach is rarely enlarged, but is often tender. Constipation and diarrhœa often alternate; diarrhœa may be very severe. The enlargement of the liver can commonly be demonstrated, and may extend to the umbilicus; it is hard, smooth, and usually painless. Retro-peritoneal lymphatic enlargements are often palpable. Hemorrhoids may be present.

Nervous.—Headache, vertigo, neuralgias, and general body pains are common; there are rarely peripheral or visceral crises. Paralysis may be due to central hemorrhages or central or peripheral pressure. Pseudo-tabes is not seen, as in pernicious anæmia. Marked mental symptoms are unusual; depression is a natural condition, but mania is rare; coma may close the case.

Special Senses.—Weakness of vision is common; partial blindness less so; sudden amaurosis rare. Exophthalmos is present in cases of retro-orbital infiltration, and may be very painful; tinnitus is common, deafness rare. The nares may be narrowed by the infiltrations.

Blood-Organs.—There may be tenderness on pressure over the bones, or spontaneous pains in them. This does not indicate medullary involvement, but rather periosteal infiltration or pressure (especially in the case of the sternum) upon underlying tissues. The spleen is enlarged early in the mixed type; it is usually below the umbilicus (it may be movable), and may descend to the pubis and into the right iliac fossa. There can be no doubt that it embarrasses the diaphragmatic movements. It is hard, smooth, may be tender, and not only distresses the patient with the sense of weight and distention, but may pain acutely. On palpation a friction sound may be elicited, and on auscultation soft systolic murmurs. The lymph-glands are markedly enlarged in most cases of the lymphatic type and irregularly in all cases; there is usually little local reaction around them. The inguinal, axillary, cervical, mediastinal, hepatic, and retro-peritoneal sets are regularly enlarged, and may form veritable tumors (usually not tender and slightly painful); such enlargements cause pressure, and produce such symptoms as venous congestion, dyspnœa, atelectasis, aphonia, dysphagia, jaundice, and chronic intestinal obstruction. Usually the glands in the neck are the first to enlarge; this may antedate blood-changes.

Diagnosis and Prognosis.—In children the difficulties attend anæmia infantum pseudo-leukæmia, Hodgkin's disease, and the severe secondary anæmiæ with splenic enlargement. The blood-examination will nearly always make the diagnosis. The lymphocytosis on the one hand and the polymorphism of the white cells on the other hand are conditions not approximated by them; it must be an anomalous case of leukæmia which cannot be diagnosed from the blood. The ratio of the cells is of no value in differential diagnosis in the cases where confusion exists. Nor is eosinophilia suggestive of leukæmia; mast-cells are. As stated, the particular organic disease cannot certainly be postulated from blood-examination. Perhaps urinary study might be of assistance. It is often not possible to exclude Hodgkin's disease. Leukæmia, up to date, has probably always been a fatal condition. Marked remissions extending over years may occur, either spontaneously or following intercurrent diseases; but of absolute cure, with complete disappearance of blood, organic, and urinary alterations, there are no reliable records.

Treatment.—The treatment is practically that of pernicious anæmia. Arsenic alone has proved of value. Under its prolonged use in large doses the leucocytes may be much reduced and the red cells increased even to normal and kept there for some time, but only rarely with much diminution of the enlarged lymphatic tissues. Iron may be used with it. Bone-marrow, spleen, and lymph-glands, or their extracts, have all been used, usually with negative results. These, as well as arsenic and eucalyptus, have also been employed by injections into the spleen. Inhalations of oxygen have in a few cases done good; but since there is no suboxidation, since the subjects have hæmoglobin and circulation enough to carry sufficient oxygen to the tissues, and since the atmosphere contains more oxygen than is needed, it is hard to see how it should act. Splenectomy is to be absolutely condemned as irrational in theory and fatal in practice. The gastro-intestinal tract usually needs special attention, and gastric and colonic lavage should be employed. A tight binder will lessen the discomfort of the enlarged spleen. Heat or cold to the splenic area or over enlarged glands, counter-irritation, blisters, cauterization, injections, and electricity have in some cases ameliorated local symptoms. Local pressure may demand operative interference, which should be undertaken with reserve. Since infectious diseases often reduce the leucocytosis, the use of toxines has been tested experimentally. Injections of tuberculin and streptococcus toxine will temporarily decrease the leucocytosis, but exercise no further influence. The same is true of injections of extract of spleen, and of spermin, and of the experimental aseptic suppuration which follows the injection of turpentine. It is quite clear that these various procedures simply superadd a chemotactic influence, but have no effect upon the leukæmic process.

Anæmia Infantum Pseudo-Leukæmia.—Under this title von Jaksch described what he believed was a clinical entity. It is not rare. Nearly all the cases have been seen under the fifth year. The symptoms have been those of severe anæmia without hemorrhagic tendency. The spleen has been noted as enlarged in all cases, often markedly. In some cases a few lymph-glands have been enlarged. The liver may be enlarged. The prognosis, apart from intercurrent disease, has been good. The mortality has not been over twenty per cent. The blood-changes consist in a marked oligocythæmia,—from 1,000,000 to 2,500,000,—a proportionate oligochromæmia, and a marked leucocytosis. The red cells are often much altered, —microcytes, macrocytes, poikilocytes, and polychromatophilic cells, in degrees varying with the oligocythæmia, together with large numbers of all sorts of nucleated erythrocytes, sometimes in cell division. The leucocytosis ranges from 50,000 to 100,000. It can often not be distinguished from a septic leucocytosis; in other cases there is a larger proportion of mononuclear cells, and perhaps a notable polymorphism. The oxyphilic cells are not regularly affected; myelocytes are rare. In the cases which have come to careful post-mortem study the splenic and lymphatic enlargements have been described as a simple hyperplasia. The liver has been occasionally described as reverted to the embryonic state. The bone-marrow has presented the changes commonly seen in the secondary anæmiæ of infants. It is thus apparent that the distinctive conditions are difficult to fix. Not only von Jaksch, but Italian writers, who have termed it an infective splenic anæmia, and the Hayem school, consider it an independent disease. A perusal of their several descriptions, however, will show that they are far from being identical. For von Jaksch it was quite a benign disease, for Luzet a very fatal one, and often becoming true leukæmia. The studies and literature upon the subject warrant the following statement. It cannot as yet be considered as a distinct disease. Some cases have been true leukæmia, some pernicious anæmia, and perhaps splenic pseudo-leukæmia. Perhaps the major portion have been cases of anæmia gravis cum leucocytose, secondary to rickets, syphilis, alimentary disease, or tuberculosis, and modified by the peculiar blood-constitution of infants. With the exception of Luzet, the condition is regarded as amenable to treatment. Proper diet and hygiene, with arsenic, cod-liver oil, and perhaps iron, iodine, or phosphorus, usually cause a return of the red cells and hæmoglobin to the normal, with the disappearance of the leucocytosis, and also of the splenic enlargement.

PSEUDO-LEUKÆMIA.

Hodgkin's disease does not as yet admit of a definition or a classification. The chief conditions are a progressive anæmia with hypertrophy of lymphatic tissues without hypertrophy of the circulating leucocytes. It occurs most often in males between the ages of fifteen and forty years, but is not unusual in childhood. Infectious diseases—syphilis, tuberculosis, rachitis, enteritis, and local septic conditions—have been held to cause it. As a matter of fact, it is at present a symptom-complex. If cases of syphilis with anæmia, leprosy, neoplasmic metastasis, and chronic infective induration were excluded, the number of cases would be notably diminished. Whether any cases would remain were tuberculosis and sarcoma excluded is a doubtful question. On the one hand it has been claimed that the disease is only a peculiar general lymphatic tuberculosis; on the other hand, all cases have been classed as lymphosarcoma. Competent authors, however, contend for cases of general lymphadenoma after all the above have been excluded. The best writers of the French school consider leukæmia and pseudo-leukæmia as practically the same pathological condition, with the difference that in the one the circulating blood is flooded with white corpuscles. Thus they refer to them respectively as leukæmic and aleukæmic lymphadenoma, and believe that there are intermediate stages leading up from the one to the other. It must be insisted upon that, to justify a diagnosis of pseudo-leukæmia, other tissues outside of the lymphatics of the neck must be affected: thus lymphosarcoma colli and the chronic cervical adenitides of children ought to be excluded. And similarly it is probable that an anæmia in which the spleen is the only lymphatic tissue involved ought not to be termed pseudo-leukæmic. Nor has the existence of an exclusively myelogenous pseudo-leukæmia been demonstrated. Furthermore, it must be diagnosed from true leukæmia, with which it is closely allied and undoubtedly interchangeable. The relationship is founded upon the anatomical resemblances, which are not identical. No valid reasons have been demonstrated in favor of their identity, why in Hodgkin's disease the white cells are not poured into the circulation and why it so rarely becomes leukæmia. Furthermore, the excess in uric acid and the nucleinic basis has not been found in pseudo-leukæmia, which weighs greatly against the theory of identity. Another consideration is the absence of tubercle bacilli, which have been found when sought for in a majority of cases of well-studied typical Hodgkin's disease. The histological resemblances are less weighty when we consider that it is often impossible histologically to distinguish syphilis, tuberculosis, sarcoma, and chronic inflammation of lymph-glands occurring as purely local lesions. It is thus apparent that the theory of identity is unproved. A large number of parasites have been described as associated with the condition. Of these, only the tubercle bacillus and the pyogenic bacteria deserve mention. The tubercle bacillus has been found when sought in perhaps a majority of the well-studied cases.

Pathology.—The lymphatic enlargements are usually very extensive. The glands become involved either singly or in chains, and are commonly not fused except by capsular attachment. Depending upon the relative proportions of cellular and fibrous elements, they may be hard or soft. They may have a chloromatous color, may be pigmented or hemorrhagic, and, although they do not suppurate spontaneously, may become softened or caseated. The skin and surrounding fascia are usually not affected, but may be attacked by inflammatory reaction and rarely necrose or ulcerate from pressure. The enlargements are most striking in the neck, axilla, and groin. The mediastinal, retro-peritoneal, mesenteric, and pelvic glands are usually affected to some degree. Lymphatic nodules and collections form at the site of lymphatic tissues in all parts of the body. Thus, the faucial and pharyngeal tonsils may be enlarged, nodules of moderate or even marked size may lie in the stomach or intestines, and ulcerations may be present. Nodules in the nose and in the bronchial walls are not rare, while peribronchial enlargements are common. In the liver, kidneys, heart, testicles and scrotum, ovaries, and adrenal bodies nodules are quite common; in the nervous system they are rare. Implication of the skin may occur during the course of the disease, appearances quite like those in leukæmia, but a dermal pseudo-leukæmia without glandular involvement is questionable. A general bronzed pigmentation has been twice described. Rarely there are nodules in the organs of special senses, in the breast, and in the nerves; thyroid and thymus implication has been more frequent. The spleen is enlarged in the large majority of cases, though not to the extent seen in leukæmia. It may be hard or soft, is often mottled, and may present distinct lymphatic tumors. It may be attached to surrounding tissues. The bone-marrow is often involved apart from the condition of red marrow (lymphadenia ossium?), usually not extensively,—a lymphatic overgrowth. The periosteum may be affected. In rare cases the internal tissues alone have been affected. The histological appearances of all seem identical. There is a marked hypertrophy of the lymphoid tissue, and also of the connective tissue. The cells, however, are not all of one type: there are some large endothelial-like cells, others of large size with polymorphous nuclei, others very large with several nuclei (giant cells?). The large mass consists of lymphatic cells of common type; in some cases a marked polymorphism has been noted. Isolated nodular collections may occur, especially in the spleen, and nests may be seen which progress through fibrous tissue like malignant neoplasms. Hemorrhages are not common. Amyloid disease may be present; more or less fatty degeneration of the various tissues is usual. The skin is of a pale yellow or white color. There may be ecchymoses or patches of pigmentation or rare accidental eruptions. Emaciation is the rule. Subcutaneous œdema is common, either general or due to venous pressure. There may be effusions into the serous cavities, or marked hemorrhages into them or the tissues, or from the mucous membranes.

The *blood* exhibits the changes of simple anæmia. In ordinary cases the red cells are reduced to below 4,000,000; in severe cases they may fall to below 2,000,000, when irregularities and nucleated cells occur, but are not marked. The hæmoglobin is usually a little lower than the cells. The number of white corpuscles, apart from complications, is usually normal; in some cases a leucocytosis of the common type persists throughout the disease; in most cases there are no qualitative changes; in a few cases the lymphocytes and large mononuclear cells have been increased, in others the oxyphilic cells (which have been found in excess in the glands). A terminal leucocytosis, due to an increase in the polymorphous cells, is quite common. The coagulability has often been diminished. As stated, transition to leukæmia has been seen.

Symptoms and Complications.—The onset is usually chronic. Acute cases with marked hemorrhagic symptoms have been described, which with fever, moderate glandular enlargement, and symptoms of intoxication proceed to an early fatal termination. Without denying the occurrence of acute pseudo-leukæmia, it must be obvious that septic and purpuric diseases have usually not been excluded from the diagnosis. The apparent beginning may be in any of the lymphatic tissues mentioned as affected, most often in the neck (where it may follow an angina or an inflammation), then in the inguinal or axillary glands, spleen, bones, or gastro-intestinal tract. In some cases general symptoms—malaise, fever, wasting, weakness—first appear; in other cases the enlargements of the glands precede the subjective symptoms, while in still other cases internal symptoms of pressure may be the first evidences of the disease. The glandular enlargements may distort the patient's appearance exceedingly. Usually they feel hard, are not fixed, not tender nor painful, though pain may radiate from them. Attachment and inflammation of the skin may occur. The enlargement may decrease or increase notably within a short time. The axillary and inguinal enlargements are often painful, due, perhaps, to the traumatism of movements; they often produce a peculiar attitude of the patient, and may press on the nerves or vessels with corresponding results. The skin is dry, petechiæ may be present, and general or local œdema. Periodical or irregular sweats may occur. Most of the other symptoms are those connected with the anæmia or the glandular enlargements.

Gastro-Intestinal Symptoms.—Stomatitis and pharyngitis are common. Dysphagia is a rare symptom. Some gastric distress is usual, but marked symptoms are rare; gastric crises have been described: hæmatemesis is rare. The gastric nodules may be palpable. The liver is often enlarged, usually not tender. Pressure of the enlarged glands may cause jaundice or ascites. Most cases have alternating attacks of constipation and diarrhœa; the latter is aggravated by ulcerations. Melæna is often seen in children.

Circulatory.—The heart's action is usually weak and rapid; anginal attacks are not rare; a basal systolic murmur is common; the jugulars in the neck may pulsate. Various arterial and venous disturbances may be produced by pressure, such as local cyanosis, œdema, even necrosis. Pericardial effusion is not common; the mediastinal growths may give marked signs. Endocarditis is very rare. Hemorrhages are less common than in leukæmia, but may occur from any tissue.

Respiratory.—Nasal and post-nasal hypertrophy may make the subject a mouth-breather. Epistaxis is more common than hæmoptysis. Laryngeal growths may modify the voice or seriously obstruct the respiration. Pressure-paralysis of the recurrent laryngeal may occur, with aphonia and the other signs thereof. The pressure upon the trachea and larynx may necessitate tracheotomy. Bronchial or pulmonary pressure may produce intense dyspnœa, cyanosis, with collapse of lung-tissue and consequent septic pneumonia, abscess, or gangrene; pleural effusion is quite common. *Renal symptoms* are usually absent, except when amyloid disease is present. Albuminuria is not uncommon; casts are rare; apart from determinations of quantity, specific gravity, uric acid, and allied bodies (which seem to have been normal), the urine has not been well studied. Obstruction of urine by pressure is very rare. The *spleen* is often painful; it is hard, and usually does not descend below the umbilicus. Pains in and along the *bones* are not uncommon; they may be tender on pressure. In women, the *menstrual* function is usually diminished. The *nervous* symptoms are largely those of pressure. Localized paralysis, areas of anæsthesia or neuralgic pains, lancinating pains, and even paraplegia, have been described. Headache and vertigo are common, as is insomnia. In many cases the glands recede and again swell within a short space of time, and in such the symptoms fluctuate notably. To the general symptoms of anæmia are added extreme asthenia, a great deal of general pain, and, in nearly all cases, an irregular fever, sometimes with rigors and sweats. The fever may be remittent, intermittent, or continuous; in some cases it rises with the involvement of new groups of glands.

Diagnosis and Prognosis.—Until pseudo-leukæmia is more accurately defined the diagnosis of atypical cases must remain a hap-hazard one, since it is from syphilis, tuberculosis, or neoplasms that it must often be distinguished. In such cases the implication of only a limited part of the lymphatic system speaks against pseudo-leukæmia, while the leucocytosis of malignant disease might be of value. In children, the question often comes up whether a condition of anæmia with enlarged spleen is a pseudo-leukæmia or a secondary anæmia (rickets, syphilis, amyloid disease, chronic gastro-enteritis). Apart from the history and other data, the limitation of the enlargements to the spleen speaks against pseudo-leukæmia. It seems as reasonable to admit a pure splenic pseudo-leukæmia as a splenic anæmia, but both are doubtful. In the cases where the onset was in the gastro-intestinal tract a diagnosis cannot be made except by the progress of the case. The *prognosis* of pseudo-leukæmia seems almost absolutely fatal; a few cures have been reported.

Treatment.—The best of hygiene, tonics, food, and the use of large doses of arsenic seem to prolong life. Mercury, iodine, ichthyol, green soap, and electricity may reduce but cannot stop the enlargements. Surgical interference for the removal of pressure is often necessary; there is, however, no evidence that extirpation of lymph-glands or spleen can cure; in the reported cases the diagnosis was more than doubtful.

Splenic Anæmia.—The view tentatively expressed in the article in this Cyclopædia (vol. iii. p. 798), that splenic anæmia exists as a clinical and pathological entity, a morbus *sui generis*, has not been borne out by subsequent studies in hæmatology. The special designation was based more upon clinical data and upon theories of the function of the spleen than upon pathological study; and Strümpell, to whom we owe the name, no longer pretends to consider it an entity, while the splénomégalie of Debove is now considered by French authors to be a precursor (!) of leukæmia or pseudo-leukæmia. The study of the severe and the secondary anæmiæ and of the spleen in various conditions has lately brought us to the conclusion that there is no physiological nor pathological basis for a purely splenic anæmia. The cases which are quite common in children are to be classed under several headings. Some are cases of atypical pernicious anæmia or leukæmia; a few are splenic pseudo-leukæmia, and the spleen is not involved alone; some are amyloid, parasitic, or malignant disease of the spleen, or other local disease attended with splenic hypertrophy; the majority are secondary anæmiæ connected with syphilis, scrofula, rickets, gastro-alimentary disease, atypical malaria, or following infectious diseases. The post-mortem findings in the spleen usually correspond to those in secondary splenic hypertrophy, and do not resemble those produced by essential blood-diseases; in other cases the conditions have been connected with some primary anæmia in which the spleen was disproportionately but not solely affected. Apart from the *rôle* of the spleen as one of the tissues of hæmolytic function, we know of no such thing as an erythrocytic anæmia of splenic origin; in lymphatic anæmiæ the spleen plays, so far as known, the same *rôle* as other lymphatic tissues, and is affected with them to a varying extent in various cases. The prognosis of the condition obviously depends upon its etiological nature. In children, splenic enlargement occurs in any and, indeed, in most anæmiæ (Luzet, Monti) of most widely varied origins. Thus, in most cases the spleen is enlarged and the blood anæmic as a result of other conditions; in the remaining cases the spleen is only one of the hæmatopoietic tissues affected, though the involvement may be very disproportionate.

Corresponding to the term splenic anæmia, the attempt has been made to set up a *myelogenous anæmia* (myeloma, lymphadenia ossium); the few cases described seem like cases of Hodgkin's disease in which the bone-marrow has been particularly involved. A strictly myelogenous anæmia, however, is both a physiological and a pathological possibility.

PURPURA.

Purpura as a cutaneous and mucous symptom appears in two forms: secondary to some well-recognized condition, and as a prominent sign of several obscure diseases termed purpuric. A comprehensive classification is that of Stengel.

(A) *Secondary purpuric conditions:*

1. Infectious: scarlatina, variola, typhoid, etc.; infectious hæmophilia of the new-born; septic purpuræ in pseudo-membranous pharyngitis, cellulitis, osteomyelitis, etc.

2. Mechanical: cardiac, atheromatous, purpuric ecchymosis in phlebectasia, mechanical embolism, etc.

3. Toxic: auto-intoxications: jaundice, pernicious anæmia, cachexia.

4. Nervous: hysteria, lesions of the brain, etc.

(B) *Primary purpuric conditions:*

1. Septic infection and intoxication: cryptogenic infection: purpura simplex, peliosis rheumatica, Henoch's purpura, purpura fulminans, purpura hæmorrhagica; scurvy.

2. Toxic: purpura toxica et medicamentosa; snake-venom; iodine; mercury, etc.

3. Mechanical: hæmophilia.

Of these only the primary conditions admit of discussion here.

Several forms of purpura appear in the new-born. The infectious hæmophilia of the new-born (Klebs) is probably a bacterial disease, although no microörganism has been connected with it. Within a week after birth the infant is attacked with hemorrhages from the mouth, nose, stomach, bowels, umbilicus, one or more, and there may be extensive cutaneous ecchymoses. The condition is acute, and seems to be quite self-limiting. If death, which occurs in one-half of the cases, and usually within the first two days, does not occur within a week, spontaneous recovery follows promptly, a fact which distinguishes it from true hæmophilia. Fever is present, the infant will not nurse, and shows symptoms of intoxication. The umbilicus is not septic, and these cases must be distinguished from pyæmia neonatorum with ecchymoses.

Morbus maculosus is a rare condition in the new-born. The onset would seem to be less violent; there is not the fever and the intoxication, nor the rapidly fatal course, nor the self-limiting character and the spontaneous recovery seen in the infectious hæmophilia of the new-born.

The subjects of syphilis hæmorrhagica neonatorum usually present no signs of disease at birth. Within the first four days hemorrhages occur from the respiratory and alimentary mucous membranes, from the umbilicus, in the skin, which may be jaundiced, and also in the viscera and internal tissues, as revealed by autopsy, which also reveals the syphilitic lesions. The condition is probably invariably fatal.

During childhood the primary purpuræ are common, especially from the third to the tenth year, after which to puberty their occurrence diminishes markedly.

In purpura simplex the purpura, as pointed out in the previous article in this Cyclopædia, is often the only symptom. There may be malaise and slight fever, diarrhœa is common, and arthritic pains are not rare. Epistaxis is the only sign of mucous ecchymosis, and it is rare. Sometimes successive crops come out, so that the case may last months; usually there is recovery within a month. No changes are known in the blood, except in some cases slight oligocythæmia and oligochromæmia. It occurs most often in children of poor constitution, living under poor conditions of hygiene, and often after infectious or debilitating diseases. It is almost never fatal.

Henoch's purpura appears to be a specific form. It begins with malaise, fever, and slight arthritic pains or gastro-intestinal disturbances. Soon the fever is high; vomiting is often marked, and may be bloody; diarrhœa is usually pronounced, and may likewise be bloody; the abdomen is distended and tender, the spleen enlarged. Arthritic symptoms are usually moderate. As a rule, the eruption does not appear until the third day. It may consist of small petechiæ or of large ecchymoses; it may be urticarial or may assume the appearance of erythema multiforme; it comes out in successive crops. The renal symptoms are often marked, and the urine then presents the signs of an acute nephritis, and may contain blood in large quantities. Both the renal and the gastro-intestinal symptoms are liable to violent exacerbations, crises. The disease is not highly fatal, yet some cases die in the first attack, while many others die in one of the many relapses which occur in most cases. The pathogenesis is unknown. The purpura fulminans of Henoch is probably not connected with this. In this condition large confluent hemorrhages occur beneath the skin, under which there is a brawny induration; mucous or visceral bleeding is not usual. Fever is often present, and may be high. The cases die in from three to five days. What relation this bears to morbus maculosus is not known.

Peliosis rheumatica is very rare under the age of puberty. Not uncommonly it occurs in epidemics, and in some places seems endemic. The arthritic symptoms are often marked; there are pains, swelling, and perhaps effusion, usually in the leg-joints. Angina is common. There are moderate fever, anorexia, and mild digestive disturbances, often slight albuminuria. Simple anæmia is rapidly produced. The skin manifestations, which usually appear with the joint-pains and affect chiefly the extensor surfaces of the extremities, are often very multiform in character. There may be simple petechia or large ecchymosis; urticaria is very common. There may be papules, which have the appearances of exudative erythema; there may be large subcutaneous nodules; and, lastly, there may be large pemphigoid formations filled with either serum or blood. Œdema is often seen, especially about the face, where it may be extreme. Internal hemorrhages rarely occur. The duration is usually not over a fortnight, but longer attacks occur, and relapses are common. Apart from septic conditions, as malignant endocarditis, this disease in children can rarely be mistaken. It resembles acute rheumatism in atypical cases only.

Purpura hæmorrhagica is uncommon before puberty, yet cases occur even in the earliest periods of life, and it has been seen directly transmitted in the new-born. It may be preceded by prodromes referred to the alimentary tract, the special senses, and the nervous system. Often it begins abruptly with a hemorrhage. This constitutes the chief sign of the disease, and in the skin may take the form of petechiæ, of large ecchymoses, or of blebs filled with blood, and there may be suppuration or necrosis of the areas. Urticaria and subcutaneous nodules may accompany the purpura. Often local pressure will provoke an ecchymosis. From the nose, mouth and pharynx, stomach and intestines, and from the kidneys and uterus, large hemorrhages may come, and any one of them may constitute the sole sign of the condition. Bleeding into the joints and into the other great serous sacs—pleura, peritoneum, pericardium—may occur, and also infarcts in the viscera, bone-marrow, and other tissues, and into the coats of the eyeball, the nerves, and the vessels; and, lastly, bleeding may occur into the central nervous organs, with corresponding symptoms. According to their severity, varying degrees of anæmia are produced, an oligocythæmia, with a more pronounced oligochromæmia, often with leucocytosis and an increased percentage of lymphocytes. The cardiac, respiratory, alimentary, and nervous symptoms of anæmia are present in degree according to its severity. Fever is common, but not invariable. The duration is a month on an average, it may be several, and relapses may occur through years. The prognosis depends very much on the occurrence and extent of internal hemorrhages. In children the diagnosis must be made from the other forms of purpura, hæmophilia, and the infectious diseases; commonly there is little difficulty. The changes in the blood have been those of marked anæmia due to the hemorrhage, with some of the alterations usually termed degenerative, together with a diminished resistance of the red cells; in several cases methæmoglobin has been noted,—all conditions which can have no etiological relations, which are entirely obscure. No alterations in the fibrin or the fibrin factors have been demonstrated, nor have bacteria been constantly associated with purpura. The arterial changes described are not constant, nor can they be held responsible. The peripheral thromboses have not been constantly found. The lymph-glands are usually enlarged, and have been noted as excessively pigmented. In a recent case ptomaines were found in the urine.

Treatment of these conditions is of little avail apart from arsenic in the simple purpura. Calcium salts are recommended, as are also nuclein substances (zymoplastic substances, Schmidt). Apart from mechanical measures, treatment serves little to check the hemorrhages,—from the sulphuric acid originally recommended by Werlhof up to the faradization of the entire skin as recently practised.

PLATE I.

Figure 1. Normal erythrocytes.

Figures 2. Poikilocytes.

Figure 3. Macrocyte.

Figure 4. Microcyte.

Figures 5. Erythrocytes with loss of hæmoglobin, as in chlorosis.

Figures 6. Normoblasts and poikiloblasts; the nuclear appearances probably do not indicate cell-division.

Figures 7, 8, and 9. Gigantoblasts; here again the nuclear changes cannot, without further evidences, be regarded as signs of cell-multiplication. They are as likely degenerative in type.

Figure 10. A free nucleus of a leucocyte, probably degenerative, may be seen in leukæmia.

Figures 11. Normal polynuclear neutrophilic leucocytes.

Figures 12. Normal non-granulated mononuclear leucocytes.

Figure 13. Normal lymphocyte.

Figure 14. Normal oxyphilic polynuclear leucocyte.

Figures 15 and 16. Myelocytes, from the blood in leukæmia, both with slight evidences of nuclear degeneration.

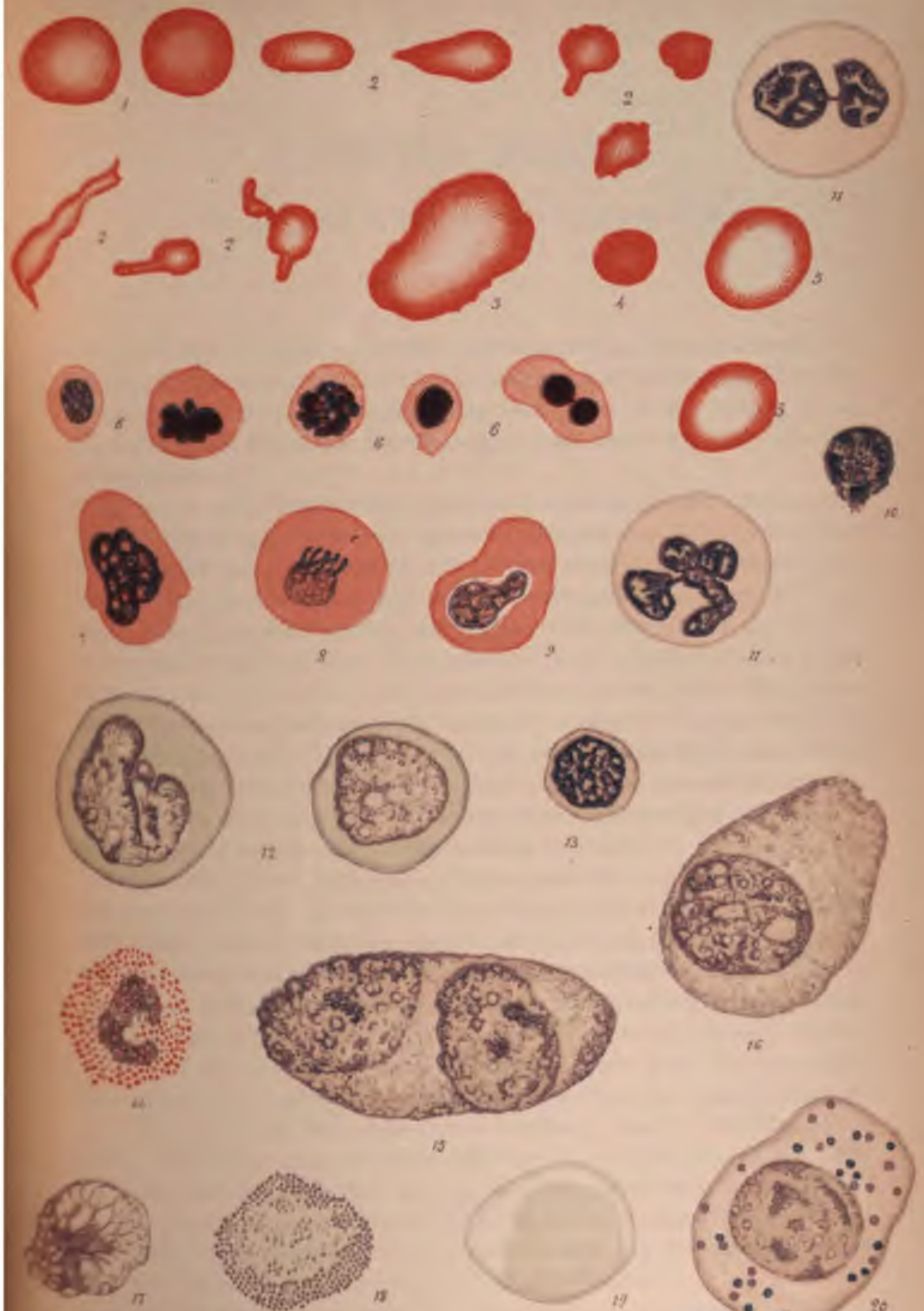
Figure 17. Nucleus in karyorrhexis, leukæmia.

Figure 20. Mononuclear mast-cell, leukæmia.

All the above fixed by heat, followed by immersion in a saturated solution of picric acid, stained by hæmatoxylon eosin, mag. 1200.

Figure 18. The myelocyte of Ehrlich with neutrophilic granulations.

Figure 19. The normal mononuclear leucocyte. Both fixed by heat, stained with the Ehrlich triple stain, mag. 1600.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ADDISON'S DISEASE.

BY ALONZO ENGLEBERT TAYLOR, M.D.

ABOUT eight hundred cases have been reported as Addison's disease, but the majority have been unconfirmed by autopsy, and in less than fifty cases has a careful microscopical examination been made of the various tissues. The pathological reports of the past eight years have been lately analyzed by von Kahlden.

Etiology and Pathology.—Despite pathological and experimental studies, the pathogenesis remains obscure. The adrenal bodies are derived from two embryological sources: the cortical substance is developed from the mesoblast, the medullary substance from the epiblast in connection with the sympathetic nervous system, and contains cells which are to be regarded as multipolar ganglion cells. Phylogenetically the relations of these bodies to the nervous system are still more definitely settled than in the human species. All vertebrates, with the exception of the acraniæ and the cystostomes, possess them. In the fishes and the amphibiæ the bodies are divided into two parts, one of which is an integral part of the sympathetic nervous system, while the other is a vascular structure, corresponding to the cortical substance in mammalia. There is much evidence that neural aplasia is connected with adrenal hypoplasia; in many carefully studied cases of hemicranius, cyclopia, and syncephalus the adrenal bodies were much smaller than normal, while in one case one adrenal body and portions of the sympathetic system were absent. Hydrocephalic monsters do not possess such anomalies. In a few cases of hypoplasia of the sexual organs these bodies have been much undersized, while in cases of infantile Addison's disease the thymus has been very small. While it is quite clear that the medullary substance is a nervous structure, the notion that the cortical substance is a blood-gland has been based almost entirely upon its vascular structure. No studies upon the blood flowing to or from these bodies have yet shown any morphological or chemical alterations other than that the venous blood is less heavily laden with CO_2 than the general venous blood,—a condition not peculiar to these bodies. Nor has it been shown that the arterial blood is more or less toxic than the venous.

In about ninety per cent. of the cases which have presented the symptoms of Addison's disease the adrenal bodies have been more or less diseased. In the vast majority of instances the changes consist in tubercular processes, collections of small round and giant cells with caseation and often calcification, and tubercle bacilli are being found in an increasing percentage of the cases. In some instances the normal tissue has been entirely obliterated; more frequently small areas of healthy tissue remain intact; in rare instances implication is very restricted. No connections have been made out between the preponderance of any one symptom and the involvement of any particular portion of the bodies. In a few instances the disease has been unilateral. Usually the organs are enlarged, but the converse may be true, and in all cases they are liable to adhere tightly to the adjacent tissues, which are commonly implicated. The capsule is thickened, its vessels may show a proliferative arteritis with areas of hyaline degeneration, and there may be round-cell infiltrations. There is no constant disturbance in the pigmentation; hemorrhages are rather uncommon. Fibrous overgrowth is always present and often markedly so, and between the strands are collections of small round cells. In many cases the ganglion cells of the medullary substance have been definitely altered, pigmentation has been excessive, the protoplasm refused to stain or stained homogeneously, the nuclei were degenerated, and there was a hyaline substance beneath the cell-wall. The normal fat is usually not present; any remaining epithelium may be fatty. The conditions in the nervous system are very important; in the majority of cases there is wide-spread implication. The semilunar ganglia in particular and the surrounding plexuses are nearly always involved. The capsule is generally thickened; there are fibrosis and round-cell infiltration in the ganglia. The ganglion cells are excessively pigmented; there are vacuolization and nuclear degeneration, with a hyaline exudate beneath the cell-wall; in whole areas these cells may have entirely disappeared. The medullary fibres are often very degenerated, and may, indeed, have entirely disappeared. The axis-cylinders are frequently knobbed, badly degenerated, and often absent. The vessel-walls are usually thickened, with hyaline degeneration. Similar changes have been seen in the cervical and thoracic ganglia, in the spinal ganglia, and even within the skull. The nerve-trunks, especially the splanchnic, are often atrophied; there is thickening of the perineurium, whose vessels are thickened and degenerated, and about which hemorrhages may be seen. The nerve-fibres and the axis-cylinders show the same degenerations seen in the ganglia. Karyokinesis is often noted in areas of deepest degeneration and about the vessels; karyorrhexis may also be seen. The degenerations may extend with the branches of the sympathetic nerves up to the cord and into the intercostal nerves. The intra-vertebral ganglia show the same changes. In the cord itself, while the degenerations have been seen in the medullary fibres and axis-cylinders, in the anterior cells, with slight arteritis and glial overgrowths, such have been very sparse, and have more often been entirely absent. It is not possible to connect the intensity of cutaneous pigmentation with sympathetic involvement; it has been absent where the sympathetic changes were present, and *vice versâ*.

The cutaneous pigmentation is probably identical with the normal pigmentation in mode of formation and in the state of deposition. It contains no iron. An excess of wandering plasma-cells is usually noted, especially about the vessels of the chorion, whose adventitia they may penetrate. In some cases the vessel-walls are hypertrophied; there may be hyaline degeneration in them, or round-cell infiltration between them. Small thromboses are not uncommon, nor are minute hemorrhages, but there is no reason for thinking that the pigment is made out of such blood-pigment *in loco.* The chorion may present free pigment. As a rule, pigmentation of the mucous membranes appears late in the case, and has been considered a sign of early termination. The conjunctivæ are quite often pigmented, and the true vocal cords have been affected. There may be striking areas wherein even the normal pigment is absent. Scleroderma, vitiligo, areas of desquamation and of local hypertrophy, spontaneous gangrene of the toes, multiple neuro-fibromata, and a peculiar blackening of the hairs have been observed. A micro-organism has been described, the cryptococcus Addisonii, but that cannot be considered seriously. There are no valid reasons for believing that the pigmentation is caused by disturbances in the adrenal bodies in the sense that through disease they have lost a pigment-destroying function. There are, however, reasons for the belief that the pigment-building cells stand under nervous control, and the theory that this function is reflexly exaggerated by adrenal or sympathetic disease is at least not improbable. It has been repeatedly claimed that the skin-pigment bears some relation to the adrenal pigment, and quite recently it has been suggested that the paucity of this pigment in the adrenal bodies of children explains the rarity of Addison's disease in them. A number of ill-studied chromogens, basic in nature, have been found in the adrenal bodies, and among them is brenzcatechin (orthodioxybenzol). This, it has been assumed, is produced in the adrenal bodies out of protocatechuic acid, and a trace of it is held to exist in the normal urine of individuals on a mixed diet, any excess being converted or destroyed by the ganglion cells of the adrenals and sympathetic ganglion. In case of increased production or of a decreased activity of the ganglion cells, the pigment is supposed to flood the body and be deposited in the skin with an accompanying intoxication. Addison's disease, however, does not occur with disproportionate frequency in vegetarians. No such metabolic activity of the ganglion cells has ever been demonstrated, nor has it been shown that the urinary brenzcatechin is derived from the adrenals, and not as an ethereal sulphate from oxidation of phenols. The fact that this substance has been found in the contents of a meningocele and in many other pathological exudations discredits the theory that the adrenal bodies produce it. The theory that adrenal disease causes an excess of taurocholic acid, which by toxic hæmolysis frees much pigment, which is then deposited in the skin, is not probable, because other undoubted toxic hæmolyses do not produce pigmentation.

Atrophy and ulceration of the gastric mucosa and the intestinal muscularis, splenification of the bone-marrow, with globuliferous leucocytes and masses of pigment and with areas of osseous softening, splenic hypertrophy, fatty nephritis, hepatic hyperæmia and fatty degeneration, the wide-spread lymphatic enlargements and fatty degenerations, bear no essential relations to the causation, and are mostly to be viewed as consequences. Nor can the gastric and intestinal atrophies explain the gastro-intestinal disturbances, since they do not always coexist.

The adrenal disease may be caused by or accompanied by tuberculosis of lungs, bones, lymphatics, intestines, and the uro-genital tract; rarely they are the only tissues affected. In many cases of generalized tuberculosis the bodies are badly involved, but with no suspicions of Addison's disease. Non-tubercular diseases of the bodies have been reported in an increasing percentage of cases; such include carcinomata and sarcomata, gummata, interstitial hemorrhagic inflammation, simple fibrosis, and simple or echinococcus cysts. In nearly five per cent. of cases clinically considered as Addison's disease the bodies have been reported as normal,—often, it must be confessed, without complete microscopical examination. In fully as high a percentage of cases, even with disease of the bodies, the semilunar ganglion and the surrounding plexuses have been intact.

Much work has been done in relation to neurin, which is held to be connected with the disease. Neurin is a basic substance,—trimethyl-vinyl-ammonium hydroxide,—a ptomaine which can be produced out of lecithin either by chemical or by bacterial action. The neurin is probably derived from cholin, and can also be derived from protagon. Neurin and neuridin have without doubt been seen in healthy adrenals and brains respectively. Thus far it has not been possible to produce neurin with unformed enzymes, nor with bacteria in the absence of oxygen. Since lecithin exists in nearly all cells, and since it has been found in all kinds of pathological tissues and fluids without its ever having become broken up and neurin formed, such formation in Addison's disease is doubtful. Neurin acts very like curare. It may be admitted as true that neurin is found in the adrenal bodies and in the urine of cases of Addison's disease, that extirpation produces symptoms like those of curare poisoning, that the blood of such animals will cause curare symptoms in others; but injections of neurin do not produce the same symptoms (apart from the muscular paralysis) as extirpation of the organs. Nevertheless, it acts much more strongly on extirpated animals than on healthy ones, although in neither case is an excess eliminated. Despite most positive assertions, it has in a number of instances been possible to keep dogs and rabbits alive indefinitely after extirpation, although the nutrition is much disturbed for a long time. In the results of different investigators nothing like Addison's disease has developed. Even admitting all the work on neurin, it still does not explain Addison's disease. Addison's view, that the bodies destroy a poison, is in the same line as are the theories that they destroy fatigue products or convert toxic metabolic proteids. The physiological theory that the adrenals or their connected ganglia contain an inhibitory centre for the intestinal peristalsis can be brought into no connection with Addison's disease. To say that it is a condition of disturbed metabolism explains nothing. Even should the pigmentation be explained by the sympathetic theory, it does not account for the obvious intoxication or the undoubted cases without pigmentation. It will not do to say in such a case that small areas of healthy tissue remained and these sufficed to keep away pigmentation, and then, in cases of the disease with pigmentation and almost healthy tissues, to say that the disease of the microscopical areas was enough to cause it. The now well-demonstrated fact that extract of the adrenal bodies is a circulatory stimulant (probably by an action upon the intra-cardiac ganglia and upon the peripheral vaso-motor system) cannot yet be used to elucidate Addison's disease, but it is very interesting in view of the cardiac asthenia in this condition.

The Urine.—Olyguria is usually present, the result of diet, but some cases have polyuria. The color is often dark. An excess of urobilin and other chromogens, the ethereal sulphates in general (including brenzcatechin), acetone, and fatty acids has been found in some cases, but not constantly. The presence of neurin and glycerin-phosphoric acid has not been demonstrated in the urine in many cases. Traces of taurocholic acid have been several times noted. Albumin is not uncommon, and casts are present according to the degree of renal degeneration.

The blood in Addison's disease has been very imperfectly studied. As a rule, the anæmia is not marked, and usually it is of the type of pseudochlorosis, although the oligochromæmia is not markedly disproportionate. Tschinkoff with the spectroscope showed in two cases that an abnormal proportion of the hæmoglobin was reduced, while in one case he reported a trace of methæmoglobin, and in some of the leucocytes there were traces of melanin. These conditions, however, cannot be used to explain the pigmentation. Very rarely have counts of red cells below 2,500,000 been recorded. Red cells have contained nuclei and degenerations and have presented amœboid motion. Leucocytosis may be present or absent in uncomplicated cases, and, with the exception of an increased percentage of lymphocytes (indeed, in two cases lymphatic leukæmia began in patients with apparent Addison's disease), there have been no qualitative changes apart from the rare presence of a myelocyte and of occasional eosinophilia. Whether the sympathetic conditions in the abdomen really produce visceral congestion through vaso-motor dilatation, as has been claimed, is not yet assured.

Symptoms.—The four cardinal sets of symptoms are the general asthenia and muscular and cardiac weakness, the gastro-intestinal disturbances, the anæmia, and the pigmentation. They do not always coexist. The anæmia in particular is, as a rule, very moderate,—in some cases scarcely noticeable,—and hæmic murmurs are uncommon. Whether or not the disease can exist without pigmentation is a mooted question. In most of such reported cases ("latent Addison's") the diagnosis was made only when the adrenal glands had been found diseased post mortem, and it is evident that advanced adrenal disease ought not to be used to make a diagnosis of Addison's disease when the clinical symptoms did not warrant it, for on the one hand adrenal tuberculosis is common without any symptoms suggesting Addison's disease, and on the other hand it requires a perusal of but a small percentage of the reported cases to realize that it is not yet as definite a clinical entity as current diagnoses would suggest. The asthenia and muscular depression (objective and subjective weakness) are usually marked, but since in moderate degree they may be present in many other chronic debilitating diseases, especially connected with tuberculosis, they cannot be depended upon too exclusively. The gastro-intestinal disturbances are much more severe and constant than are seen either in phthisis or in tuberculosis of the intestines and peritoneum, and are a sign of much value. Occasional attacks of cardialgia and distressed heart's action are probably of nervous origin. Pseudo angina pectoris has been noted. In the oft-noted instances of mania, convulsions, coma, and paralysis, uræmia has not always been eliminated. They bear no likeness to neurin poisoning, nor can they be attributed to glycerin-phosphoric acid, taurocholic acid, or acetone.

Diagnosis.—Differentiation must be made from malignant cachexia, pernicious anæmia, and tuberculosis. The pigmentation must not be confounded with the skin tints of argyria and arsenic poisoning, cyanosis, jaundice, pernicious anæmia, vagabondism, nigrities, and dirt. It must also be differentiated from the pigmentation of the menopause, tinea versicolor, chloasma, arthritis deformans, diabète bronzé, and mesenteric tuberculosis. The history, physical examination, blood, urinary, and parasitic studies will usually eliminate the doubtful pigmentation. The diagnosis from tuberculosis, essential anæmia, or marked cachexia is, without pigmentation, as a rule, not possible.

Treatment.—Much has been hoped from the use of extracts of adrenal bodies, but thus far the hope has not been fulfilled, for though in a few cases improvement has been noted, more often it has not occurred. Since the methods of treating the organs and of their administration have not yet been well studied, the question as to their value cannot now be decided. Recently a cure has been reported following operative removal of the tuberculous bodies.

ESTIMATION OF THE CORPUSCULAR RICHNESS OF THE BLOOD:

A NEW HÆMATOKRIT AND A NEW TECHNIQUE.

BY JUDSON DALAND, M.D.

MOST members of the profession have had more or less experience in the counting of blood by the hæmocytometer, and can recall many hours spent in the enumeration of the red and white blood-corpuscles; the difficulty of correctly measuring, diluting, mixing, and placing the blood on the slides and making sure that the top cover was in the proper position; the annoyance and difficulty of estimating the exact quantity of the diluted blood that should be placed upon the slide; and the frequency with which, even under the best of circumstances, their efforts have proved valueless because of using too much or too little of the diluted blood, from the presence of foreign bodies, or from want of success in placing the top cover in position. Furthermore, when all these conditions appear to have been complied with and a successful result was apparently assured, disappointment has been experienced, upon examining the blood, by finding that the corpuscles were distributed over the field unequally, or, despite all the precautions taken, that a fine fibre from the material used in cleansing and drying the slide was present, making it necessary to repeat the whole procedure. Having at last secured a perfect specimen, all are aware of the effects upon the eyes of counting each corpuscle in each of the small squares, especially as experience has shown that no estimate is of value unless sixty-four small squares are counted. The best results are obtained by counting sixty-four squares in each of two preparations. The eye-strain, nervous irritation, and fatigue induced by three or four hours of this work are such as to make the most ardent clinical observer shrink from its unnecessary repetition, particularly as the results have not been so accurate as one was at first disposed to believe. The sources of error in counting the blood-cells by the hæmocytometer are numerous, as a series of observations made by myself and Dr. Carl Sadler, of Prague, demonstrates.

The first count was made in a case of pericarditis occurring in a boy aged seventeen who was febrile, and the difference in these two counts, which were made at the same time, was 1,043,500. The second observation showed a difference of 337,000. The second case was one of tubercular peritonitis in a man aged thirty-two who was febrile. Each counted the same squares and obtained the same results, but when the count was repeated there was a difference of 150,000. The third case was one of Weil's disease occurring in a woman aged thirty, the difference between the counts in this case being 187,500, and when each counted this same blood from the same mixture the difference was 356,250. When this blood was diluted and each made preparations from the same blood, the difference was 162,500. The fourth case was one of typhoid fever occurring in a patient aged twenty-three. The average of my two counts, compared with one count made by my colleague, showed a difference of 750,000, and when this was repeated the difference was 550,000. When a larger number of squares of the same blood was counted, under the same conditions, this difference was reduced to 275,000. The fifth case was one of rheumatic polyarthritis in a man aged twenty who was afebrile, and the difference was 362,000 when but thirty-two squares were counted, but when sixty-four squares were counted it was reduced to 50,000. Case VI. occurred in a man aged twenty-three suffering from the same disease, and the difference was 313,500. Case VII. was one of pericarditis occurring in a boy aged seventeen, the difference here being 862,500. In Case VIII., occurring in a man aged forty-seven suffering from an acute pleuritic affection, who was febrile, the difference was 887,500. The foregoing observations show that when the same sixty-four squares are counted with the minutest care by two observers a difference of at least 50,000 may be expected, as was observed in Case V., and when the same slide is counted in the ordinary way a difference of 362,000 may readily occur. When from the same diluted blood two preparations are made, one immediately after the other, and counted at once by the same observer, a difference of from 187,500 to 525,000 may occur, as was noted in Case III. It is therefore fair to conclude that in practical clinical work the results obtained in counting blood-corpuscles are extremely variable,—much greater, in fact, than would be inferred from the literature on the subject. These differences do not represent the actual number of corpuscles per cubic millimetre in the cases observed, but show the different results obtained by two independent observers. It is probable that at times each observer obtained a result greater or less than the real number, under which circumstances the counts would be almost identical, as in either case one may count in excess of and the other less than the real number, so that the difference was greater than the absolute number. For example, in Case I., though my colleague's count was 4,443,500 and mine was 3,100,000, the actual number was probably 3,600,000, the difference being 500,000, whereas the difference in the two counts was 1,000,000. The two chief sources of error in the results obtained seem to be, first, the difficulty in securing a thorough mixture of the blood with the diluting fluid, so that each drop of the mixture shall contain the same number of blood-cells, and, secondly, when this drop of diluted blood is placed upon the slide, that the layer of blood to be counted shall represent exactly its share of the red blood-corpuscles. These sources of error are most difficult to overcome. The first is, in a measure, obviated by prolonged shaking of the mixture which it receives, not less than three or four minutes, but the second must always remain a variable factor. In Cases IV. and V., where the greatest difference exists, it was noted that the blood in the Thoma-Zeiss counting pipette coagulated with unusual rapidity, thus preventing uniform admixture with the diluting fluid.

The foregoing observations led me to search for another method of estimating the corpuscular richness of blood, and upon learning that Professor Blix had suggested the use of centrifugal force for this purpose at a meeting of a medical society held at Upsala, Sweden, in December, 1885, and that Hedin, following this suggestion, had constructed an instrument for this purpose, I began a series of observations with this instrument, the hæmatokrit. On pages 824–828 of the *Fortschritte der Medicin*, No. 20, October 15, 1891, in an article entitled "A Volumetric Study of the Red and White Corpuscles of Human Blood in Health and Disease by Aid of the Hæmatokrit," a series of observations is detailed, showing the influence of nineteen different solutions upon the volume of red blood-corpuscles, and the results may be summarized by the statement that a two and one-half per cent. solution of bichromate of potassium proved to be the best for the purpose.

A measured quantity of blood was drawn into a pipette, and an equal quantity of the bichromate solution added and thoroughly admixed in a watch-glass. The hæmatokrit tube was then filled with this liquid and rotated 10,000 times. An examination of fifty-five healthy males showed an average volumetric percentage of one hundred and three, and a similar examination of eight female nurses gave an average of eighty-eight per cent., which is less than the true percentage, as most of the women presented evidences of anæmia. As the count of the blood of twenty-five healthy males gave an average of 5,130,248, we may consider that 5,000,000 is the equivalent of the normal, or one hundred per cent., and therefore to convert the number of corpuscles into volumetric percentage it is necessary to remember that one percentage volume is approximately the equivalent of 50,000 red blood-cells. It must not be forgotten, however, that this is only approximately correct, and is only intended for statistical work. The hæmatokrit deals solely with the estimation of red blood-corpuscles by volume. Whenever necessary the remaining tube in the frame may be filled with blood from the same incision and a control observation be made.

When the two and one-half per cent. solution of bichromate of potassium was used as a diluting fluid, frequently in normal blood a very faint white band could be seen at the proximal extremity of the column of red blood-corpuscles which represented the white blood-corpuscles, and when leucocytosis was present it could readily be determined, provided it exceeded 30,000 per cubic millimetre. For the accurate estimation of leucocytes the blood-counter must be employed.

A prolonged series of observations was made upon the same cases to demonstrate any variability that might occur in the use of the hæmatokrit, with the result that a difference of from one to four per cent. may be expected. Forty-four patients suffering from diseases which one would ordinarily find in a hospital service of fifty beds gave results varying between seven and eight per cent., and these results agreed very closely with the enumeration which was made at the same time with the Thoma-Zeiss hæmocytometer. The greatest variation in the hæmatokritic examination, as well as in the blood-count, was observed in such conditions as rheumatism, gout, and chronic pneumonia, and in all febrile states where the coagulability of the blood is greatly increased : the loss in these conditions is probably about eight per cent.

As was pointed out in the above-mentioned article, it is evident from these observations that the following are the principal sources of error in the use of the instrument:

1. Incorrect measurement of the blood and diluting fluid.
2. The impossibility of always securing an absolute admixture of the diluting fluid and the blood.
3. Increased coagulability of the blood, as in fevers, pneumonia, and rheumatism, by which coagulæ formed before the diluting fluid was thoroughly admixed, in consequence of which the readings were uniformly lower because so many of the red blood-cells could not be removed from the pipette.
4. The loss by evaporation prior to drawing the diluting fluid into the hæmatokrit tube, tending to increase the percentage.
5. The error occasioned by the adherence of the red blood-cells to the side of the pipette employed in measuring the blood.
6. Gravitation, which quickly caused the red blood-cells to sink to the bottom, so that the blood drawn into the hæmatokrit tube is disproportionably rich in red blood-cells.
7. The occurrence of air-bubbles as the blood is mixed with the diluting fluid. Their presence in the hæmatokrit tube makes a correct observation impossible.

I also pointed out that this entire process required considerable skill in technique, and, on the whole, these were the principal sources of variable errors with which the hæmatokrit contended.

The instrument which I employed in this series of investigations was constantly getting out of order, and possessed a number of disadvantages. I therefore devised a new one, which has been frequently improved during the past six years, and in its present form is entirely satisfactory.

This instrument,[1] as will be seen in Fig. 1, is composed of a set of wheels by which the upright spindle (Fig. 1*a*) may be rotated ten thousand times per minute. The metal frame which is placed upon this spindle is shown in Fig. 2, and is so arranged that it carries two glass tubes, the outer end of each fitting into a small cup-like depression the bottom of which is covered with a thin rubber disk. Each of the inner ends of the tubes fits into a similar depression, which is so fastened to a spring (Fig. 2*a*) that it may be pressed towards the centre (Fig. 2*b*) while the tube is being introduced. As soon as the tube is in place, this spring holds it securely in position. This arrangement is extremely simple and effective. The glass tube is placed exactly between the two arms which constitute the frame, as atmospheric resistance to rapid rotation would be greatly increased if the tube projected above or below the frame (Fig. 2). The metal employed in making the frame is so thin that it cuts through the air, thus reducing to a minimum the resistance offered by the air when the frame is being rapidly rotated. The glass tube (Fig. 3) measures fifty millimetres in length with a lumen of half a millimetre, and upon it is a scale representing one hundred equal parts. Immediately above the scale the glass forms a lens arranged like a lens-front thermometer, which magnifies the column of blood, making more accurate the reading of the scale. One end

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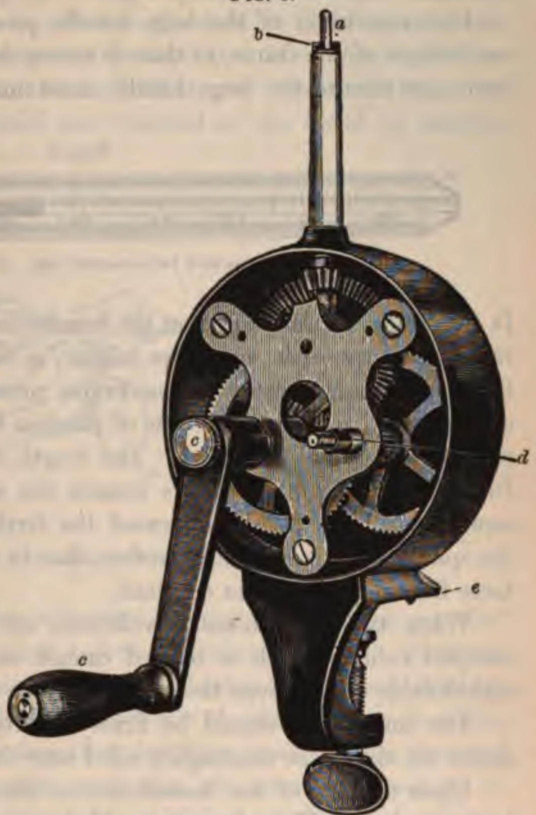
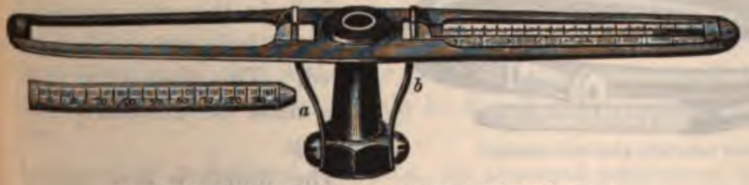
[1] Manufactured by the Bausch & Lomb Optical Company, of Rochester, New York.

FIG. 1.

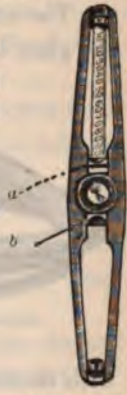


a, spindle; *b*, bayonet lock; *c*, axle and handle for blood attachment; *d*, second axle for urinary attachment; *e*, clamp for attachment to table.

FIG. 2.



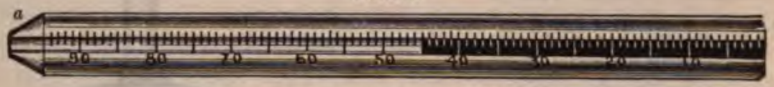
a, spring in natural position; *b*, spring pressed towards centre.



of this tube has a blunt point (Fig. 3*a*), which greatly facilitates the filling of the tube with blood. The frame is securely fastened to the spindle by a modified bayonet lock (Fig. 1*b*).

One revolution of the large handle produces one hundred and thirty revolutions of the frame, so that to secure ten thousand revolutions of the frame per minute the large handle must make seventy-seven revolutions.

FIG. 3.



Glass tube twice natural size. *a*, blunt point.

It is absolutely necessary that the handle should be moved at the uniform rate of seventy-seven turns per minute, as ten thousand revolutions of the frame per minute, under the conditions present, constitute a fixed amount of centrifugal force. If the rate of rotation be lessened the volume of blood will be larger, and *vice versa.* The length of the tubes and their distance from the spindle must always remain the same, as it is manifest that the centrifugal force will be increased the farther the blood is removed from the spindle. It is evident, therefore, that in order to obtain uniform results these factors must remain constant.

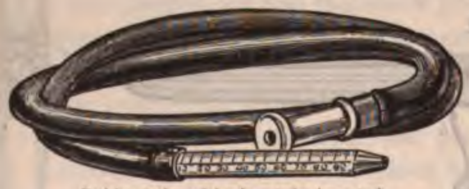
When twenty thousand revolutions are secured in two minutes, the compact volume which is formed cannot be still further reduced to any considerable extent, even though rotation be continued for several minutes.

The instrument should be firmly secured to a solid table, and when in active use should be thoroughly oiled once daily.

Upon the side of the hæmatokrit to which the handle is attached there is a second axle (Fig. 1*d*) so arranged that the handle may be removed from the first axle and placed upon the second, whereby, when the urinary attachment is placed on the spindle, a rotation of twenty-five hundred per minute may be secured.

The method of using this instrument is simple. To facilitate filling the glass tube a rubber tube is slipped over that end of the capillary pipette which is *not* blunt-pointed. To the extremity of this rubber tube is attached a mouth-piece (Fig. 4), precisely in the same manner as when the hæmocytometer is employed. This glass tube or pipette must be absolutely *clean* and *dry.*

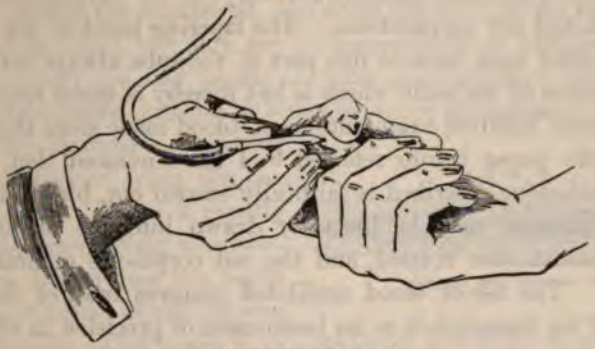
FIG. 4



Rubber tube with glass tube inserted.

The thumb is first cleansed with distilled water, then with alcohol to remove the water, and then with ether to remove the fat and any remaining water or alcohol. The skin is then antiseptically clean and dry, and any fibre from the towel should be carefully brushed away. If the patient be anæmic, the hand is allowed to hang by the side for a minute, the skin is punctured, the tube held horizontal (Fig. 5), and the blunt point brought cautiously in contact with the emerging drop of blood, when by suction the tube is filled quickly and completely with blood. The finger of the operator is at once applied to the blunt extremity of the tube, and the rubber tube is removed by a slow rotatory movement, so as to avoid the removal of the blood by suction. Immediately the capillary pipette is inserted into the frame, taking care that the distal extremity is well in place before removing the finger, otherwise the blood will escape from the tube. It is absolutely necessary that the entire procedure should be performed as quickly as possible, so as to anticipate coagulation. Immediately thereafter the frame is rotated ten thousand times per minute for two minutes, and then the percentage volume of blood may be read from the scale. The divisions of the scale are one-half millimetre apart, so that one may read this scale without difficulty. When greater accuracy is required, a small magnifying-glass may be employed. The entire procedure need not occupy more than three minutes, and requires no special or unusual skill, while the elements of error are so few that the results are trustworthy even when performed during the hurry of clinical work.

FIG. 5.



Method of filling glass tube.

Upon a former occasion I suggested a tube of seventy millimetres and a scale of two hundred, but a few experiments proved that such a tube is useless.

The filled capillary pipette should be cleansed as quickly as possible by means of water, alcohol, and ether. Should there be any difficulty from drying, the column of corpuscles may be dislodged by means of a very fine straw.

The unit of centrifugal force—*i.e.*, ten thousand rotations of the frame per minute for two minutes—was decided upon because this amount of force gave a compact volume which was but little reduced, even though the rotation were continued for several minutes. The hæmatokrit tube was increased in length to fifty millimetres, and the scale increased from fifty to one hundred divisions to permit of more accurate reading of these divisions, which are only half a millimetre apart. The lumen, which is half a millimetre, was decided upon because this small-sized lumen required but a small quantity of blood. In practical clinical work large quantities of blood are obtained with considerable difficulty, and when the puncture is properly made and everything is in readiness, the blood from this one puncture will be sufficient not only to fill the hæmatokrit tube and for the hæmoglobin examination, but also that quantity necessary for the microscopical examination of fresh and dry preparations. The tapering point of the hæmatokrit tube was decided upon because this part of the tube always carries plasma, and that portion of the scale which is lost thereby is never employed.

From many hundred examinations of blood made since the publication in 1891 of the paper before referred to I am convinced that, in all cases where the technique described is faithfully carried out, blood, as it emerges from the puncture, may be instantly drawn into the hæmatokrit tube undiluted and at once rotated, and the red corpuscles separated prior to coagulation. The use of blood undiluted removes most of the objections to the use of the hæmatokrit as an instrument of precision in clinical work, and so simplifies the entire technique that the small percentage of error remaining is due solely to the mechanical construction of the instrument, variations in the construction of its different parts, and carelessness.

The percentage volume is at once obtained from the scale upon the tube, and the entire examination may be accomplished within three minutes, without the exercise of any special skill.

This simple discovery that blood may be drawn into the tube and rotated before coagulation takes place makes the hæmatokrit the equal, if not the superior, of the hæmocytometer so far as accuracy is concerned, while from the stand-point of convenience, quickness, and ease with which blood may be examined by rotation the advantage is certainly upon the side of the hæmatokrit.

I believe the percentage of error in the use of this instrument will fall within two per cent.

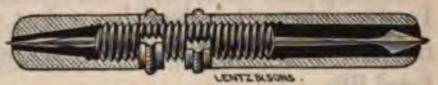
Since the employment of the new technique, and since undiluted blood has been employed, a moderate grade of leucocytosis will appear as a faint white band about a line in thickness capping the column of red blood-cells at its proximal extremity. When the leucocytosis is considerable it is not only readily recognized, but may be measured with a fair degree of accuracy. The hæmatokrit is unable to detect a slight leucocytosis; under such circumstances it is necessary to employ a blood-counter.

It is a matter of importance that all observers should decide to use one instrument, so that the results obtained would be the same wherever made. It is evident that the slightest modification in the rate of rotation, in the length of the hæmatokrit tube, or in the exact position this tube occupies in relation to the spindle, will exercise a marked influence upon the results. A modification of any one of these conditions will make a difference of at least five or ten or more per cent., and would therefore render such records useless.

The average normal percentage volume is about fifty-one per cent., and, for convenience, fifty per cent. is considered normal. To facilitate comparison, the results obtained from the various examinations of the blood should be expressed in percentages, as is the custom in reference to hæmoglobin. The normal volume of red blood-corpuscles may be considered arbitrarily as one hundred per cent., and therefore to secure this result it is necessary to double the number as read from the scale of the tube.

In obtaining blood, not only for the hæmatokrit but also for other purposes, the ordinary pin and needle are often unsatisfactory, and frequently necessitate undesirable pressure in order to secure the amount required. I have therefore devised a concealed lancet[1] (Fig. 6), modifying the one made by Gower. It has a rapidly widening point, resembling an arrow-head, so that the greater the depth the wider the incision, as will be seen in the illustration. This lancet is mounted upon a cylindrical piece of metal the lower two-thirds of which are like the lower third of a screw, and this screws into a shell of the same metal which has the same diameter as the widest part of the lancet. By this screw the movement of the lancet is controlled, and therefore the depth and width of the incision predetermined. It differs from those ordinarily employed in that it is made of metal, is longer, of a greater diameter, and the lancet proper is of the shape of an arrow-head. It may be rendered antiseptic by boiling, and is most convenient not only for hospital and laboratory work, but also for children and nervous women in private practice.

FIG. 6.



Lancet used for puncture.

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Professor Blix, at a meeting of a medical society held at Upsala, Sweden, in 1885, first suggested the use of centrifugal force for the separation of the red and white corpuscles and their volumetric estimation, taking advantage of the marked difference in specific gravity. Later Dr. S. G. Hedin, acting upon this suggestion, constructed a centrifuge and demonstrated its practicability.[2]

In the following year the author published the results of his study with this instrument: A Volumetric Study of the Red and White Corpuscles of Human Blood in Health and Disease by Aid of the Hæmatokrit, University Medical Magazine, November, 1891, and Fortschritte der Medicin, Nos. 20 and 21, October 15 and November 1, 1891.

Since this time Bleibtreu, Hedin, and the author have made additional contributions to this subject.

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[1] Manufactured by Messrs. Charles Lentz & Sons, Philadelphia.

[2] Ueber die Brauchbarkeit der Centrifugalkraft für quantitative Blutuntersuchungen. Separat-Abdruck aus dem Archiv für die gesammte Physiologie, Bd. lx.

DISEASES OF THE THYROID GLAND.

BY A. H. WENTWORTH, M.D.

Anatomy.—The thyroid gland in the human embryo consists of three separate parts,—two lateral and one middle. Later in development the lateral portions become united by means of the middle portion, or isthmus. In the embryo the gland has a duct which opens at the foramen cæcum at the base of the tongue. This duct becomes obliterated towards the end of fœtal life.

At birth the thyroid gland consists of two lateral lobes united towards their lower ends by a transverse portion called the isthmus. Each lateral lobe lies on the side of the trachea, extending from the fifth or sixth ring to the thyroid cartilage. The isthmus commonly lies across the second, third, and fourth rings of the trachea.

Variations in the size, shape, number, and position of the lobes of the thyroid gland are common. At times the gland consists of two separate parts on each side of the trachea, or there may be only one lateral lobe, or the three lobes may not be united.

Quite commonly there is considerable difference in the size of the lobes. Sometimes the lateral lobes are partly divided into a number of smaller ones, without, however, being completely separated. At times the position of the gland is altered so that the isthmus lies more to the side of the trachea.

The occurrence of accessory glands is of clinical interest, inasmuch as it helps to explain in certain cases the lessened severity of the symptoms following extirpation of the gland. These accessory glands are found in the region of the aorta, in the supra-clavicular fossæ, and to the side of and behind the pharynx and large vessels of the neck. The thyroid gland is larger in females than in males, and weighs from six to twelve grammes from the first year of life to puberty. It is invested with a capsule which extends into the gland in the form of a mesh-work and divides it into lobules. The lobules contain multitudes of closed acini held together by loose connective tissue. The acini are lined with a single layer of cubical epithelium and are filled with colloid material.

The gland is very vascular, and derives its blood-supply from the superior and inferior thyroid arteries of each side. At times a fifth vessel, the thyroidea ima, is found. These vessels are of relatively large size and form frequent and large anastomoses. They terminate in a capillary network in the loose connective tissue surrounding the acini, in intimate connection with the epithelium.

The veins are large, and are said to be valveless. They ultimately form large plexuses on the surface of the gland, from which a superior, a middle, and an inferior thyroid vein are formed on each side. The first two empty into the internal jugular vein, and the last empties into the innominate.

The lymphatics form numerous and large anastomosing trunks, both on the surface and throughout the substance of the gland. They originate in the connective tissue surrounding the acini, without, however, appearing to communicate with the cavities of the latter. They contain lymph and colloid material similar to that found within the acini.

The nerves are derived from the middle and inferior cervical ganglia of the sympathetic and from the pneumogastric. They accompany the blood-vessels, and have here and there ganglion cells in their course. Their mode of termination is unknown.

Function.—A consideration of the older hypotheses concerning the function of the thyroid gland can be omitted. It will be sufficient to mention the two most recent theories regarding its function.

The results of many investigations have shown that the thyroid gland is essential to the normal performance of the functions of the various organs of the body. One explanation of the way in which the thyroid gland accomplishes these results is that the gland secretes a substance essential to the organism, which is carried by the blood to the various organs. This theory attributes a nutritive function to the gland. The second and more generally accepted hypothesis attributes an antitoxic function to the gland. The supporters of this theory believe that the thyroid gland prevents auto-infection of the organism by destroying or altering certain products of metabolism which would otherwise prove poisonous. The morbid symptoms which follow extirpation or absence of the thyroid gland are not those of disturbances of nutrition, but rather the symptoms of an acute or chronic poisoning, especially of the central nervous system. The majority of investigators deem it probable that the thyroid gland secretes a substance, prepared from the blood, which antagonizes certain toxic products of metabolism present in the blood, and that this substance is, therefore, antitoxic in its action.

Experimental removal of the gland from animals produces, after a varying length of time, cachexia, anæmia, severe nervous disturbances, such as tetanic convulsions, paresis and paralysis, tachycardia, and so on, trophic disturbances, and, finally, death.

A limited number of experiments have been made to determine the effects produced by injecting or feeding healthy animals with thyroid preparations. The results obtained were practically the same in all cases. After a varying length of time, depending upon the animal, the quantity of the active principle of the thyroid used, and the methods of administration, the animals showed loss of weight, tachycardia, and increased blood-pressure, and finally died of exhaustion. In some cases polyphagia, polydipsia, and polyuria were observed. The symptoms were strikingly like those of exophthalmic goitre, except that there was no exophthalmus or increase in the size of the thyroid gland.

The thyroid gland produces a colloid substance which micro-chemical examination has shown to be albuminoid in character. It is apparently a product of the epithelium of the acini. This function is said to begin from the sixth to the eighth month of fœtal life. Attempts to increase this secretion by irritation of the nerves supplying the gland have given negative results. These experiments have led to the conclusion that increased activity of the gland is caused by the presence or absence of unknown substances in the blood.

The activity of the gland is most marked in early life. In later life there is more or less atrophy of the epithelium and increase in the interstitial tissue, together with lessened colloid formation. For this reason operative removal of the gland in early life is more likely to be followed by morbid symptoms than its removal at a later period. These morbid symptoms consist of various nervous, nutritive, and trophic disturbances similar to those which occur in the disease myxœdema. The symptoms are grave, and are dependent upon the cessation of the gland's function. The cessation of function may be caused by degeneration of the gland as well as by its operative removal. The name *cachexia thyroidectomica* is applied to the symptoms which follow operative removal of the healthy gland, and *cachexia strumipriva* to the symptoms which follow extirpation of a goitrous gland.

The symptoms may occur days, weeks, or even months after the operation.

The present article is limited to the consideration of hyperæmia, acute thyroiditis, malignant growths, syphilis, tuberculosis, and epidemic, sporadic, and endemic goitre.

HYPERÆMIA.

In consequence of the extreme vascularity of the thyroid gland, enlargement due to an increase in the quantity of blood in the gland is not uncommon. This increase in size is not associated with changes in the structure of the gland unless the cause is persistent, and should not be confounded with enlargement due to acute inflammation of the gland or with enlargement caused by chronic changes such as occurs in goitre. The hyperæmia may be active or passive, temporary or lasting.

Active hyperæmia frequently occurs as a so-called neuropathic hyperæmia in females, especially at the time of puberty, during the menstrual periods, and at times during pregnancy.

Passive hyperæmia is associated with chronic circulatory disturbances, as in heart-disease, compression of the veins of the neck from any cause, occupations which cause venous congestion in the vessels of the neck, such as carrying heavy burdens, and so on.

Symptoms.—There are frequently no symptoms except the enlargement of the gland, which may be considerable. The skin over the gland is normal, and there is neither pain, tenderness, nor other subjective symptoms. If the enlargement is extreme, it may cause symptoms due to mechanical pressure. If the cause is persistent, hyperplasia of the gland-tissues and chronic dilatation of the vessels, either arteries or veins, occur.

ACUTE IDIOPATHIC THYROIDITIS.

Acute idiopathic inflammation of the thyroid gland is a very rare disease. Up to the present time about eighty cases of the disease have been published. A considerable number of these were cases of *acute strumitis,*—that is, inflammation occurring in a goitrous degenerated gland,—and should not have been classified with the idiopathic affection.

Etiology.—The disease is assumed to be due to bacterial affection, although the only proof which has been adduced is based upon the analogy between this disease and a certain number of cases of metastatic inflammation of the thyroid in which bacteria have been found. A number of cases of thyroiditis have occurred in connection with rheumatism, malaria, typhoid, erythema nodosum, diphtheria, erysipelas, puerperal septicæmia, and parotitis. It is probable that these cases should be regarded as complications of the above diseases rather than as idiopathic thyroiditis. The disease is very rare in infancy and childhood. A number of cases of primary thyroiditis have occurred at the time of puberty in females oftener than in males.

Pathology.—The pathology of the disease is unknown, because most cases have ended in recovery.

Symptoms.—The disease is characterized by acute swelling of the gland, accompanied by fever and malaise. The swelling is usually moderate, and may affect one or both lobes. The consistency of the swelling is at first elastic, and later more doughy. The skin becomes reddened and is tender to the touch. As the swelling increases, all movements of the gland, whether active or passive, are painful. There is spontaneous pain in the neck, frequently radiating into the arms and chest. Deglutition is painful. Respiration may be painful and difficult. Pressure-symptoms occur, dependent upon the amount of swelling and its location. Hoarseness, dyspnœa, and even asphyxia may occur. Pressure on the cervical and brachial plexuses may cause neuralgic pains in the parts supplied by these nerves. The fever is usually moderate, and to some extent proportionate to the amount of swelling.

The disease terminates in resolution, suppuration, or gangrene.

In about one-fourth of the cases resolution occurred within from three or four days to a week. The average duration of the disease is from two to three weeks. When suppuration occurs the disease may last much longer. When resolution occurred the symptoms gradually subsided with the disappearance of the swelling. Sometimes recovery was delayed by the persistence of small areas of swelling.

Suppuration occurred in from sixty to seventy per cent., and usually circumscribed areas of fluctuation appeared. The symptoms were more severe and persistent, and usually gradually subsided with the spontaneous or operative evacuation of pus. Sometimes the suppuration was more diffuse, and the surrounding tissues became œdematous and of doughy consistency, followed later by the formation of deep-seated abscesses. The rupture of such abscesses into the trachea, œsophagus, or mediastinum caused a fatal termination of the disease in some cases.

The occurrence of gangrene was reported in only six or eight cases. At first the course of the disease was similar to that of the cases in which suppuration occurred. After incision or spontaneous evacuation of pus, the gland-tissues were found to be gangrenous, and the surrounding tissues showed phlegmonous inflammation. In spite of the severity of the process, most of these cases recovered.

Diagnosis.—Hyperæmia causes less swelling, and there are no symptoms of inflammation or of general infection. The location and acute character of the swelling, together with the accompanying symptoms, differentiate the disease from other swellings in this region.

Prognosis.—The prognosis is always guarded, especially in those cases in which suppuration occurs. But in the latter the early evacuation of the pus permits of a favorable prognosis in most cases. Death may occur from asphyxiation, from perforation and extension of the pus into neighboring organs, or from sepsis. Of the cases thus far reported about twenty-five per cent. terminated fatally.

Treatment.—The application of leeches and treatment by means of mercurial inunctions have no effect on the inflammation, and may do harm. Leeches have caused collateral œdema, and mercurial inunctions have caused severe stomatitis in a number of cases. Cold applications in the form of compresses, ice-collar, or Leiter's coil are recommended. Laxatives are frequently indicated, especially magnesium sulphate. Small doses of morphine are often needed to relieve the pain. Antipyretics are rarely necessary. Stimulants may be required. A high and long-continued temperature generally indicates the presence of pus, even in the absence of pronounced fluctuation, and requires surgical treatment.

METASTATIC INFLAMMATION OF THE THYROID GLAND.

Etiology.—A number of cases of metastatic inflammation have been reported which have occurred as complications in various infectious and septic diseases. In many of these the specific bacteria of the original disease have been found, such as the typhoid bacillus, the diplococcus pneumoniæ, the streptococcus pyogenes, and others. In a number of cases the thyroid affection occurred late in the course of the original disease or during convalescence.

Symptoms.—The course of the disease is modified as regards fever and acute symptoms from that of idiopathic thyroiditis. In most cases a "cold abscess" forms without acute symptoms. Pain is frequently slight or absent, and the fever is generally moderate. The formation of pus is gradual, and its presence may not be suspected until fluctuation occurs.

Diagnosis.—The occurrence of thyroid enlargement in connection with an infectious or a septic disease would suggest the nature of the swelling, and the formation of an abscess would confirm the diagnosis.

Prognosis.—The prognosis of idiopathic thyroiditis applies equally well to this disease.

Treatment.—The treatment consists in the early evacuation of the pus.

MALIGNANT TUMORS.

The thyroid gland is one of the rarest seats for malignant growths. They occur in advanced life, and in almost every case are preceded by some variety of goitre. The growth may be either sarcoma or carcinoma.

SYPHILIS.

Syphilis is very rarely manifested in the thyroid gland, and, so far as known, is always associated with visceral syphilis. The presence of gummata has been noted in a certain number of cases of congenital syphilis.

TUBERCULOSIS.

Tuberculosis of the thyroid gland is not so uncommon as was formerly supposed, especially the occurrence of miliary tubercles in connection with general miliary tuberculosis. The chronic form consisting of caseous nodules is uncommon. Enlargement of the gland in these cases rarely occurs, or is so slight as to escape notice. One case of primary tuberculosis has been reported.

GOITRE.

Synonyms.—Kropf, Struma, Goître, Gozzo, Wen, Derby neck, Bronchocele.

Goitre may be epidemic, sporadic, or endemic. The course of the disease may be acute or chronic, depending upon the variety. These varieties will be considered separately in order to avoid confusion. The epidemic and sporadic forms of the disease require but a brief description.

EPIDEMIC GOITRE.

Occurrence.—This form usually occurs in localities where goitre is endemic, although its occurrence has been reported in localities free from goitre. It occurs most frequently in summer and autumn, and affects individuals who have not resided long in the place. The epidemics are confined to barracks, seminaries, and so on, where large numbers of people live together in close proximity. The other inhabitants of these localities are not affected.

Pathology.—The enlargement of the gland is of a vascular character, due to acute hyperæmia, without any change of the gland-tissues. The presence of loud vascular murmurs heard over the gland, especially in children, and the complete and rapid disappearance of the enlargement, with or without appropriate treatment, prove that the process is a vascular one. As a rule, the entire gland is affected. The disease is acute, and the swelling is produced within from a few hours to a few days or weeks. Recurrence is common, and in these cases the enlargement may become persistent, and is then associated with changes in the gland-tissues.

Symptoms.—The enlargement of the gland is the chief symptom. The subjective symptoms are slight, and, as a rule, do not incapacitate the individual for work. Sometimes the enlargement is of sufficient size to cause pressure-symptoms.

Prognosis and Treatment.—Recovery usually occurs either spontaneously or after change of location, drinking-water, and so on.

SPORADIC GOITRE.

Occurrence.—This form of goitre occurs everywhere. It is frequently observed in localities in which goitre is not endemic. The disease is much commoner in females than in males, and occurs more frequently in young people than in old. Oftentimes it occurs in girls at the time of puberty, in some of these cases associated with chlorosis. Certain cases combined with exophthalmus and tachycardia are of neuropathic origin (exophthalmic goitre). In other cases sporadic goitre is associated with menstruation, gravidity, trauma, and occupation. In the latter class of cases it appears essential for the occupation to be one that produces a persistent or frequently recurring congestion of the gland. Finally, anomalies of development may produce a predisposition to the disease in some cases.

Pathology.—The enlargement is due to long-continued or frequently recurring hyperæmia of the gland. If the cause persists long enough, tissue-changes are produced, and the enlargement becomes permanent.

Symptoms.—Usually the only symptom is the increase in size of the thyroid gland. The swelling is generally slight and not very noticeable.

Prognosis.—In most cases when the cause is removed the swelling disappears.

Treatment.—Occupations which produce persistent or frequently recurring hyperæmia of the gland should be given up. Cases associated with anæmia require proper hygienic and tonic treatment.

ENDEMIC GOITRE.

Occurrence.—This variety of goitre, as its name implies, occurs in certain localities. These localities are distributed throughout the world, although in some countries they are more numerous than in others. Variations in the intensity of the disease are observed at different times in the same locality. The disease may become endemic in places previously exempt.

Endemic goitre may be congenital or acquired.

CONGENITAL GOITRE.

This variety occurs almost exclusively in localities where goitre is endemic. It is not a common disease. In most cases one or both parents have goitre. Generally, congenital goitre is of the hyperplastic type, although a number of cases have been observed in which secondary changes had occurred. The goitre is usually retro-œsophageal or submaxillary. The increase in size is apt to be rapid and cause pressure-symptoms, especially tracheal stenosis. The latter may be so extreme as to cause death. It is stated that in some cases the goitre may be so large as to interfere with the birth of the child.

ACQUIRED GOITRE.

This occurs much oftener in females than in males, usually at the time of puberty, although it may happen at any time in later life. The course of the disease is chronic, and causes progressive enlargement of the gland, although variations in size may occur from time to time.

Etiology.—Many causes have been assigned, such as drinking-water, altitude, character of the soil, temperature, climate, season of the year, light, air, occupation, and trauma. The generally accepted theory at the present time is that the disease is miasmatic, and that the noxious element is conveyed into the system through the drinking-water. It might properly be included in the list of infectious diseases. A certain predisposition of the individual is assumed to be essential, besides certain conditions of the soil, to enable the organism to thrive.

Pathology.—The enlargement of the gland may be partial or general. In the latter case, however, one lobe is usually more affected than the others. Special names are used to designate the occurrence of goitre in certain regions, as substernal, retropharyngeal, submaxillary.

The process consists of hyperplasia of the gland-tissues subsequent to hyperæmia. The classification of the varieties of goitre depends upon the predominance of the hyperplasia in certain tissues of the gland, and upon secondary changes which may occur later in the goitre.

The primary change in almost all cases is a hyperplasia of the acini with proliferation of the epithelium. Accompanying this change there is a new formation of blood-vessels and usually a slight increase in the interstitial tissue. This form of goitre is commonly of moderate size and of soft consistency, and is called *parenchymatous goitre*. A second variety, *colloid goitre*, is developed from parenchymatous goitre by the formation within the hyperplastic acini of an excessive quantity of colloid material. This form of goitre causes a more uniform enlargement of the gland than the preceding, and attains a larger size. The consistency is soft and doughy.

A third variety, *cystic goitre*, is caused by the constant increase in the production of colloid material within the hyperplastic acini. As the latter become more and more distended, atrophy of the interstitial tissue and blood-vessels is produced and the acini become confluent. In this way cysts are formed, which in turn become confluent and produce larger ones. In extreme cases the whole affected portion of the gland may become transformed into a single large cyst, or more frequently into several. This cystic enlargement often attains a great size. Secondary changes usually occur in the walls and contents of these cysts. Hemorrhages into the cysts are frequent, and in some cases may be extensive enough to warrant the name of hemorrhagic goitre. Calcification may occur in the cyst-walls, as well as in other parts of the parenchyma.

A fourth variety, *fibrous goitre*, is caused by hyperplasia of the interstitial tissue between the acini. This rarely occurs primarily, but usually takes place in the first-mentioned variety, the parenchymatous goitre. The increase in interstitial tissue is followed by fatty degeneration and atrophy of the gland-substance, and by the formation of small, hard, nodular masses. These masses may become more or less agglomerated and produce a considerable enlargement of the gland.

A fifth variety, *vascular goitre*, occurs when the hyperplasia affects chiefly the blood-vessels. This variety is also secondary and develops in a pre-existing goitre. In addition to the new formation and aneurismal dilatation of the arteries, there may be varicose dilatation of the veins. These vascular changes may be so extreme as to cause the atrophy and disappearance of the greater part of the gland-substance. The terms aneurismal and varicose goitre are used to designate one or the other variety. Some or all of the above-mentioned changes may be present in the same goitre and give rise to a very mixed picture. It is only in the early stages that one finds the first-mentioned variety alone. As the individual grows older, one or more of the secondary changes occur. In children parenchymatous goitre is the variety usually found.

Symptoms.—The symptoms consist of enlargement of the gland, associated with secondary mechanical symptoms, due to pressure and congestion.

Venous congestion may be caused by the hinderance to the venous circulation by pressure on the neighboring veins, by varicose dilatation of the veins in the gland, and by obstructed expiration produced by tracheal stenosis or by paralysis of the recurrent laryngeal nerve. The symptoms consist of cyanosis of the face, headache, dizziness, oppression.

Cardiac symptoms may be caused by pressure on the accelerator branch of the vagus, and consist of palpitation, increased frequency and irregularity of the heart's action.

Respiratory symptoms may be caused by direct compression of the trachea or by paresis or paralysis of the recurrent laryngeal nerve from pressure. The respiratory symptoms increase with the growth of the goitre. At first they are temporary and depend upon accidental causes, such as tracheal catarrh, more or less violent exertion, and so on, which produce increased congestion of the mucous membrane of the trachea. The already diminished orifice of the trachea becomes still smaller, and causes inspiratory dyspnœa. At times the dyspnœa may become so marked as to necessitate tracheotomy. A substernal enlargement of the gland is especially likely to cause acute attacks of dyspnœa, owing to its situation between the sternum and the vertebral column. In the early stages, before the goitre becomes securely fixed to the neighboring tissues, such attacks are frequently relieved by the temporary escape of the gland from beneath the sternum. In some cases, after the goitre has become fixed it may be so completely hidden by the sternum as to escape observation, and thus prevent the recognition of the cause of the dyspnœa.

With the increase in size of the goitre the symptoms of tracheal stenosis assume a permanent character, and both respiration and speech are difficult. Much the same effect may be produced by paralysis of the recurrent laryngeal nerve. In most of these cases, however, the symptoms consist of milder respiratory disturbances and hoarseness. A certain proportion of these cases are subject to acute attacks of suffocation, resembling asthma. These attacks last a number of minutes, and may be so severe in some cases as to cause the immediate death of the individual. The consequences of chronic respiratory disturbances are bronchial catarrh, emphysema, passive congestion of the lungs, hypertrophy, and dilatation of the heart.

Disturbances of deglutition may occur from compression of the œsophagus. This occurs oftenest in connection with retropharyngeal enlargement of the gland. The compression may become so extreme as to interfere seriously with the nutrition of the individual.

Diagnosis.—As a rule, the diagnosis of goitre is not difficult when it is not substernal or retropharyngeal. The presence of a tumor in the region of the thyroid gland which moves with respiration and the action of swallowing, the gradual increase in size, together with the character of the swelling, and the outward dislocation of one or both carotid arteries, are sufficiently characteristic.

The diagnosis of enlarged lymph-glands occurring in this region in Hodgkin's disease or in lymphosarcoma may present difficulties at times. In these cases, however, it is usually possible to detect separate glands in the mass; other glands in the vicinity and elsewhere are enlarged, and the patient is cachectic. Other tumors, such as branchial cysts, and aneurism, are rarely difficult to diagnosticate. The differential diagnosis of the variety of goitre has been sufficiently indicated in the description of the pathology of goitre. The size, shape, and consistency of the tumor indicate the variety, with the exception of the colloid and cystic forms of goitre. In the case of the two last-mentioned varieties, an exploratory puncture with a sufficiently large trocar may be necessary to establish the diagnosis. The danger of severe hemorrhage in these cases is slight, because these varieties of goitre are not so vascular as the others.

Prognosis.—In some cases a change of residence to a locality in which goitre is not endemic has been followed by a cessation in the growth or even by diminution in the size of the gland. Certain methods of treatment have also produced improvement.

Death may ensue from laryngeal stenosis and its sequelæ, from trauma, causing hemorrhage, from acute inflammation, and from malignant growths developing in the goitre.

Acute inflammation may be caused by injury or bacterial infection. The symptoms and course of the disease are similar to those of acute thyroiditis, and need not be recapitulated. The symptoms of compression, however, are naturally much more marked in these cases than in primary thyroiditis. The cyanosis and dyspnœa often reach an extreme degree.

Treatment.—The treatment of endemic goitre is prophylactic, medical, and surgical. It is self-evident that the best prophylaxis consists in avoiding those localities in which endemic goitre occurs. Since the infectious material may be carried into the system by means of the drinking-water, the latter, if obtained from springs or wells in the locality, should always be boiled, or rain-water may be substituted. Improvement in the drainage and general hygienic surroundings of such localities tends to exterminate the disease.

The only drug which has been found to be efficacious is iodine. It may be administered internally in the form of the tincture or as Lugol's solution, in combination with potassium as iodide of potassium, or in combination with iron as iodide of iron.

The tincture is given at first in small doses (five drops) two or three times a day, and gradually increased to ten drops three times a day. It should be given after meals, and always well diluted. The compound solution of iodine (Lugol's solution) is preferable to the tincture, and is given in doses of five drops three times a day after meals, increased one or two drops every other day until fifteen or twenty drops are taken. Should "iodism" occur, or if, after one or two months' treatment, no reduction of the enlargement has taken place, iodide of potassium may be substituted in doses of from five to twenty grains three times a day. When anæmia is associated with the disease, iodide of iron and cod-liver oil may be alternated with the above-mentioned remedies.

Iodine may be applied externally in the form of Lugol's solution or in that of iodine ointment. It is recommended to apply Lugol's solution in the morning rather than at night, to avoid the danger of iodism from inhaling the fumes of iodine.

The treatment of goitre by parenchymatous injections of iodine is less common than formerly. There exists some doubt as to its superiority over the internal administration of iodine or its compounds, and its administration in this way has been followed by acute inflammation or severe neuralgic pains in many cases, and in a few cases by sudden death. Moreover, the recent advances in surgery have tended to supplant this method of treatment. In general, it may be stated that the earlier the treatment with iodine or its compounds is begun the better the chances for improvement.

Diminution of the enlargement and relief of the symptoms have been noted in many cases. The older forms of goitre are not benefited, as a rule.

The most recent treatment consists in the administration of thyroid and thymus preparations. The number of cases thus treated which have been reported is comparatively small, and there is still considerable uncertainty as to the efficacy of the treatment. In a certain percentage of the cases a diminution in the size of the goitre has been observed. According to some observers, if any reduction in the size of the goitre occurs, it does so within two or three weeks if the treatment is employed daily. The best results have been obtained thus far in the treatment of the parenchymatous variety of goitre occurring in young individuals, whereas the cystic, colloid, and fibrous varieties have been only slightly or not at all affected by the thyroid treatment.

For a consideration of the theories which have been advanced to account for the beneficial action of thyroid preparations on certain morbid conditions following the loss of function of the thyroid gland from any cause the reader is referred to the article on myxœdema. The thyroid preparations are usually administered internally in the form of powders or tablets of the extract. It is necessary to remember that overdoses of thyroid extract may produce disagreeable or even dangerous symptoms, consisting of anorexia, loss of weight, sleeplessness, elevation of temperature, tachycardia, pains in the extremities, dyspnœa, stenocardial attacks. If any of these symptoms occur, the treatment should be stopped for a time and smaller doses given afterwards. It is safe to begin with one grain of the extract three times a day and increase the dose gradually.

A consideration of the surgical treatment of goitre does not come within the scope of the present article.

DISEASES OF THE THYMUS GLAND.

Development.—The thymus gland is of epithelial origin, and arises as a diverticulum from an anterior pair of the visceral clefts.

Anatomy.—The gland consists of two lobes in close contact in the median line. "In its mature state in an infant under two years of age it appears as a narrow, elongated, glandular-looking body, situated partly in the thorax and partly in the lower region of the neck; below, it lies in the superior mediastinal space, close behind the sternum, as far down as the fourth rib-cartilage, and in front of the great vessels and pericardium; above, it extends upward upon the trachea in the neck as high as the lower border of the thyroid, being covered by the sterno-hyoid and sterno-thyroid muscles." (Quain.) The average weight of the gland at birth is fourteen grammes; marked variations in weight are common. The gland increases in size up to the second year, then undergoes fatty degeneration, atrophies, and disappears generally at puberty. Exceptionally, it persists even after the twentieth year.

Structure.—The structure of the gland resembles that of lymph-glands. The medulla contains peculiar corpuscles with a concentric striation composed of an envelope of epithelial cells enclosing a central mass formed of granular cells, the so-called corpuscles of Hassall. In the adult the small remains of the gland are usually composed of adipose tissue.

Function.—It is regarded as one of the blood-forming organs. Experimental removal of the gland from young animals appears to produce no constitutional effects. Experimental removal of the thyroid gland from young animals is sometimes followed by a slight increase in the size of the thymus gland, without, however, preventing the characteristic constitutional symptoms which follow this operation.

DISEASES.

Inflammation.—Suppurative inflammation, secondary to pyæmic processes, especially thrombo-phlebitis of the umbilicus, rarely occurs.

Primary inflammation is reported, but lacks proof. The diagnosis of the reported cases depended upon the presence of puriform material in the gland, but in these cases the diagnosis was not verified by microscopic examination of the gland-tissues. The presence of this puriform material may be accounted for by post-mortem softening of the gland, or in some cases by the softening of gummata. The occurrence of apparently multiple abscesses has been considered pathognomonic of congenital syphilis, and was first described by Dubois (Dubois's abscesses).

These areas may be due to post-mortem softening, or, as Chiari has shown, to the formation of cysts within Hassall's corpuscles by the extension and growth of the tissue of the gland into them. They probably have no connection with syphilis.

Thymic Asthma.—A number of cases of sudden death have been reported in which at autopsy the thymus gland was found enlarged and no cause for death was detected in the other organs. These cases occurred chiefly in infants and young children who were considered healthy. These deaths have been attributed to asphyxia from compression of the trachea or bronchi by the enlarged thymus.

This view is opposed by good observers, who assert that these individuals were not perfectly healthy; that they showed general nutritive disturbances of the lymphatic system, such as enlargement of the spleen, lymph-glands, and tonsils; furthermore, that in no case has actual compression of the trachea or bronchi been found at autopsy; that the patients died with symptoms of paralysis of the heart, and not of asphyxia; and, finally, in certain of these cases tracheotomy, artificial respiration, and application of electricity to the phrenic nerves have failed to restore the respiration, although resorted to immediately.

Consequently, the evidence is against death from asphyxia.

Pott believes that enlargement of the thymus gland may be the *indirect* cause of sudden death. He assumes that an enlarged thymus may press upon the pulmonary artery and heart, and so cause reflex paralysis of the heart.

Other observers believe that the enlargement of the thymus gland is only one element in a general nutritive disturbance of the lymphatic system, and that it is not a direct cause of sudden death.

Syphilis.—Gummata may occur, associated with fibrous induration of the gland.

Tuberculosis.—Miliary tubercles and caseous tuberculous nodules occur, almost invariably associated with tuberculosis in other organs.

Tumors.—Enlargement of the thymus gland occurs in leukæmia in connection with the enlargement of the other lymphatic organs. Sarcoma may occur, but is rare.

THE NORMAL PRÆCORDIA OF INFANCY AND CHILDHOOD.

BY ARTHUR R. EDWARDS, A.M., M.D.

FEW themes in medicine contain such meagre material and varying statements as the normal præcordia of infancy and childhood. Many authorities in pediatrics and heart-disease either wholly disregard the subject or touch it lightly, as Baginsky,[1] Finlayson,[5] McClellan,[6] Osler,[7] Cheadle,[9] Sansom,[10] Fothergill,[3] Bamberger,[4] Fraentzel,[8] Cutler,[3] Garland,[3] Friedreich,[9] Loomis,[11] Guttmann,[12] Eichhorst,[13] Henoch,[14] Skoda,[52] J. Lewis Smith, and others. Again, individual treatments of the topic vary so widely that an approach to its elaboration is possible only by citation of diverse opinions.

The Size of the Heart.—The statements of Venois,[38] Bednar,[40] Bizot,[39] who held that the heart grows but little from the fourth to the thirteenth year, and Rilliet and Barthez,[29] are not wholly reliable, being determined only by work on the cadaver. Müller[25] first established the relative weight of the heart as being largest up to the fifth year. While in embryonal life the right is to the left ventricle as 6 is to 7, in the first four weeks post partum the left ventricle increases while the right decreases in size, so that at the end of the first year the right is but half as large as the left.

Müller notes four epochs in the heart's later growth:

1. In the first month the heart does not respond to the demands upon it, but remains as at the end of gestation.
2. Thence to the fifth year the relative weight decreases.
3. From the sixth year the heart varies with sex, increasing in males, decreasing in females.
4. At puberty, from the sixteenth to the twentieth year, it increases in both sexes, as first ascertained by Peacock[27] and Boyd.[42]

The Site of the Heart.—Gerhardt's Handbuch says but little, while Henke,[18] Luschka,[23, 24] Rüdinger,[28] and Skoda[52] merely allude to the high position of the diaphragm. Hammernik[19] describes two types: in one the heart cannot be dislocated, being wedged in between the sternum, diaphragm, and left lung, while in the second type the heart can be luxated, since the diaphragm becomes lower. Luschka[24] finds that the heart can always be displaced. Sahli[30] holds that only the central part of the diaphragm stands high, but not that part upon which the heart rests. Von Starck[16] thinks that the part of the diaphragm upon which the heart rests is variable in elevation. He disregards Sahli's results, since he did not specify age. From Rüdinger's plates the heart's position seems higher than in adults, although Rauchfuss[15] and Steffen[31] believe that the heart lies horizontally in the thorax. (For upper and lateral borders, see Percussion, hereafter.)

The form and development of the thorax, as Rauchfuss proved, have an influence on the heart's location. In the new-born the sterno-vertebral and transverse diameters of the thorax are nearly alike (8 centimetres). The ratio becomes 1 to 1.4 (measurements in adults are 19 and 26 centimetres). At the sixth year the ratio is 14 to 18; from the tenth to the twelfth year, 14.5 and 20 centimetres. Gierke[17] determined contrary results. Henke[18] describes those alterations in the infantile thorax which necessitate a small transverse and deep sagittal diameter. Hueter[20] explains the transverse increase by osteochondral epiphyseal growth. Wintrich[34] does not mention this variation from adult findings. Von Starck summarizes the subject as follows. The marked bulging of the chest in earliest childhood and the relatively large heart bring the heart nearer the chest-wall, and hence it is more accessible to the percussing finger. The bulging chest-wall, on the other hand, so covers the heart that we cannot determine the apex beat. Since the transverse thoracic diameter increases, the heart becomes small.

Location of the Apex Beat.—The nipple, although considered somewhat unreliable as a landmark by Momberger,[26] Luschka,[24] and Sahli,[30] is sufficiently accurate for clinical purposes. It is usually in the fourth interspace or on the fourth rib, but may be on the third or the fifth. Steffen,[31] one of the first to establish the characters of the child's heart, says that the apex is usually located in the fifth interspace at the nipple line, or may even be external thereto, without being pathological. More than 1 centimetre external to the nipple line is pathological. The apex is rarely observed in the fourth or the sixth interspace. Gierke[17] finds it in the fifth interspace in the nipple line, with a maximum variation of 1 centimetre externally and 1½ centimetres internally. Its location is variously described by different authors: ½ to 1 centimetre external to the nipple line, or even 2 centimetres, between the tenth and the twelfth years (Rauchfuss); even 3 centimetres external to the nipple line (Rosenstein); farther to the left than in adults, in the fifth interspace at the nipple line, rarely internal thereto (Von Dusch[44]); in the fifth interspace, external to the nipple line (Gerhardt[45]); 1 centimetre external to the nipple line in the fifth interspace (Guttmann[12] and Baginsky[1]); in the fourth interspace (Vogel and Biedert[33]); in the fourth or fifth interspace, from 1 to 2 centimetres outside the nipple line (Wassilewski[35]), taken from eighteen hundred and twenty sick children; in the fourth interspace in the first years, in the fourth or fifth in middle childhood, in the fifth in later childhood (Sahli).

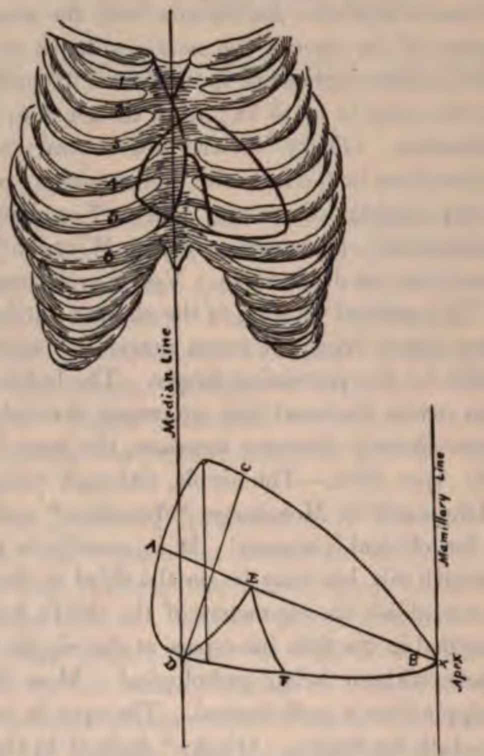
Von Starck's conclusions regarding the location of the apex beat:

1. In the first years of life the apex beat is often indeterminable.

2. Up to the fourth year, in the majority of cases, the apex lies beyond the nipple line, but less and less frequently as we approach the twelfth year. It is not found external to the nipple line after the thirteenth year.

3. The apex is rarely found in the nipple line in the first year; more frequently up to the seventh; less frequently later; in the fourteenth year it is frequently found there.

FIGS. 1 AND 2.



A, B, long axis; *C, D*, transverse; *D, E, F*, small dulness; *A, D, E* includes large arterial trunks and right auricle. (After Gierke and Steffen.)

4. The apex is not found internal to the nipple line before the second year; rarely so up to the seventh; in the majority of cases between the ninth and thirteenth years; and later almost invariably.

5. In the first year the apex is found in the fourth interspace; more and more infrequently as time advances.

6. The apex is rarely found during the first and second years in the fourth and fifth interspaces; frequently from the third to the sixth year; later infrequently.

7. In the fifth interspace rarely in the first and second years; later more frequently; usually there from the seventh to the thirteenth year; invariably after the thirteenth year.

8. Extremely infrequently in the sixth interspace.

As a general statement, then, the apex lies farther externally in proportion to the size of the thorax than in adults, and lies in earliest childhood beyond the nipple line, in mid-childhood in that line, and later within the same. The apex is higher than in adults, in the earlier half of childhood being in the fourth or fifth, and in the later half in the fifth interspace.

Cardiac Dulness.—Steffen and Gierke (*loc. cit.*) describe the heart's location as horizontal. (See Figs. 1 and 2.) The relative dulness reaches the second interspace above; its right border runs from one to three and three-quarters centimetres (according to age) to the right of the median line, terminating in the fourth right interspace or under border of the fourth rib (fifth rib when the diaphragm is low); the left border passes in a curved line from above through the nipple to the apex; the lower boundary is horizontal, and, according to Steffen, can be differentiated from the liver. The absolute dulness is triangular or quadrangular, with the vertical longer than the horizontal side.

According to Rauchfuss (Fig. 3) (*loc. cit.*), the relative dulness is in early life indeterminable above, but later is limited by the second ribs or interspaces. The right border, beginning in the second interspace or third costal cartilage near the sternum, is a line curved convexly to the right, being farthest from the sternum in the right parasternal line at the height of the diaphragm, whence it curves gradually in towards the sternum, making an angle whose obliteration is common in pericarditis. The left border is a curve convex externally, which intersects

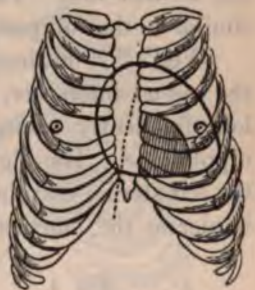
The third costal cartilage in the parasternal line,

The fourth costal cartilage in the nipple line,

The fifth costal cartilage from one-half to one centimetre external to the nipple line, and

The sixth costal cartilage from one to two centimetres external to the nipple line.

FIG. 3.



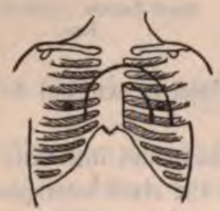
Heart dulness. (After Rauchfuss.)

The lower border can be outlined when the stomach and intestines are distended, and in varying positions, extending from the sixth interspace to from one to two centimetres external to the left nipple line. The absolute dulness extends downward from the sternal end of the fourth to that of the seventh left costal cartilage, along the latter outward to the sixth costal cartilage at the left parasternal line, upward in the same line to the fourth costal cartilage.

According to Whitney,[3] in children to the sixth year relative dulness is practically as in adults,—namely, above and to the left, a curved line extending from the junction of the third rib or interspace and sternum outward and downward to the apex of the heart in the fourth or fifth interspace near the mammary line. The right boundary (the most important, being most variable in certain pathological conditions) is perpendicular, corresponding nearly to the left border of the sternum. The resonance of the lower half of the sternum is, therefore, the same as its upper half in children under six years of age. From the fifth to the ninth year the præcordial dulness varies. In a few, even at six years, some slight dulness will be found over the lower half of the sternum. While at five years all cases presented what Whitney designates "the infantile præcordia," at six there were two infantile to every one enlarged; at seven equal numbers of each; at eight three enlarged to every two infantile; and at nine the infantile præcordia gave place to the "enlarged præcordia" of the second half of childhood,—*i.e*, beginning with the sixth year the "infantile præcordia" begins to disappear, until at nine years all cases are uniformly enlarged. During this period, therefore, the results of cardiac percussion must often be equivocal. In children between eight years and puberty the præcordia invariably differs widely from that of the first half of childhood and adult life. The upper border is generally higher, often as high as the second interspace (occasionally the second rib). The right border, instead of following the left sternal margin, is curved and meets liver dulness outside the right sternal edge from one and a quarter to one and a half inches from the median line. The præcordia then resembles Weil's plate, somewhat similar to that of pericardial effusion in the adult.

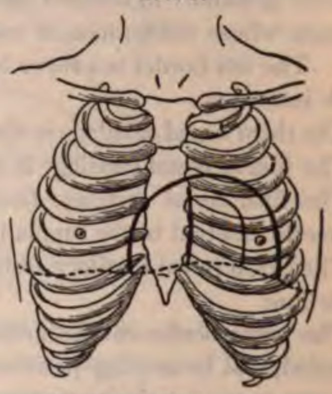
Weil's[36, 37] outlines for relative dulness are as follows. Beginning in the second interspace, it curves to the left, frequently transgressing the left nipple line. The right border leaves the left sternal margin, reaches its right border at the fourth rib, and from the fourth to the sixth rib is from one to three centimetres to the right of the sternum. (See Fig. 5.)

FIG 4.



Cardiac dulness. (After Sahli.)

FIG. 5.



Dulness, according to Weil.

Sahli's landmarks (Fig. 4) closely resemble Weil's, except that the angle between the right border below and the liver is more obtuse.

Baginsky follows von Dusch,[44] who emphasizes the exactness of the relative heart dulness as compared with the less accurate findings in adults and with the more variable infantile absolute dulness. Von Dusch's limits are: superior angle in the second interspace (or third costal cartilage) near the sternum; the right border more vertical than the left, passing outside of the right sternal line in the third, fourth, and fifth interspaces; the left border passing through the nipple line, beyond it and the apex beat.

Von Starck,[16] who has given the subject very careful study, reconciles the apparently divergent opinions in the authors cited by finding them correct within limits. In earliest infancy Sahli's results seem most reliable, in mid-childhood Rauchfuss is followed, and in later childhood Weil's contour is admitted. Von Starck's measurements on three hundred healthy children justify three types:

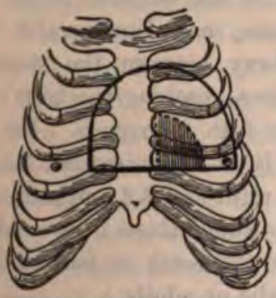
Type I. To the end of the first year.

Type II. End of the first to the sixth year.

Type III. From the sixth to the twelfth year.

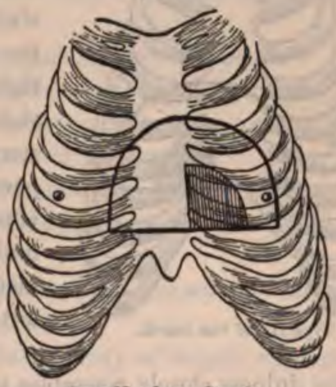
Type I. Under the first year. (See Fig. 6.) The dulness to the right is relatively great, from right ventricle hypertrophy. The relative dulness is astonishingly large, being highest at the sternal end of the second left rib (rarely under the sternum and never to the right thereof). The left border passes convexly to the left, over the third rib at the nipple line; the fourth rib, from 1 to 1.5 centimetres external to the same line; the fifth rib, from 2 to 2.5 centimetres external to the same line. The right border, convex to the right, cuts the right sternal border in the second right interspace, and approaches or coincides with the right parasternal line in the fourth interspace (or upper border of the fifth rib), being farthest from the sternum at the level of the nipple. The heart is bounded below by the fifth rib, determinable since the left lobe of the liver does not reach equally far to the left. Absolute dulness is bounded by the lower border of the third rib and the left sternal line, nearly reaching the nipple line, quadrangular in shape, 3 centimetres high and 3.4 centimetres broad. At the level of the nipple the heart's breadth is from 6.6 to 8 centimetres, 5.1 to 5.6 centimetres being to the left, and 1.6 to 2 centimetres to the right of the sternum.

FIG. 6.



Type I. of von Starck.

FIG. 7.



Type II. of von Starck.

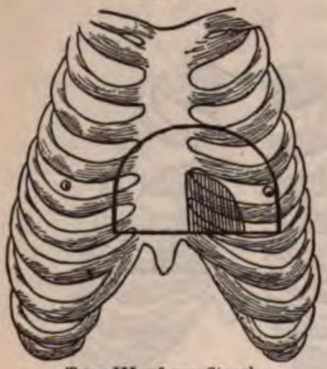
Type II. From the end of the first to the sixth year. (See Fig. 7.) The highest point of relative dulness is at the sternal end of the second interspace (sometimes at the border of the sternum, sometimes nearer the second or the third rib). The left border falls more vertically than in Type I.

It intersects the third left rib in the parasternal line (even external to it); the fourth left rib in the parasternal line; the fifth left rib from 1 to 1.5 centimetres beyond the nipple, or from 1 to 2 centimetres to the left of the apex beat.

The right border is more vertical than in Type I., and tends at the level of the nipple to leave the vertical line and curve inward. It no longer reaches the right parasternal line, although not far from it, and meets the lower border of the fifth rib. The maximum breadth is 10.2 centimetres, 2.9 centimetres to the right and 7.3 centimetres to the left of the sternum. Absolute dulness extends from the upper border of the fourth rib downward 3.6 centimetres, thence outward 4 centimetres.

Type III. From the sixth to the twelfth year. (See Fig. 8.) The upper limit is in the third left interspace. The left border passes over the third rib in the parasternal line, the fourth rib within the right nipple line, the fifth rib at or without the nipple line, and meets the sixth rib from 1 to 1.5 centimetres beyond the apex beat. The right border passes the right border of the sternum in the fourth right interspace, and lies parallel to the sternum midway between the sternal and parasternal lines, reaching the heart's lower border in the fifth interspace. The maximum width is 11.5 centimetres, 3.3 centimetres to the right and 8.2 centimetres to the left. The absolute dulness is as in adults.

Fig. 8.



Type III. of von Starck.

From the thirteenth or fourteenth year relative dulness closely resembles the præcordia or adults.

But few measurements of the heart's lateral dimensions are recorded. Hapke[53] has devoted special attention to the subject, and his figures are detailed, since they differ, as does his division of the time of childhood, from those of von Starck:

Distance from the Sternum.

Ages.	Maximum to the Right. Centimetres.	Maximum to the Left. Centimetres.
One and three-fourths to four years	1.5	6.2
Five to eight years	2.2	6.0
Nine to thirteen years	2.3	6.3

No argument is necessary to prove that pathological changes can be fully appreciated only when the physiological præcordia is accurately demarked. The wide variations in published dimensions result from differences in methods of physical examination, some authors using relative and others absolute dulness, and from variations physiological in youth itself. The data of certain writers are derived from sick children (Wassilewski), while others have purposely examined healthy children (von Starck). From the diagrams and descriptions enumerated the heart generally seems to extend to the right of the sternum at all epochs of infancy and childhood. Von Starck expressly describes and illustrates the presence of this characteristic during the first year. Rotch, in this Cyclopædia, remarks, under the chapter "Pericarditis," that the infantile præcordia has the same contour as the adult, while from the second (or third) year to the ninth or tenth there is frequently some dulness over the lower half of the sternum. Whitney[3] states that "in a child three years old a præcordial dulness reaching the second interspace above and extending an inch or more to the right of the sternum would cause strong suspicion of effusion or enlargement; in a child of eight years such dulness would be normal." His emphasis of the necessity of having regard to the heart's normal contour in the diagnosis of pericarditis or cardiac luxation from pleural effusion, and of cardiac hypertrophy, is vitally important. Whitney suggests as explanation of the occasional enlargement of the heart in adolescence that a seeming hypertrophy in young boys may be persistence of the "enlarged præcordia" of late childhood.

Summarizing, I offer the following points in reconciliation:

1. The præcordia of infancy and childhood varies more than in adults.
2. When observers of renown and accuracy differ, it is well to consider whether all do not represent at least a part of the truth.
3. The heart is located higher in infants than in adults, though that part of the diaphragm which supports the heart may vary in height. Occasionally the heart may be horizontal.
4. In early childhood the heart is relatively and absolutely large, since the viscus is closely apposed to the chest-wall.
5. The heart's subsequent decrease in size may be largely apparent, due to increase of the transverse thoracic diameter.
6. The apex beat does not correspond with the true apex, which is hidden beneath the lung, and which consists of the right ventricle. It is often indeterminable in earliest life; it is located beyond the nipple line in early childhood, but in or within that line in later childhood, and is most frequently seen in the fourth or the fifth interspace.
7. The relative dulness is determined by light palpatory percussion. Errors are frequent from percussion of the sternum, which, as in adults, tends to vibrate as a whole.
8. The weight of evidence inclines to the view that in all epochs of infancy and childhood the heart is found to the right of the sternum.
9. The three types of von Starck best unify the divergent findings of many indisputably accurate clinicians, and coincide with the personal experience and investigations of the collaborator.

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FUNCTIONAL DISORDERS OF THE HEART.

BY J. M. DA COSTA, M.D., LL.D.

FUNCTIONAL disorders of the heart occur in children from exactly the same causes as in adults. But they are not such common affections, because some of the causes are rarely or never present, as, for instance, sexual aberrations, hysteria, the abuse of tobacco, of alcohol, of coffee. Functional disorders of the heart in childhood are usually owing to gastro-intestinal irritations, to worms, to teething; to anæmia, to great impressibility of the nervous system, to lithæmia, or to the action of certain drugs, such as digitalis and opium. In point of frequency the disorders due to gastro-intestinal irritations stand first.

The *symptoms* presented by functional cardiac disorders of childhood are the same as in adults, especially palpitation, shortness of breath, irregular action, and uneasiness or pain. A child, fortunately, is unconscious or ignorant that it has a heart; hence the mental disquietude or distress, the fear engendered by watching the disturbed action, has no counterpart in the manifestations of the case. The palpitation, as in adults, comes on at uncertain times, and is sometimes distinctly provoked by violent emotion or by exercise unsuited to tender years, as by walks too long. The shortness of breath, or, more strictly speaking, the rapidity of breathing, bears a relation to the palpitation. But it is not excessive; and, as the respiration in infants and young children is normally far more variable than in adults, the fluctuations in the breathing are much more marked. The irregular action of the heart, its intermission, its perverted rhythm, its slow beats followed by beats hurrying to make up for the delay, are the most characteristic features of the functional cardiac disorders of childhood. Yet the degree of the disturbance has not the same value attached to it as in adults; for up to about the seventh year the heart's action is often of unequal strength and rhythm, prone to be irregular in the healthiest children during sleep, the pulse being less full during the act of inspiration, and, indeed, at all times greatly influenced by the acts of breathing; but when the irregularity is marked and persistent during the waking hours and during quiet breathing it bespeaks a cardiac disorder, except in those instances in which, joined to other signs of a cerebral malady, it points to meningeal disease. The heart-beats and the pulse-beats do not always correspond. In the irregularly acting heart of childhood it is not uncommon—quite as common, indeed, as in adults—to note more heart-beats than pulse-beats. Of the symptoms of functional affection of the heart mentioned, pain is the least conspicuous; it is much rarer than in adults; uneasy cardiac sensations, too, are seldom to be found. In some instances pain in the region of the heart, without disturbance of rhythm, may occur in paroxysms of reflex origin that are due to the irritation of adenoid vegetations in the vault of the pharynx.[1]

The physical signs of the disturbed action of the heart in early life are, in the main, the same as later,—increased impulse, normal percussion-dulness, distinct second sound, and first sound either weak and short or sharp and valvular. The outlines of the percussion-dulness are difficult to determine with accuracy. Functional murmurs and those of blood-origin are, in my experience, of great infrequency.

In the diagnosis of functional disorder of the heart, and in the appreciation of the symptoms of irregular action which this exhibits, we have first to make sure that the manifestations of disturbed function do not exist in connection with organic cardiac disease. This we do mainly by noting the physical signs furnished by the impulse of the heart and by auscultation, and, though to a less degree, by percussion. At one time most cases of irregular heart-action in children were supposed to be due to a cerebral lesion, especially to tubercular meningitis. But we now know that, in the absence of severe head-pain, fever, and other symptoms, such a conclusion is unwarranted. Irregular heart-action without the presence of an endocarditis or a pericarditis is a frequent symptom of infectious fevers and other infectious diseases. It is seen in scarlet fever, in measles, in typhoid fever, in diphtheria, and in pneumonia. In all of these it is most common when the acute symptoms are over, and thus belongs to a late period of the disease. There are, however, exceptions. Steffen[2] details a case of irregular heart at the beginning of scarlet fever. It may occur early or at the height of typhoid fever, and wrongly be thought to bespeak a meningitis; it may precede the crisis of pneumonia. While very common in diphtheria, it is, as the observations of Veronese[3] prove, very unusual until the disease is subsiding, or in convalescence. I have often noticed the same in influenza.

Turning aside from the instances of functional disturbances of the heart in connection with or following acute maladies to the common groups associated with causes that act more slowly, we find the irregular heart of anæmia, which, besides its physical manifestations, betrays itself not simply in the appearance, but also in headache, fretfulness, and sleeplessness. In anæmic, rapidly growing children there may be a disproportion between the size of the heart and the width of the arteries,[1] a state from which hypertrophy of the heart may result.[2] When the disturbance is lithæmic, as it occasionally is in children, the condition is generally inherited. Besides the history, pain in the joints and deposits of urates in the urine give a clue to the irregular cardiac action.

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[1] Wilson, in Starr's American Text-Book of the Diseases of Children.

[2] Klinik der Kinderkrankheiten, Bd. iii. S. 21, 1888.

[3] Die postdiphtheritische Herzlähmung, Wien. klin. Wochenschr, 1893.

Concerning the irregular heart produced by drugs, we find that quinine, digitalis, and opium are the ones to which the morbid state is most commonly due. But this is a rare form of the cardiac malady in children. The most common cause, after all, is some digestive disturbance, disorder of the stomach and bowels, or the intestinal irritation consequent upon worms; and in point of diagnosis we should be always on the lookout for a cause of this kind. Constipation, too, is a more frequent source of the functional heart disorder than is generally supposed, and this may give rise to an auto-intoxication that greatly disturbs the cardiac rhythm. In such a case, reported by Heubner,[3] large quantities of acetone were also found in the urine. Convulsions or comatose symptoms may coexist.

There is yet another form of irregular action of the heart which is of all the most peculiar and has not received the attention it deserves,—a form in which the disturbed rhythm seems to constitute the whole malady; at all events, there is neither anæmia nor lithæmia, nor gastric nor intestinal disorder, nor are there worms, nor is, indeed, anything to be found except the irregular heart. These cases may be called idiopathic. I have seen them both in boys and in girls, in boys more frequently than in girls, and the children are often ruddy, and appear typically healthy except in their circulation. The heart's action is at times preternaturally slow, and in the sixties, or even the fifties. Intermissions are common, or a series of small beats followed by fuller strokes is noticed. The first sound is apt to be somewhat defective; the organ is always very impressible. It exhibits in the most marked manner the influence of the respiratory acts, especially in deep breathing, and becomes very irregular if the breath be held. Medicines depress the heart quickly. I have known quinine in moderate doses to send the pulse to forty-four without rendering the rhythm more regular. On the other hand, during any febrile state the first effect—and, indeed, a lasting effect until the temperature declines—is the disappearance of the irregularity in the accelerated pulse. The age at which the changed rhythm shows itself is from three to six years; it is very rarely found in infants. It may be intensified by the disturbance of dentition, but it neither appears nor disappears with dentition. I have encountered the affection in children with impressible nervous systems; in one case there was considerable twitching of the muscles of the face, yet the cardiac disorder continued when this stopped. It is, indeed, more common in excitable children, but I have also met with it in those of stolid temperament.

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[1] Kreyssig, Die Krankheiten des Herzens.

[2] Hauner, Beiträge zur Pädiatrik, Bd. i. S. 147, 1863.

[3] Zeitschrift für klinische Medicin, 1894, S. 496.

It seems to run in families. Thus, I have watched two brothers, children of a mother with an extremely slow heart, who both are typical instances of the malady under consideration. In the eldest, now twenty-nine years of age, the irregularity was first distinctly noticed at the age of six. He has had an excellent digestion; teething did not specially influence the pulse, which has been always rather accelerated, and from his sixth year on never free from intermittency; occasionally, too, it presented halting rather than arrested beats. During the first stimulus of any febrile attack the pulse invariably became regular. The irregularity has lessened much in the last years, but it has not disappeared; the beats are not quite equal, some are smaller than others, and more beats can be counted over the heart than at the pulse. There is no murmur, no enlargement. The pulse some years ago was eighty-four when quiet, the temperature normal; the respirations showed nothing peculiar; the intermissions were very distinct after strong breathing. The heart's action is now eighty-two; it does not intermit. While at college he became aware that he could row with force for a time only, and could not run long, yet he could swim for a mile. The cardiac condition does not now interfere with exercise. He can play tennis for a long period, or cycle, without noticing any disturbance. The brother, two years younger, had similar symptoms, but the changed rhythm showed itself especially in extreme slowness.

Neither of this kind of cases, nor of any one of the whole functional group, is the pathology more certain than it is of the functional cardiac maladies of adults. There may be histological and chemical changes, but they have not been detected. The affection may have its starting-point in a weak muscle, or, what is vastly more probable, in the nervous system. The suddenness with which it often appears and its disappearance without traces are greatly in favor of this view. But the exact seat of the nervous disturbance it is not always possible to make out. In some cases, as in those due to gastric or intestinal affections or to intestinal parasites, the disorder of the cardiac nerves is clearly reflex; in others the irritation points to the cervical portion of the spinal cord or to the cervical sympathetic; only few instances indicate disorder of the cerebral centres. In some it is conceivable that the nervous mechanism within the heart itself is at fault. The idiopathic functional disorder, the impressible heart described, may be of this character, though it is more properly explained by localized disturbances of the centres in the medulla, or, yet more likely, of the cardiac centre in the cortex of the brain.[1] Heubner, in commenting on this form of cardiac disorder to which I have called attention, attributes it to a derangement of the pneumogastric. He also holds that in some of the other forms, as in those associated with infectious maladies, or where an auto-intoxication is possible, as in obstinate constipation, the irregular heart-action may be due to a poison acting on the heart nerve-centres or directly on the heart-muscle.

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[1] Loc. cit.

The prognosis of the functional disorder is a favorable one: the cause being removed, the malady ceases. This is certainly true of the affection as seen in consequence of gastro-intestinal maladies and of anæmia, except in the instances of narrow vessels already mentioned. It is also true that the process is a slower one in children who have inherited or acquired lithæmia, and in the disturbed heart after fevers or from malaria. The most tedious cases are those of the idiopathic disorder, or "impressible heart." They last for years. I have watched cases from early childhood to manhood where irregularity still exists; on the other hand, I have seen it gradually disappear after puberty, both in boys and in girls, but not immediately. The most important question connected with the whole matter is, whether in this or any other form of the functional affection organic disease ever follows. I am not aware of any records on the subject, and can, therefore, only say from personal experience that I have not met with a single instance. Dilatation of the heart would be the condition most likely to occur; but I have never seen it happen. A certain amount of readily disturbed action and of breathlessness on exertion is, however, apt to remain. Yet I have known a boy who had for years presented a marked instance of the impressible heart become a champion runner,—it is true, only for dashes. Another interesting question also here claims solution. As there is so strong a nervous element in many instances of the cardiac disorder, is this the forerunner of chorea, epilepsy, or other kinds of neurotic ailment? Appealing again to personal experience, I have never traced any such connection. In what appear like associate cases the nervous malady is the first to show itself.

In the treatment of the functional cardiac disorder of childhood it is evident that we must aim chiefly at removing the cause of the affection. A number of cases will be found to yield to careful attention to diet and to correcting digestive disorders, to allaying vomiting, and to removing constipation. It is especially important that large amounts of food should not be taken at one time. A strict watch for worms will readily determine whether vermifuges should be employed. The lithæmic state will require the same directions, especially as to diet, as gout or lithæmia in adults. The anæmic heart is benefited by a liberal meat diet, by iron, or by small doses of arsenic long continued, and by life in the open air and sufficient sleep. Moderate exercise suits all cases, and I have seen even benefit from making the little patients run certain distances daily, carefully graduated to suit their strength and slowly increased. Light gymnastics are also serviceable, steady, graded day by day, stopped when tiredness comes on, and not limited to exercise for the arms, but made to include movements for the legs and resistance exercises. Nor need out-door sports, if not abused, be interdicted, except it be rowing. From sea-bathing, provided the bath be not too long and the skin be well rubbed afterwards, I have seen the happiest effects. Thus, in a young girl with the idiopathic irregular heart above described, whose case I watched for years, a cure was brought about by three weeks of sea-bathing. She had taken digitalis off and on for many months, always with temporary benefit, but not with permanent result; the pulse, after the cessation of the treatment, returned to upward of ninety, and intermitted from every sixth to every fourteenth beat. Her general health was excellent, and she lived a great deal in the country, amid the best surroundings. It was a disappointment that the establishment of menstruation did not make a change in her cardiac condition. After the course of sea-bathing the irregularity ceased; there remained for a time a little hurrying of the heart subsequent to a number of beats, and now, some years afterwards, the heart is always steady and not above eighty.

Greater stress has been laid, in the management of the functional disorder, upon remedies which remove the cause and upon hygienic means than upon so-called heart-tonics. Indeed, these, I think, ought, as a rule, to be used only for temporary purposes. When they are called for, digitalis will be found to be the most trustworthy.

Should it be decided to give it with a view to its more sustained action, it is best administered in courses of about a month each, with an interval of ten days between each course. This treatment it is my habit to direct to be carried out for from four to six months, and then to resume it only as may be necessary. The preparation usually employed is the tincture: the dose is from three to five drops for a child six years of age. In many instances a morning and an evening dose are sufficient, and, after the heart becomes more regular, a single evening dose will keep it so. This dose may be stopped at about the end of the fourth week of treatment, when generally the influence of the remedy is perceptible and the pulse for the time being steady. But it does not at once so remain, and further courses will be required. Belladonna is also a drug of use in the functional disorder, either as a temporary substitute for digitalis or in combination with it. From strophanthus or chloride of barium, too, good results may be at times obtained.

DISEASES OF THE SPLEEN AND THEIR OPERATIVE TREATMENT.

BY LOUIS McLANE TIFFANY, M.D.

WHILE a period of not many years (now about ten) has elapsed since the first volumes of the Cyclopædia were issued, the surgery of the spleen has undergone changes—for the better, undoubtedly—resulting directly from increased knowledge of the subject. During the last few years abdominal surgery has made great advances and abdominal section has become frequent. While such section may have been done for disease of one organ, necessarily other parts within the peritoneal cavity have been investigated, and our knowledge of the spleen has been greatly increased by such procedures.

SUPPURATIVE SPLENITIS AND PERISPLENITIS. ABSCESS IN AND AROUND THE SPLEEN.

Suppuration in and around the spleen is probably always secondary to some other trouble, usually septic. In the spleen suppuration is the result of metastatic infarction. It may be that the inflammation is the direct result of traumatism, either penetrating or not. In the former case infection will have been directly inoculated; in the second case infection is carried through the circulation. Occasionally inflammation extends from adjacent organs. Points of inflammation from infarction occurring may suppurate and coalesce, thus producing a large abscess. Adhesive inflammation may be set up, and the pus make its way in one direction or another, perhaps into an adjacent organ, perhaps into the peritoneal or the pleural cavity. The existence of a diffuse suppurative splenitis is questionable. The diagnosis of splenic abscess is difficult, especially if the focus of suppuration be entirely within the parenchyma; a dull, heavy weight without acute pain is generally present. Pain is to be expected only when inflammation shall have extended so as to involve the peritoneal coat. In that case the symptoms of acute peritonitis may be recognized. An abscess, of course, being present, acute sepsis is always possible. Fluctuation is to be expected only when the abscess is very large. Pain in the left shoulder is referred to by writers. The constitutional symptoms of suppuration will be present, probably, but if the splenic inflammation is secondary to some other serious disorder they may escape notice. It is probable that a diagnosis will not be made early in the course of the disease, if at all.

Treatment.—Aspiration of the spleen in hopes of evacuating abscess is not to be thought of, the reason being that the diagnosis is not clear, and a puncture of the spleen may give rise to fatal hemorrhage. If fluctuation be present, of course there need be no hesitation about making an opening. Such a condition of affairs is so exceptional that it can be accepted without hesitation that splenic inflammation should be treated by abdominal section as a much safer proceeding than aspiration. If the spleen be enlarged and project below the ribs there will be no difficulty about making an incision upon it. Should it not project below the ribs, then an incision parallel to the cartilage of the ribs and of length sufficient to expose the organ is called for. A long incision is safer than a short one, since, the organ being then better exposed, handling and traction, which are fraught with danger, become unnecessary. The organ should be exposed, surrounded with gauze packing, and the abscess opened and drained. If the whole organ is destroyed or involved in the inflammation, splenectomy may be called for. Theoretically, abscess of the spleen should be opened through the chest, as is an abscess of the liver, but that a diagnosis will be so accurate as to justify such operation is more than problematical; however, it might happen.

GANGRENE OF THE SPLEEN.

Gangrene of the spleen would occur as the result of obstruction to the circulation, would be rapid in its onset, and the general symptoms would be those of acute sepsis. Should a diagnosis be made, there will be but one treatment,—immediate extirpation.

WOUNDS OF THE SPLEEN. GUNSHOT AND STAB WOUNDS.

From the position of the spleen, lying in the body, as it does, well protected, a wound of the spleen alone is not usual. Generally other organs are involved,—from behind, the kidney and lung; from above and the outer side, the chest; from in front, the alimentary canal. Twice it has been my fortune to see an injury of the spleen and kidney from gunshot wounds. A number of cases are reported of gunshot injury of the spleen followed by recovery. In these cases the spleen has protruded through the wound, and hernia of the organ has taken place. Such a condition of affairs seems not to be fatal. Most of the cases of primary spleen injury which have recovered belong to this category. Unquestionably the spleen has been wounded again and again, and death has followed without recognition of the organ injured. Again, a certain number of spleen wounds have been received and not recognized, the scars having been found later,—years afterwards, perhaps. Stab wounds of the spleen may be considered with gunshot wounds. The general direction of the vulnerating force is to be considered. Unless there be prolapsus of the spleen through the wound, or a very large wound which permits inspection, a diagnosis is uncertain. Internal hemorrhage is the symptom upon which the most reliance is to be placed, considered with the direction from which the violence comes. The danger to life is from hemorrhage, and justifies abdominal section. Peritonitis and septicæmia may occur.

Treatment.—Of late years it has been found expedient to open the abdomen for penetrating wounds,—gunshot or stab wounds,—and splenic wounds are to be placed in this category. The spleen having been well exposed by abdominal section, arrest of hemorrhage is to be brought about by through-and-through stitching of the organ, after which the peritoneal cavity is cleaned and closed in the usual way. Should gauze packing and suture be insufficient to arrest hemorrhage, extirpation of the spleen is called for. The amount of gauze packing will vary with the injury. The necessity for gauze packing after injury of the abdominal organs has become more and more apparent, and the spleen lends itself readily to such treatment.

RUPTURE OF THE SPLEEN.

The spleen is more friable than other organs of the abdomen, and may be injured by blows and falls. In the absence of a wound, the symptoms of intra-peritoneal hemorrhage appearing, peritoneal section is called for and suture and gauze packing are to be made use of, which failing, removal of the spleen is indicated. In other words, rupture is to be considered as are other splenic wounds. The spleen has been ruptured in several places and torn quite in two, in which case the symptoms of hemorrhage will come on very rapidly and call for immediate action at the hands of the surgeon.

TUMOR OF THE SPLEEN.

Tumors of the spleen are rare. Primary cancer or sarcoma is very rare, and the diagnosis uncertain. If recognized sufficiently early, extirpation is proper. In not a few cases tuberculosis of the spleen is recognized as existing when the same affection is present in other abdominal organs. It is seen in peritoneal miliary tuberculosis. Burke has recorded a case of splenectomy for tuberculosis. For sarcoma extirpation has been done a few times. Syphilis of the spleen in the form of gumma or diffused hyperplasia is noted as a lesion of inherited syphilis. The usual evidences are enlargement, dulness on percussion, and protrusion below the cartilage of the ribs. Other evidences of inherited syphilis will be present, and special treatment directed towards the spleen alone is not called for.

Hydatid cysts of the spleen have been noted; they do not differ from hydatid cysts elsewhere, and so call for no extended notice. Probably it would be difficult to make a diagnosis between hydatid of the spleen and hydatid of the kidney, and either might be confounded with a cystic tumor of the ovary. Hypertrophy of the spleen probably is the most frequent abnormality noted, and it may occur without changes in the lymph-glands and without the presence of malarial organisms. In the former case it is denominated simple hypertrophy, and when present during the course of malarial infection it is spoken of as malarial hypertrophy. In leukæmia the spleen may become enormous. The diagnosis of leukæmic spleen is made by blood examination, and the same may be said of malarial spleen. In the absence of leukæmia and malarial infection an enlarged spleen may be considered as simply hypertrophic. The size of an hypertrophied spleen may be great; it may extend into the pelvis or fill much of the abdomen.

The indications for removal of the spleen in childhood do not differ from the indications present in the adult, nor does the examination of the blood in the one case differ from that in the other. Removal for leukæmia is followed by a very great mortality, and is contra-indicated; the splenic enlargement is only one symptom of many, and should not be treated as a disease. On the other hand, in malarial hypertrophy, or in simple hypertrophy, malaria not being present, splenectomy can be undertaken with advantage. The technique of the operation in a child is the same as with the adult, save only that the loss of blood and the shock are to be most carefully guarded against. It would be well, therefore, to bind the child's extremities with cotton to preserve the bodily temperature, and to operate in the Trendelenburg position. The statistics of the operation for various causes are well considered in a paper by Richard Douglas, M.D., in the Southern Surgical and Gynæcological Transactions for 1895.

ADENITIS.

By CHRISTIAN FENGER, M.D.

THE lymph-glands or lymph-nodes located in the course of the lymphatic vessels are elastic, round, flattened, pale-red bodies, from two to thirty millimetres in diameter, having afferent and efferent lymph-vessels, the latter less numerous but wider. The lymph-glands are located in tracts of adipose tissue, part of which forms around groups of glands, the so-called adipose capsule. Each gland has a fibrous capsule with septa, including a parenchyma, in which can be distinguished a cortical substance with a globular arrangement and a medullary substance with a trabecular arrangement. The parenchyma consists of a fine vascular reticulum filled with lymph-cells. Under normal conditions the volume of lymph-glands is largest in children, and decreases in the course of years.

Adenitis—that is, inflammation of the lymph-glands—will be limited in this article to the diseases caused by the pus microbes. Tuberculosis, venereal diseases, pseudo-leukæmia, leucocythæmia, and tumors of the lymph-glands have been considered elsewhere.

We do not know that all inflammation or swelling of lymph-glands is caused by microbes, but recent bacteriological investigations point in that direction, and a so-called simple, non-microbic adenitis is gradually becoming a thing of the past.

The action of the microbes on lymph-glands has been studied experimentally by Halban.[1] The staphylococcus aureus, on subcutaneous injection into the peripheral end of an extremity, appeared in the corresponding lymph-gland in a few minutes; introduced into a deep punctured wound, in an hour; and rubbed into a subcutaneous wound, in four hours.

The time between invasion and finding of the microbes in the lymph-glands varies with the form of the bacteria. The bacteria least affected by the glands appear early, while the bacteria energetically destroyed by the glands are not found until later, when they gain the upper hand by coming in a continual stream from the place of invasion. The lymph-glands possess powerful bactericidal properties in two of their elements,—namely, the white cells (phagocytosis) and the alexins, the bactericidal substances in blood-serum and lymph, also a product of the white cells. It is due to these enormous conglomerations of white cells inserted as lymph-glands in the channels of absorption that the bacteria and other formed elements—namely, pigment and colored particles—are not only held back, as in a filter, but are fought against and destroyed by the vital forces in the lymph-cells. Pathogenic bacteria are found in the lymph-glands later and in smaller numbers than non-pathogenic bacteria. The former seem to be destroyed much more energetically than the latter.

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[1] Bericht über die Verhandlung der Deutschen Gesellschaft für Chirurgie, XXVI. Congress; Centralblatt für Chirurgie, Nr. 28, 1897.

The typical behavior of the bacteria in the glands is as follows. The microbes disappear from the glands after one or two hours. After a latent stage of from five to seven hours they reappear, to disappear again later. This manœuvre—appearance and disappearance—may be repeated several times. Finally the non-pathogenic microbes permanently disappear; the pathogenic microbes come to stay, increase, destroy the gland, and eventually the individual. Through the continued absorption from the place of infection the bacteria gain the upper hand for a time, during which they can be found in the gland. This explains the remittent character of the septic fever.

The lymph-gland reacts by swelling and increase of the lymphoid cells. After ten days of infection with staphylococcus aureus the gland is enlarged from twenty to twenty-five times its normal size without, as yet, any visible abscesses being found in it. Direct entrance from the wound into the circulation probably does not take place; the microbes must pass through the lymph-glands that form a barrier, which is most often at least temporarily effectual. The amputation of legs of rabbits infected with anthrax culture from two to two and a half hours after infection has saved the animals. The intermittent invasion of the glands from a point of infection which continues to produce bacteria for a considerable period of time explains why disinfection of the place of invasion can save life and cause the disappearance of the adenitis as long as the lymph-glands are able to destroy the microbes.

Acute Adenitis (*Adenitis Acuta Simplex*).—Clinically we distinguish between swelling without suppuration and swelling terminating in suppuration. Besides the pus microbes, there have been found in the lymph-glands the typhoid bacillus, diphtheria bacillus, colon bacillus spirillæ from the mouth or hollow teeth, and the gonococcus. It is likely that future investigations in this as yet only slightly cultivated field may reveal many more.

The poison, whether microbic or non-microbic, rarely introduced through a direct wound of the gland, has usually a distant place of invasion. Lymphangitis is often absent, and when present is rarely followed by adenitis, as the microbes have spent their force before reaching the gland.

The place of invasion is either the skin or the mucous membranes. On the skin a wound or abrasion of any kind, a burn or diseases of the skin destroying the epidermis, erysipelas, excoriations around the anus, mamma, or genital organs, may be named as instances. Inflammations in deeper tissues, such as osteomyelitis or infections of the joints, rarely cause adenitis. Invasion of the mucous membranes may take place in any portion of the intestinal, respiratory, or genito-urinary tract.

Localization of microbes in lymph-glands through the blood-current is rare, but is believed to exist when acute infectious diseases, such as typhoid fever, are followed by suppuration of the cervical or inguinal glands, for instance, with no visible peripheral lesion in the territory of the glands affected. It is likely, however, that a minute lesion at the place of invasion has been overlooked, or that microbes have passed through the mucous membranes without appreciable lesion.

In the stage of swelling the gland is enlarged, round or flattened, hard, reddish or brownish, with small ecchymoses. The fluid from the cut surface contains, besides possible microbes, lymph-corpuscles and swollen epithelioid cells. The swollen gland is difficult to inject; it has lost to some degree its permeability for liquid, because the lymph sinuses and cavernous substances are filled with lymph-cells, fibrin, and débris of disintegrated leucocytes. The fibres of the stroma are thickened and the capillaries are dilated and show the ordinary signs of inflammation. In this stage the inflammation may terminate in absorption without suppuration, leaving a thickening of the stroma, or connective tissue formation, leading to sclerosis or atrophy of the gland.

If suppuration takes place the gland becomes soft and friable. Small multiple, disseminated, gray or yellow foci form, which may unite into one abscess cavity after destruction of the stroma of the gland. The fibrous capsule of the gland resists for a time and becomes thickened, and the periglandular adipose tissue becomes the seat of beginning inflammation. The abscess may remain intra-glandular, but in the more rapid infective cases the fibrous capsule is destroyed in places and suppuration takes place in the periglandular adipose tissue (suppurative peri-adenitis), and pus forms here in the irregular spaces between the lymph-glands. Thus, we may have separate abscess cavities in the glands and outside of the glands, or communicating intra- and periglandular abscess cavities. It is evident that it is almost impossible to disinfect and drain effectually irregular and sinuous cavities of this kind. In the most acute cases the glands as well as the periglandular tissue are rapidly destroyed, and the whole territory of glands, with the adipose capsule, may be transformed into an abscess cavity with rapid extension to the skin, which sometimes may be partially destroyed by necrosis.

The fate of the glands which have been the seat of suppuration depends upon the degree of the latter. In lighter cases, possible resolution and restitution of function may take place; in severe cases, destruction and atrophy.

The practical side of this question—namely, restoration of lymph-circulation and lymph-gland function in the territory affected—has been studied by Bayer.[1]

When a territory of lymph-glands is destroyed by suppuration or removed by extirpation, reparation takes place in the following manner. In this process the adipose capsule of a gland or group of glands plays a very conspicuous and hitherto unobserved part. The adipose tissue becomes vascular; almost every fat-cell is surrounded by a capillary. Around the capillary is seen a row of epithelial cells signifying that a perivascular lymph-space has formed around it. The fat-cells proliferate, and in the connective tissue stroma numerous lymph-cells appear. The whole territory becomes partially granulation or embryonal tissue. The fate of the embryonal tissue may be that of terminating either in a cicatrix with destruction of function or in the formation of new lymph-glands from groups of lymphoid cells, and of new lymph-vessels from pre-existing and newly formed lymph-spaces, forming afferent and efferent vessels for the new lymph-glands. Function is thus restored. All this takes place in the adipose tissue surrounding the destroyed territory of glands. It is, therefore, important that adipose tissue should be left during extirpation of the glands. This quiet process of repair may be disturbed by infection; it is thus important that operation wounds should heal by first intention.

The symptoms of non-suppurative adenitis are the following. There is tenderness over the swollen gland or group of glands, if it extends from one gland to the whole chain. The swollen gland is first hard; movable as long as no peri-adenitis has taken place; later immovable, but the covering skin remains normal. Lymphangitis or fever may be present or absent. In time, after days or weeks, the pain and tenderness gradually disappear; the gland either disappears after a time or remains enlarged.

In suppurative adenitis more severe local and general symptoms set in. The swelling and pain increase, often accompanied by fever and rigor. The gland becomes immovable; on the top of the swelling there are softness and fluctuation, œdema of the skin, and redness. The whole group of glands presents a mass in which the single glands form multiple abscesses in different places. Suppuration sometimes develops rapidly in a few days, but usually not until after one or two weeks. The intra-glandular abscess is small, and gives only a little serous pus when incised, whilst the periglandular abscess gives a larger quantity. The most extensive abscesses, however, are formed by the phlegmonous inflammation with acute destruction of all the glands in the group, together with the periglandular adipose tissue. The periglandular abscess is sinuous and irregular, and thus difficult to drain from a single opening. In its cavity there is an entrance to one or more small intra-glandular abscesses, which causes still more difficulty in drainage. From the remainder of the living tissue in the suppurating glands luxuriant granulation tissue may grow out and protrude through the incision opening. The scraping away of this granulation tissue is likely to leave portions of the gland containing suppurating foci, which prevent the now fistulous tract from closing, and the suppuration may go on indefinitely until the entire gland is either destroyed or removed.

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[1] Langenbeck's Archiv, Bd. xlix., Hft. 3, S. 647.

In grave septic infections, or in the infectious fevers, such as scarlatina, the destructive inflammation in the glands may cause, on the one hand, rapid, sometimes gangrenous, destruction of the skin, or, on the other hand, downward septic destruction of the intermuscular spaces with formation of deep abscesses which, when not opened in time, long before any signs of fluctuation or redness are seen may get beyond control. On the neck, for instance, the deep-seated inflammation may pass down in the mediastinum or destroy the sheaths of the large vessels, with the concomitant dangers of either hemorrhage from opened arteries or veins, or septic thrombosis of the latter. If the patient escapes death from sepsis, the loss of strength is great and the recovery slow. In the more acute forms of rapidly fatal sepsis we may, however, find only a moderate involvement of the glands and comparatively slight symptoms at the place of invasion.

The diagnosis is usually easy, especially in the superficial lymph-glands, from the location (neck, axilla, groin), from the history of the case, giving a lesion or disease at the place of invasion, and from preceding or present lymphangitis. Suppuration in deep-seated glands is more difficult to recognize, and aspiratory puncture may aid in the diagnosis.

As there is always a place of invasion at a distance from the glands, as it takes a certain time before the microbes reach the glands, and a longer time before the glands are overpowered by repeated invasion, the adenitis can be prevented or aborted by treatment at the place of invasion, protection or disinfection of wounds or granulating surfaces (chronic ulcers), adequate treatment of skin diseases, lymphangitis, and abscesses, and treatment of the diseases of the accessible mucous membranes. Warm baths, rest in bed, and the appropriate constitutional treatment aid the organism in its struggle against the microbes.

Before suppuration is manifest a great many local applications over the glands have been employed: mercurial or iodine ointments, tincture of iodine, tincture of iodine and potassium iodide (Churchill's tincture), hot fomentations in the shape of wet aseptic or antiseptic dressings covered with rubber tissue or oiled silk, compression bandages if tolerated. Intra-glandular hypodermic injections of carbolic acid, three per cent., tincture of iodine, or bichloride of mercury may also be used.

When suppuration has become manifest the pus has been evacuated by incision or by small multiple punctures. Through these, in the more chronic forms, has been injected one per cent. nitrate of silver (Lang's method) or one per cent. benzoate of mercury (Welander's method). Drainage and washing out with various antiseptic fluids, such as carbolic acid, bichloride of mercury, tincture of iodine, iodoform, ether, and turpentine, curetting of fistulous tracts and granulating cavities with a view to the removal of the débris of suppurating glands, have also been practised.

In more obstinate cases, or when demanded by grave constitutional symptoms, free opening of the whole territory is indicated with thorough curettement of débris, suppurating glands, and fistulous tracts, followed by disinfection and packing.

If by this treatment it is still impossible to overcome the multiple foci of infection, the last resort is radical operation,—that is, extirpation of the whole territory of glands, which will be described under chronic adenitis.

Chronic Adenitis (*Adenitis Chronica Simplex*).—Up to very recent times "chronic hypertrophy, chronic swelling, chronic engorgement" of lymph-glands has not been sufficiently recognized etiologically, and has consequently not been differentiated from tuberculosis, syphilis, "lymphadenoma," pseudo-leukæmia, or neoplasms. The so-called hypertrophy of the lymph-glands of the neck is tuberculosis in ninety-five per cent. of the cases (Riedel).[1] Malignant lymphomata occasionally develop, but in a small percentage of the cases simple chronic inflammation exists, and here we can, in almost every case, find a place of initial invasion. The disease at the place of invasion causes intermittent or permanent swelling of the glands. Microbes once imported into the gland may remain dormant after the disease at the place of invasion has come to an end, occasionally to be aroused. If the disease persists, however, there is a constant or remittent invasion of microbes (ulcers, skin diseases, repeated attacks of dermatitis, elephantiasis, erysipelas). In twenty-eight out of thirty-two cases of varicose eczemata and ulcers on the legs, the inguinal glands were enlarged (Lejars)[2] and became more painful and more swollen upon over-exertion.

The chronic cervical adenitis formerly ascribed to taking cold is probably most often tuberculosis (Koenig).[3] But among the laboring classes, especially when they do not or cannot observe the ordinary rules of cleanliness, particularly of hands and feet, this neglect or the nature of their occupation affords abundant opportunity for the invasion of microbes. Caries of the teeth, with the subsequent inflammation around the root, is often followed by chronic adenitis of the submaxillary glands, which disappears after the teeth have been treated.

It is probably almost always microbes that cause adenitis, but the etiology has not yet been fully investigated. We know, however, that staphylococcus and streptococcus infection (either with acute origin and chronic course or chronic from the beginning) will cause chronic adenitis, which will either disappear when the invasion ceases, or will remain if it continues or if multiple foci remain in the gland.

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[1] Koenig's Lehrbuch der allgemeinen Chirurgie, Berlin, 1889.

[2] Duplay and Reclus, Traité de Chirurgie, t. i., Paris, 1891.

[3] Deutsche Chirurgie, Lief. 36, 1882, Koenig and Riedel, Die entzundlichen Processe am Hals und die Geschwulste am Hals.

The gland, from the size of a hazel-nut to that of a walnut, is hard; the cut surface reddish brown or violet, with a grayish or yellowish centre; afferent and efferent vessels dilated; the capsule of the gland thickened; the periglandular adipose tissue infiltrated with cells and œdematous, with atrophy of the adipose tissue. The parenchyma of the lymph-glands may gradually disappear, to be replaced by connective tissue or adipose tissue, until finally only small islands of lymph-cell tissue are left in the peripheral portion of the gland. Deposits of cheesy or calcareous matter are probably most often due to tuberculosis, although it is possible that small intra-glandular, non-tuberculous abscesses may have the same form of retrograde metamorphosis. Calcified glands are often found in old people, the same as pigmented glands in coal-miners and workers, where particles are deposited in the perivascular spaces of the cavernous substance of the gland.

By far the greatest practical importance attaches to the cases of staphylococcus and streptococcus infection causing small multiple foci in the glands, sometimes six or eight foci the size of a pin's head in a gland two centimetres in diameter. They are stationary, and have no power to destroy the surrounding gland tissue or perforate the capsule. Thus the process never comes to an end, but may be stationary for years, causing only a little pain on exertion. Such glands are most often found in the inguinal region following chancroid, but may also be caused by non-venereal excoriations and balanitides. They may be more rarely found in the axilla or the neck, and must be carefully differentiated from tuberculosis and syphilis. But antispecific treatment has no effect. Fatigue or exertion causes pain and increased swelling. Every now and then a small abscess comes to the surface and leaves a fistula. This process is repeated every two or three months for years, making the patient an invalid from slight attacks of fever and loss of strength, exposing him to acute invasions through the open fistulas, and to attacks of erysipelas which may lead to deep abscesses or general sepsis. Happily, these grave, obstinate cases are in the minority.

The symptoms are in the main the same as those of simple acute adenitis, but without the pain. The swelling develops slowly or succeeds an acute attack. If more than one gland is affected, they may, after being movable for a time, become united by peri-adenitis and form a nodular cord. The swelling, increased temporarily during each exacerbation, does not disappear or become reduced to its former volume, and small abscesses and fistulæ are formed. As a rare complication must be mentioned chronic œdema in the distal territory of the group of glands. This is most often found in the lower extremity, where it may be permanent and lead to conditions similar to elephantiasis. It is found more rarely in the upper extremity, head, and neck, where it ordinarily disappears after a time.

The final condition of the glands in chronic adenitis varies according to the severity of the disease. In a number of cases they are reduced to normal dimensions when the inflammation at the seat of distal invasion comes to an end. In other cases repeated attacks of suppuration destroy all the infected glands, and finally cicatrization becomes permanent. Further, atrophy, sclerosis, and fibrous or lipomatous transformation often ensue.

What becomes of the gland's function we have seen already in the investigations of Bayer.[1] In the great majority of cases a sufficient number of glands remain, or new glands and lymph-vessels form sufficient to restore function in the territory, with only the rare exceptions already mentioned.

As to diagnosis, we must exclude tuberculosis, syphilis, malignant lymphoma, and neoplasms. A visible place of invasion, smaller size of the glands, limitation to a certain group corresponding to the place of invasion, and excision of a gland for microscopic examination will in most cases give a positive diagnosis.

As to treatment, we must first, if possible, cure the disease at the place of invasion,—skin diseases, ulcers, diseases of the teeth, ingrowing nails, and corns. The treatment by the injection of antiseptic or alterative substances is the same as in acute adenitis. The same may be said of local applications and rest. Methodical compression is more extensively used and probably more effective in chronic suppurative adenitis as an aid to incision and disinfection, as described above. When in obstinate cases of multiple suppuration the minor surgical procedures have been used in vain, there remains, as a final resort, radical operation,—that is, extirpation of the glands. We distinguish between enucleation of one or more glands, leaving the periglandular tissue intact, and complete extirpation of the whole infected group, including the periglandular adipose tissue.

Enucleation of one or more glands without removal of the periglandular tissue possesses the advantage of requiring less extensive operating, and of leaving the periglandular adipose tissue for restoration of function by new formation of lymph-channels and glands. This operation, which is not applicable to malignant tumors, and usually not effective in tuberculosis, because scores of small yet tuberculous glands cannot be enucleated, is applicable in chronic suppurative adenitis, and should probably, as an operation of choice, be attempted before total extirpation is resorted to.

In chronic suppurative adenitis it is especially important to save the surrounding adipose tissue for the re-establishment of function so as to avoid permanent œdema of the corresponding extremity. The cases of this kind first mentioned by Riedel were not cases of tuberculosis, but were all cases of simple chronic adenitis, in which total extirpation was followed by permanent œdema of a severe type, and Riedel came to the conclusion that total extirpation should not be practised in such cases.

The technique of the operation is comparatively simple: incision down to the capsule of the gland, enucleation by blunt instruments, if possible without rupture of the capsule, to avoid unnecessary infection of the wound. More than one gland may be removed through a single large incision over the whole infected territory, or the glands may be removed through multiple smaller incisions. Periglandular abscesses when encountered are curetted and all visible pieces of glands removed. The wound should not be closed, but after disinfection with any of the ordinary agents should be packed with gauze, and the edges of the wound approximated by sutures to a distance of one centimetre. The sutures are tied loosely, so as simply to hold the gauze in place. This, the most perfect method of drainage, should be employed, because the wound of operation is necessarily infected, and primary union is not desired, as subsequent local applications to the wound cavity may be required.

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[1] Loc. cit.

Total extirpation comes into consideration only as a last resort after incision and drainage of abscesses have been tried and have proved ineffectual. The three territories in question are the groin, the axilla, and the neck. Inasmuch as it is permissible in chronic adenitis to leave parts of infected territory for disinfection, if needed, nerves and vessels should always be respected during the total extirpation for this disease. When a suppurating gland or a periglandular abscess is inseparably connected with the wall of the large veins,—namely, the femoral, the axillary, or the deep jugular,—it is preferable to leave a small territory of infected tissue rather than to extirpate the vessel.

In extirpation of the inguinal glands the incision is made, according to the shape of the group, in the direction of the greatest diameter. It is well first to secure the end of the long saphenous vein, thus commencing the dissection from below and working up towards the falciform process and the femoral vein. When Poupart's ligament has been reached, the incision is prolonged outward for removal of the glands on its outer surface, if required. The operation may end here; if not, the glands in the iliac fossa must be removed also. If this, as in rare instances, be the case, we must gain access to the iliac fossa without opening the peritoneum. Division of Poupart's ligament over the vein may be necessary if the glands in or above the cribriform septum are infected, as these glands, when not removed, may cause abscess formation in the prevesical space. The group of iliac glands is located not on the vein, but anterior and external to the external iliac artery, from which they can be isolated *en masse* after incising the abdominal wall parallel to Poupart's ligament, external to the inguinal canal, and pushing the peritoneum upward over the mass. Extirpation of this group of glands when suppurating will prevent the formation of a retro-peritoneal iliac abscess, and thus prevent the serious operations which such an abscess might necessitate, such as resection or trephining of the ilium.

In the axilla an incision is made parallel to and an inch behind the outer border of the pectoralis major muscle, circumscribing possible fistulous openings, down to the surface of the conglomerated group of glands. It is advisable first to find the axillary vein at the distal end of the mass to be removed. By means of careful blunt dissection the vein is now separated from the mass and successive division between ligatures of the vessels traversing the axilla made in the following order:

1. Circumflexa scapulæ.

2. Subscapularis, during the isolation of which we have to secure the subscapular motor nerve, usually one trunk sending branches to the subscapularis muscle, the teres major, and the latissimus dorsi. The nerve is secured by a loop of gauze and dissected out through the glandular mass.

3. The thoracica longa, close to which we must look for the posterior thoracic nerve for the serratus anticus major.

If the pectoralis minor muscle is invaded, it may be divided so as to gain access to the deep portion of the axilla, where the small acromio-thoracic and superior thoracic vessels may be encountered. In this place the anterior thoracic nerves to the pectoralis major and minor are rarely seen. The cutaneous branch of the second intercostal nerve passes transversely through the mass, but as it is an exclusively sensory nerve it is less essential to save it, as its dissection will occupy considerable time.

The mass of glands is now isolated from the remainder of its surroundings, removed, and the wound packed for secondary union.

Partial or total removal of the glands of the neck is rarely if ever called for by simple suppurating adenitis, but usually by mixed infection with tuberculosis. The accessible abscesses must first have been drained, leaving multiple discharging, fistulous openings. If all the glands, superficial as well as deep, are involved from the ear to the clavicle, an incision can be made along the middle of the sterno-cleido-mastoid from the mastoid process to the sternum between the sternal and clavicular portions of the sterno-cleido-mastoid muscle. The external jugular vein is divided between ligatures, the skin and platysma loosened from the outer surface of the sterno-cleido-mastoid, the sternal and clavicular portion of the latter are separated by blunt dissection, and the clavicular portion divided an inch above the clavicle. The middle tendon of the omo-hyoid muscle is lifted from the sheath of the deep jugular vein and divided. The glands along the posterior border of the deep jugular vein are now accessible. So as not to be disturbed by the respiratory movements of a large jugular vein, I prefer to isolate the latter about an inch above the sternum and tie a provisory ligature around it, thereby also guarding against possible air embolism if the vein should be opened during the section later on.

Dissection of the glands and surrounding infiltrated adipose tissue is now commenced from below, and the deep jugular vein laid bare up towards the middle of the neck. Before passing over this point, search must be made for the branch of the spinal accessory nerve to the trapezius muscle. It passes out from under the posterior border of the sterno-cleido-mastoid at about its middle, and it is very common to encounter at this place fistulous openings into suppurating glands, necessitating very careful search and dissection. By pinching the tissues in this territory until the muscle is seen to contract, the nerve can, as a rule, be found, and must then be isolated down to the trapezius muscle and secured by a loop of gauze. The dissection is now continued along the deep jugular until it is isolated up to the posterior belly of the digastric. When the whole outer and posterior surface of the deep jugular vein is free, the mass of glands is easily removed from the posterior muscles of the neck and the subclavicular triangle, the lower outer portion of which may have to be made accessible by an additional transverse division of the skin. In the rare cases in which the glands extend down behind the clavicle, care must be taken not to wound the subclavian vein. In this territory on the right side the thoracic duct may be divided, giving rise to a flow of milky chyle in jets synchronous with the respiratory movements. Ligature or forcipressure will close the duct, and as yet no grave results have been reported from this complication.

If the glands in the upper anterior triangle of the neck demand removal, an additional transverse incision from the division of the carotid forward to below the chin will give access both to the glands in the bifurcation and to the submaxillary glands, which are then removed, together with one or two glands in the space between the anterior bellies of the digastric muscles.

There now remains only the group of glands along the temporal vessels embedded in the parotid gland, which can be removed without any additional incision from the upper corner of the original wound. It is rarely necessary to remove the internal jugular vein when surrounded by suppurating glands from which it cannot be isolated. Its removal, however, does not cause any serious circulatory disturbances.

After disinfection of the wound, for which I usually employ tincture of iodine, the provisory ligature is removed from the deep jugular vein, the tendon of the omo-hyoid muscle and the divided clavicular portion of the sterno-cleido-mastoid reunited, and the wound packed with gauze, as described in the other operations.

Although the wound is enormous, and the operation often requires two hours or more for its performance, it is remarkable how slight is the effect on the patients. It is not uncommon for them to sit up in bed on the second or third day, feeling perfectly well.

It is sometimes advisable, when multiple suppuration with many fistulæ make the dissection very difficult, and when the patient is not strong enough to bear a prolonged operation, to close the wound when the operation on the lower part of the territory has been completed, and to postpone operating on the upper part of the territory for a month or two.

The only objection to total extirpation in any of the territories named is the possibility of non-re-establishment of the lymph circulation, resulting in chronic œdema of the lower or upper extremity or the corresponding half of the face or head. But this complication is extremely rare. Riedel[1] mentions two cases of permanent œdema of the lower extremities following extirpation of the inguinal glands in adults, and one case of œdema of the upper extremity in a boy of twelve years, following removal of the axillary glands. Bayer saw a temporary œdema and a condition resembling sclerema on the right side of the face, forehead, eyelids, and cheek following the removal of the cervical glands in a case of multiple fistulæ and suppurating foci, which disappeared after a number of months.

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[1] Loc cit.

No case of permanent œdema has been observed following the thousands of recorded operations on the glands of the neck. The lymph circulation in this territory is, therefore, always re-established. We need consequently have no apprehension of the radical operation in this locality. The cases reported from the axilla and groin, where the operation is much more rarely called for than on the neck, may lead us to prefer the less radical operations in these territories, and to endeavor to leave at least a part of the adipose capsule of the groups of glands for the re-establishment of lymph circulation.

CLEFT PALATE.[1]

By JOHN ROGERS, Jr., M.D.

Cleft Palate.—This may be either congenital or acquired. If of the latter variety, it is due to traumatism or disease, generally syphilis, less often tuberculosis or cancer. The congenital form occupies the median line, and is encountered most frequently and in its mildest grade as a fissure of the uvula or of the soft palate. In more severe cases the fissure may extend through part or all of the hard palate and the alveolus. The latter may be divided in the region of the lateral incisor teeth on one or both sides, very rarely in the median line, and then is apt to be complicated by total absence of the intermaxillary bone; the vomer and sometimes even the nasal bones may be lacking. Single or double hare-lip and projection of the intermaxillary are common accompaniments. The worst varieties of the deformity present almost no roof to the mouth and no anterior boundary above the lower lip.

The first difficulty to be overcome in pronounced cases is that of deglutition. If hare-lip is present, this may necessitate an immediate cheiloplasty, which, whether performed in the first few days of life or at a considerably later period, will tend, in the course of a couple of years, to narrow materially the cleft in the palate. Some writers have advised that a similar and more complete result be obtained by forcibly squeezing together the superior maxillæ and retaining them in the desired position by a silver wire passed transversely through the body of the maxillæ above the palatal process, or through the alveolus from side to side, or circularly around its base. As a general rule this is to be condemned, as it would involve a practical obliteration of the nasal fossæ in a wide cleft, while in a narrow one uranoplasty or an obturator would suffice. It would also deform the face and render occlusion of the teeth impossible, a matter subsequently of considerable moment in its influence on mastication and proper nutrition and general health. Cheiloplasty performed just after birth simply to narrow a cleft in the palate would not compensate for the increased dangers accompanying very early operation. It should be done about the sixth month, and at the end of a couple of years the advantage gained by earlier interference would not be noticeable.

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[1] The reader is referred to the very excellent article on Hare-lip, by J. Ford Thompson, M.D., vol. ii. p. 995. This article still remains a standard contribution; our knowledge of the subject has not advanced sufficiently to warrant rewriting it.

It is conceivable that an instance might very rarely be encountered in which forcible adduction of the superior maxillæ could be considered. If, for example, there were imperfect development of the lower jaw, and at the same time of the vomer or turbinated bones, so that the nasal fossæ could not be closed and the jaws rendered incapable of meeting, then this practice might be thought of; otherwise it is bad surgery.

In addition to the difficulty in deglutition above mentioned, food enters the nose, where it may be retained, and in decomposing set up more or less rhinitis; thence the inflammation extends through the Eustachian tube, causing otitis media with its accompanying earache, and later deafness. But the most unpleasant and marked of all the disturbances is defective phonation. The proper production of speech depends mainly on the integrity of the soft palate. Unless this organ is capable of touching the posterior and upper part of the pharynx, and so shutting off the nasal cavity, the pronunciation of a great number of sounds is impossible.

After the child has survived the dangers of infancy, which are mainly dependent upon the ability to take and absorb a sufficient amount of nutriment, the most important and almost the only object for which the physician is consulted is articulation. The rhinitis and the proper mastication of food are secondary matters. The former can generally be controlled by a little ordinary care as regards cleanliness and antisepsis; the latter, which might be thought serious, the patients learn to accomplish very satisfactorily. Hence any mode of interference which does not have as its primary object the attainment of improved speech must be considered a failure. It was formerly the rule to unite every cleft in the palate which could possibly be brought together. But this, in the light of subsequent experience, has been proved unsound. The result aimed at—namely, betterment of speech—can often be much more satisfactorily obtained by the insertion of an artificial palate or obturator of hard rubber. Soft rubber would be more ideal; but it decomposes so rapidly, causing in the process much irritation, and it requires such frequent renewal that it must unhesitatingly be condemned. In the cases to which it is adapted—and they form a far larger proportion than is generally recognized—the hard-rubber obturator is excellent. It should be applied as soon as the child is under control, which is ordinarily about the sixth or seventh year; then the same careful training in articulation is as necessary as after operative treatment,—that is, the mother must patiently and persistently teach the child to properly pronounce sounds which are found difficult. One who has had experience in training deaf-mutes to talk is well fitted to undertake such instruction. If the fissure involves both the hard and soft palates, and operation seems unwise, then it is advisable, as soon as possible after the development of the deciduous teeth, to make a plate to bridge the cleft in the hard palate. This will prove of benefit in mastication and articulation, and will prevent liquids from passing out of the anterior nares. It will also materially assist in the acquirement of the habit of nasal breathing. These appliances are temporary, and must be replaced by new ones when the permanent teeth are developed,—in other words, when the child is twelve or thirteen years old; afterwards, with proper care and cleanliness, they will last indefinitely. The teeth are of great importance in holding the obturator in place, and should be under the care of a dentist at an early age.

The obturator is indicated in all acquired cases in which there has been anything more than a very trifling loss of substance. Of course, when there is evidence of active local disease, this must be terminated by appropriate treatment before any steps can be taken to remedy the damage. The traumatic cases are more amenable to surgical interference than the pathological, but, as a general rule, whenever there has been destruction of tissue, mechanical measures are more apt to prove successful.

The making and fitting of plates require special experience, and a dentist's assistance should be sought; but a few remarks as to the general principles upon which they are constructed may not be amiss.

Take an impression in plaster of Paris of the hard palate the same as for artificial teeth; from this make a plate of rubber or gold to cover the hard palate, and accurately fit the lingual surface of the teeth posterior to the first bicuspids. This size is for those cases in which the velum alone is fissured; but if the cleft involve the hard palate, the plate must extend far enough forward to cover the opening, and when the intermaxillary bone has been removed or is absent, it must fill the resultant gap. The plate should not extend above the floor of the nasal cavity. It covers only the under surface of the cleft. As the soft palate is movable, it is impossible to take an accurate impression of its fissure and of the adjacent parts suitable for the construction in hard rubber or metal of an obturator; hence one has to be made, as follows. To the upper surface of the back part of the plate already made is attached a piece of clasp-gold about an eighth of an inch in width, and nearly equal in length to that of the fissure. This should conform to the curve of the fissure at an elevation of an eighth of an inch above it. Around this gold attachment a preparation of three parts of wax to two of paraffin is moulded. The appliance is inserted and removed and added to or taken from, as the case may require, and reinserted, until an obturator is formed about an eighth of an inch in thickness, and, if the muscles and tissues will admit, an eighth of an inch wider than the fissure above and posterior to it, and extending backward nearly to the pharynx and downward to the extreme end of the fissure. After this has been worn a couple of hours to become adapted to the shape and position of the surrounding tissues, it is removed and reproduced in hard rubber. A portion of the central gold strip is utilized to aid in the attachment of the obturator to the anterior segment of the plate. There is, of course, no appreciable motion in this contrivance, but with the necessary careful training it will be found that the superior constrictor of the pharynx will gradually attain the power of touching its posterior extremity, and so shut off the nares.

A hinge at the junction of the hard and soft palates can be used when a full set of artificial teeth is worn, but it is liable to become clogged or to pinch the adjoining tissues. If, after attempted operative treatment, the soft palate unites and the hard does not, and further surgical measures seem inadvisable, the soft palate, as a rule, must be split apart again to render the successful application of an obturator possible.

Acknowledgment must be made to the distinguished dentists, Dr. Norman W. Kingsley and Dr. Kasson C. Gibson, of New York, for their great services to the profession in the study and treatment of this deformity. To Dr. Gibson, in particular, I must render thanks for much assistance in the preparation of the foregoing matter.

Operative Treatment.—As before stated, this is seldom indicated in acquired cases. In the congenital deformity it should not be attempted before the second year, or when the child first begins to speak. If postponed till later, the prognosis as regards articulation grows worse with age. There is less chance of subsequent development of the palatine muscles. Therefore, about the second or third year is the time of election. Then note the width of the gap and the amount of soft parts which can be utilized to close it. This must be done with everything in as nearly a normal and relaxed condition as possible. If it is believed that the soft palate when united will be loose enough to enable it to be drawn up and back to meet the posterior wall of the pharynx,—in other words, if each half of the bifid uvula extends as far down in the relaxed state as a normal one, and if there is enough soft palate on each side to permit of coaptation of the segments without tension,—staphylorrhaphy should be performed. In general, there must be an abundance in length and breadth of soft tissue. Furthermore, when the hard palate is involved it is necessary to judge of the amount of shortening of the velum which will be produced by the mobilization and adduction of the muco-periosteal flaps ordinarily used to close the anterior gap. Exceptionally, when the palatine arch is very high and the palatine processes project upward into the nose, instead of horizontally inward, the muco-periosteal flaps will be found more adequate for their purpose than at first suspected. Their inner edges in dropping from a vertical to a horizontal plane approach each other. Likewise, a soft palate which immediately after operation seemed hardly large enough will by careful training and use gradually stretch, and in the course of several years develop to fill a space one-third or one-fourth greater in proportion than it originally did.

To sum up. After operation, successful so far as union of the parts is concerned, the chief complaint is defective phonation. This is dependent on insufficiency of the soft palate; hence operation requires abundance of velum. This may seem present, but in cleft hard palate the traction exerted by mobilization of the muco-periosteal flaps may greatly shorten it. On the other hand, increase in size of the velum not exceeding one-quarter or one-third may, in the course of several years of careful training, be expected. Consequently, if the soft palate when united cannot fill more than half of the vault of the pharynx, an obturator will give by far the better result.

Staphylorrhaphy is the name given to the operation for closing a cleft in the soft palate, *uranoplasty* for closing one in the hard palate. Complete anæsthesia is always necessary, generally with the patient in the horizontal position. It is sometimes advantageous to allow the head to hang down from the edge of the table, to facilitate the escape of blood. The mouth must be held open by some form of gag, to which is attached a tongue-depressor. If the cleft extend through both hard and soft palates, the latter may be united first and the former postponed for a subsequent operation, especially if much difficulty or hemorrhage is encountered; but in general the entire fissure should be closed at one sitting. In staphylorrhaphy the soft palate is drawn tense on each side by seizing at its tip first one and then the other half of the bifid uvula; its edges are then shaved or freshened by a sharp, narrow-bladed knife. Oblique paring of the margins of the cleft to increase the extent of raw surface is preferable to a straight, vertical cut.

Silk sutures are then inserted about an eighth of an inch apart and the same distance back from the edge. An ordinary curved needle and holder can be used, but one on a long shank bent like a fish-hook is better. This, with the thread in the eye at its extremity, is introduced on the upper or nasal surface of the velum from behind forward, the thread seized below, and the needle withdrawn and then passed through the opposite side. Care must be taken to grasp the proper end of the thread before the final withdrawal of the needle, so that the latter is not left hanging on the suture between the two points of puncture. To escape this difficulty the suture can be passed double through one side and then withdrawn, dragging with it the free end of a suture in the opposite side, as shown in Fig. 17, vol. ii. p. 1011. The suture is secured by a slip-knot reinforced by another knot over the loop (Fig. 17, vol. ii. p. 1011). This method of tying is preferable to the simple square knot, as the tension can be regulated more exactly.

Marcy recommends splitting the edges of the gap longitudinally with a knife having its tip bent at a right angle to the flat of the blade. When these flaps are folded back a raw surface is obtained equal in width to twice the depth of the cut, and no tissue has been sacrificed. Beginning at the anterior end of the cleft, the superior or nasal flap is first united by catgut sutures passed from before back through the centre of the split surface and knotted on the dorsum of the velum. A second row of fine silk knotted on the lingual surface is then used to draw together the raw areas of the lower flaps.

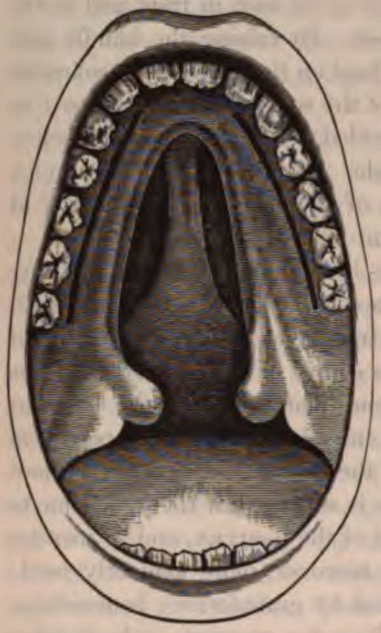
Tension must be relieved by dividing the tendon of the tensor palati muscle close to the inner side of the hamular process. Feel for the latter just above and behind the posterior extremity of the alveolus. The tendon lies in the centre of a line joining this and the orifice of the Eustachian tube, which can usually be seen through the cleft. Grasp the tip of the uvula with thumb forceps and draw it downward and forward to render the parts tense, and pass a narrow-bladed knife obliquely backward through the velum at this point, or about a quarter of an inch in front and to the inner side of the tip of the hamular process. By raising the handle and withdrawing the blade while the tissues are kept on the stretch a considerable liberating incision is made on the dorsum of the soft palate. Thus, part or all of the tensor and levator palati are divided, while the aperture below need only be the width of the knife-blade. If there is still too much tension, the anterior and posterior pillars of the fauces may be seized at their centres with forceps and cut with blunt-pointed scissors.

In Billroth's clinic the same object has been accomplished by severing the hamular processes at their bases. Place the edge of a narrow-bladed chisel immediately behind the posterior extremity of the alveolus, direct it backward, upward, and outward, and by a couple of sharp blows cut the inferior extremity of the internal pterygoid plate close to the hamular process. The latter is then dislocated far enough inward with a periosteal elevator to permit the freshened edges of the soft palate to meet without tension. This method of relieving tension is supposed at the same time to bring the soft palate a little nearer the roof of the pharynx, and so increase its efficiency. Division of the tendon is, however, more generally used; the small punctured wounds are left to heal by granulation; hemorrhage, which is slight, is controlled by pressure for a few moments or by ligation of the bleeding point.

Bryant relieves tension by cutting through the soft palate from its posterior border forward near its lateral attachments in a direction inclining a little outward from the cleft (Fig. 18, vol. ii. p. 1011). If the latter reaches to or just into the hard palate, after freshening the margins and before stitching, the velum should be detached from the posterior border of the hard palate with a periosteal elevator entered through the raw edges; otherwise failure of union is liable to occur in the front part of the suture line, owing to the tension at this point.

Uranoplasty, or closure of a cleft in the hard palate, is accomplished ordinarily as follows. The edges of the cleft are pared, and an incision is made on each side along the inner surface of the gums from the region of the lateral incisor teeth, or from just in front of the anterior end of the cleft, almost to the posterior extremity of the alveolus. (Fig. 1.) Close to the inner side of the base of the latter lie the vessels, which run obliquely forward to a point in the median line just behind the base of the alveolus in front. The incision should not cross these, at least posteriorly. With a curved elevator entered through these lateral incisions the muco-periosteum containing the palatine vessels is separated from the under surface of the bone and from the posterior border of the hard palate. Two flaps are thus fashioned, which are attached at, and receive nourishment from, their anterior and posterior extremities. These are slid together and sutured along their median edges, as described for the soft palate. Hemorrhage may be considerable, and is controlled by quickly completing the dissection and then applying pressure, an artery clamp, or, rarely, the Paquelin cautery.

FIG. 1.



If the cleft extends through the alveolar arch in front, Runge advises that the anterior pedicle or extremity of one flap be cut and the flap thus loosened in front slid inward to a greater extent. For very wide clefts Davies-Coley makes one flap in this fashion, and slides its raw surface over a similar raw surface obtained by turning over into the cleft a second flap from the under surface of the opposite side. The second flap is ⊃-shaped, with its pedicle attached to the whole length of the margin of the cleft in the hard palate. It is rotated on the edge of the cleft as a hinge till its palatal surface looks into the nose.

Similar successful operations have been reported with flaps cut from the muco-periosteum of the septum. The lower border of the latter supports the pedicle, the flap being turned down from above and united to the freshened edges of the hard palate. Flaps have even been cut from the posterior pharyngeal wall to make a velum. Kraske has utilized the inferior turbinated bone by severing its attachment to the superior maxilla, except at the posterior extremity. On this as a pedicle the bone is rotated on its long axis down into the gap, to the margins of which it is sutured. After union has occurred, the pedicle is severed and lowered to the required level. These are all complicated operations of doubtful value, especially when the possibility of employing an obturator is considered. If uranoplasty or staphylorrhaphy is to be performed, the method by flaps attached at each end to the roof of the mouth is much the best.

As mentioned before, the bad cases of cleft palate are usually accompanied by more or less pronounced hare-lip, prominence of the intermaxillary bone, and flattening and broadening of the alæ of the nose. The closure of the hare-lip at about the sixth month has already been described. The pressure exerted by the restored lip will, in the course of the next two years, when operation or a plate is to be considered, narrow the width of the cleft in the palate by about one-quarter or one-third. But a projecting intermaxillary bone may have to be dealt with first. The worst cases of its displacement forward, with a broad fissure in the roof of the mouth, may require very early operation to permit of proper feeding; and if the necessary treatment involves much hemorrhage, the operation may have to be divided into two stages,—the replacement of the intermaxillary and then, after a few days or a week, cheiloplasty. Ordinarily, however, it can be completed at one sitting, and this should preferably take place, of course, when the child is six months old.

If the bone is found to be only slightly in advance of the alveolar arch, its edges and those adjoining of the alveolus should be freshened, and the projecting bone forced back, fracturing, if necessary, its attachments. The closure of the hare-lip, which is always present and is generally double, should suffice to hold the raw surfaces in apposition. If it does not, a silk suture may be inserted through the anterior muco-periosteum of the alveolus and intermaxillary. Should this simple treatment be insufficient, a wedge, apex up, may first be excised from the vomer and septal cartilage close behind the protruding bone. As this necessarily divides the naso-palatine vessels and may cause serious and troublesome hemorrhage, Blandin's method is better. It consists in splitting the muco-periosteum of the septum along its inferior border, detaching it upward with a blunt elevator, and then with scissors exsecting the wedge of exposed bone and cartilage. The vessels remain attached to the soft parts and are not divided. The size of the wedge is regulated by the amount of displacement to be overcome. In general its base should extend as far forward as the line of the alveolar arch.

If transplantation of the intermaxillary bone in this way is impracticable, owing to its size or shape as compared with the alveolar arch, it must be extirpated. Although this necessarily sacrifices the incisor teeth, the loss is not as great as it seems, for in the majority of instances the teeth are so misplaced and defective that their extraction is imperative. Furthermore, fibrous union is the rule, and this leaves the anterior segment of the alveolus permanently weak and movable.

In removing the intermaxillary bone all the skin is first dissected up from its anterior surface, and after severing the attachments of the bone at the level of the lower edge of the septum the skin is turned in to form the columna of the nose.

AUTOSCOPY OF THE LARYNX AND THE TRACHEA.

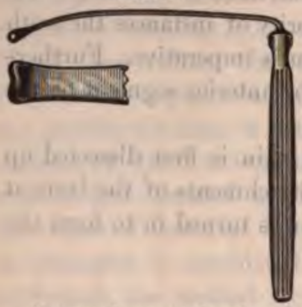
DIRECT EXAMINATION OF THE AIR-PASSAGES WITHOUT MIRROR.

BY MAX THORNER, A.M., M.D.

AUTOSCOPY is the method of getting a *direct* view of a patient's larynx and trachea without the use of a reflecting mirror or any other optical appliance. Thus it differs from laryngoscopy, the *indirect* method of examination. Autoscopy is the invention of Dr. Alfred Kirstein, of Berlin, who began his experiments early in 1895, and who has described his method in a number of publications which show the gradual evolution of his discovery from its crude beginnings to its present perfected form.[1]

The underlying principle of this method rests upon the possibility of bringing the imaginary axis of the laryngo-tracheal tube and of the buccal cavity, which ordinarily form an obtuse angle, into a straight line. In order to do this the patient is seated before the standing examiner, with his neck slightly stretched forward, while his head is tilted backward. Other obstacles to direct vision are the epiglottis and the base of the tongue, which must be removed from this imaginary line. This is effected by a specially constructed tongue-depressor. (Fig. 1.)[2] By firm downward and forward pressure upon the base of the tongue, and especially upon the median glosso-epiglottidean ligament, the epiglottis is elevated and the straight line of vision above mentioned is cleared of all obstacles, and thus it is possible to get a direct view of the well-illuminated larynx and of the trachea. The illumination is furnished by a forehead-mirror, or, better, by an electric forehead-lamp. (Fig. 2.) This is the method by which autoscopy is, as a rule, practised at present in adults. There are, however, cases which require a more complicated apparatus, the original autoscope

FIG. 1.



Tongue-depressor for pharyngoscopy and direct laryngo-tracheoscopy.

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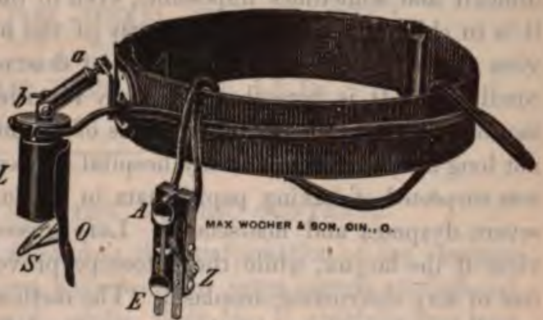
[1] Autoscopy of the Larynx and the Trachea. By Alfred Kirstein, M.D. Philadelphia: The F. A. Davis Company, 1897.

[2] The illustrations of this article are taken from Kirstein's work, mentioned above.

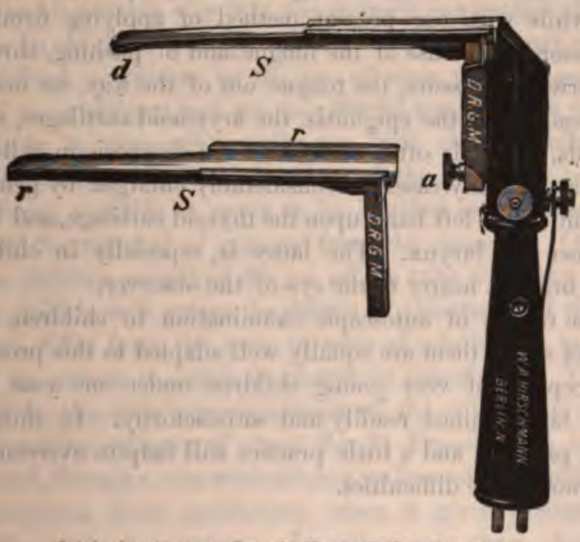
of Kirstein. (Fig. 3.) This instrument is probably better adapted to the examination of children than the simple tongue-depressor. (Fig. 1.) It consists of a grooved spatula (*S*, Fig. 3), the tip of which must be thickened and well rounded, in order to avoid injury to the mucous membrane. The tip (*d*) is notched to receive the glosso-epiglottidean ligament, and is bent downward so that its free border is one centimetre below the level of the bottom of the grooved portion. This spatula is attached to an electroscope, which forms at the same time the handle of the instrument. It consists of an electrical hand-lamp, the rays of which are collected by a lens and deflected ninety degrees by a prism, so that the rays of light are thrown along the spatula into the laryngo-tracheal tube, which, thus illuminated, may be readily inspected by the observer. The electroscope is, of course, attached to a source of electricity (*e.g.*, a storage battery) by cords. When used, the current is turned on by pressure upon the contact button of the electroscope.

FIG. 2.



Kirstein's electric forehead-lamp.

FIG. 3.



Original autoscope. Spatula (*S*) attached to electroscope.

Autoscopy has been found by practical experience to be an important addition to our method of examining the air-passages of adults. In children, however, the autoscopic examination is of far greater importance.

The laryngoscopic examination of children is, as a rule, exceedingly difficult and sometimes impossible, even in the hands of experts; and yet it is in children that it is frequently of the utmost importance to gain a view of the larynx for the purpose of determining the cause of stenotic conditions. It is here that autoscopy furnishes us frequently with information not obtainable by other means of examination. I had opportunity, not long ago, to examine in my hospital service a child of three years who was suspected of having papillomata in the larynx. There were at times severe dyspnœa and hoarseness. Laryngoscopy utterly failed to give a view of the larynx, while the autoscope proved the larynx to be entirely free of any obstructing neoplasm. The method of applying the autoscope in children is, in the main, the same as in adults, yet it is frequently necessary to employ chloroform in order to obtain a good result. In such cases the original autoscope (Fig. 3) is necessary. This appliance must also be used in autoscopic operations. However, in many cases it is sufficient to use, even in struggling children, the plain tongue-depressor depicted in Fig. 1. It is astonishing what a different view we may get by the departure from our former method of pharyngoscopy, in which we used to almost timidly place our tongue-depressor in front of the papillæ circumvallatæ, thus pressing the base of the tongue and the epiglottis backward and downward, as if we were bent upon shutting off any view of the lower structures; while with our present method of applying firmly the long tongue-depressor to the base of the tongue and of pushing, through downward and forward pressure, the tongue out of the way, we may in a good many cases readily see the epiglottis, the arytenoid cartilages, and the ary-epiglottic folds, which is often sufficient for a diagnosis in children. This good view may in many cases be considerably enlarged by gently pressing with the thumb of the left hand upon the thyroid cartilage, and by elevating in this manner the larynx. The latter is, especially in children, easily elevated and brought nearer to the eye of the observer.

As to the results of autoscopic examination in children, it must be stated that not all of them are equally well adapted to this procedure; but, with the exception of very young children under one year of age, the majority can be examined readily and satisfactorily. In this, as in other matters, tact, patience, and a little practice will help to overcome otherwise almost insurmountable difficulties.

DISEASES OF THE STOMACH.[1]

By WILLIAM PEPPER, M.D., LL.D.

METHODS OF EXAMINING GASTRIC CONTENTS.

WHILE the methods for determining the conformation of the stomach and the condition of its functions have been for years eagerly studied and widely extended in dealing with adults, their use in children has been sadly neglected until recent years, and even now is but too rarely attempted. It is certainly along this line that we now see the best opportunities for making our knowledge of the disorders of the stomach in childhood more complete; and it is in the hope that more interest will be shown in such research that a few remarks are introduced upon the use of the stomach-tube or a substitute and the examination of the stomach contents. Methods for determining the size and position of the stomach will be found in the chapter on dilatation.

In infants and children under two or three years the introduction of a soft-rubber tube of suitable size into the stomach is a matter of often surprising ease. In children, too, approaching puberty this may often be accomplished without opposition by the use of some patience and kindness and the aid of an intelligent and not too fearful temperament in the child. Children midway in years are usually too little able to understand our motives and too readily frightened to allow of the procedure being carried out without force; and this is never to be justified except when life may be saved, as by the removal of poisons from the stomach. Occasionally, however, these children permit of the use of the tube without a struggle.

When the child is well grown the details of the choice of the tube and its introduction differ from the same in cases of adults only in the size of the tube and the extent to which it should be introduced. These are to be determined by the physical proportions of the child. In very young patients a soft-rubber catheter of about No. 21 French scale is ordinarily best used instead, though a tube with thinner walls, and consequently greater lumen, is sometimes more satisfactory, since it gives passage to larger particles of the stomach contents. In either case there should be two good-sized lateral openings with smooth and well-rounded edges. As Holt suggests, I use in the further details of the apparatus a short piece of glass tubing, one end of which is inserted into the outer end of the tube or catheter, and a longer tube of rubber, at one end slipped over the free end of the glass tube, and at the other end holding a funnel, or expanding into a funnel-like enlargement.

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[1] The editor wishes to call attention to this, perhaps the last work of the distinguished author, and to congratulate himself and the readers upon its completion just before Dr. Pepper's untimely death.

When it is to be introduced, the child is held on the lap of the assistant or nurse, with a towel pinned around its chest to hold its arms to its side and the head somewhat forward. The left forefinger may, if desired, be placed along the dorsum of the tongue and beyond the epiglottis, to guide past the larynx and into the œsophagus the catheter, which, marked at ten inches and well moistened with warm water or dilute glycerin, is slipped gently but rapidly into the œsophagus, and then, with light pushing, glides easily into the stomach. The ten-inch mark indicates the average length to which it should be introduced, though the varying size of the child makes its retraction or further advancement often necessary before the stomach contents escape or water flows freely in and out.

When the tube is in the stomach, expression of a test-meal is accomplished by the straining efforts of the child, sometimes aided by pressur from below the stomach. If these fail, gentle aspiration may be used Lavage is best done with warm water, or, if there be much mucus present a little sodium bicarbonate may be added. With infants under six months old only about two ounces of water should be introduced and then siphoned off, the process being repeated until the water runs clear. In older infants and in children larger amounts of water are used in the same way. The best test-meal that I have found for infants is made by shaking the white of an egg in a pint of water. An amount of this proper for the age of the infant is used, as at six months three ounces, and expressed after one hour.

This contains albumen, the important ingredient; its own reaction is so slightly acid as to be unimportant, and it is usually readily taken and easily expressed. Cow's milk is very acid itself, thus interfering with the subsequent examinations, and it forms large curds, which are expressed with difficulty if at all, but it also may be used. It is valuable when testing the motor power of the stomach, used then in place of the test-dinner in the adult, and should be left in the stomach from three to three and a half hours. Older children who are on mixed diet should be given a suitable quantity of the usual Ewald meal of bread and water (or weak tea) for testing the acidity and ferments, and this expressed after one hour For motor tests they may be given a small test-dinner of soup, Hamburg steak, and bread, and this expressed after five or six hours.

The macroscopic appearance of the contents will at once give a rough idea of the condition of affairs. When expressing after the time given for testing the motor power one should, of course, obtain no contents, or at most but slight remnants. If much still remain, it is evidence of distinct motor weakness. With the test-meals the gross appearance is important. If the particles are finely divided and in a pap-like mass, it is evidence of normal digestion, or perhaps of hyperacidity, while large, undigested particles at once show that digestion is subnormal. Much mucus makes the contents tenacious and glairy and difficult of filtration, and evidences a gastritis with subacidity.

In testing the filtrate should be used.

Free HCl is determined by Günzberg's or Boas's reagent, either of which is used by mixing in a porcelain dish a few drops each of the reagent and of the filtrate of the contents and gently heating over a Bunsen or alcohol flame. On evaporation a brilliant rose-red color appears with Günzberg's reagent if free HCl be present, while Boas's reagent gives a rose-pink.

The Boas reagent does not give quite so striking a reaction as Günzberg's, and the evaporation must be done with a little more care. On the other hand, it is much cheaper, more readily procurable, and does not spoil on exposure to light, while Günzberg's reagent does. The two are made as follows: Günzberg—phloroglucin, two grammes; vanillin, one gramme; absolute alcohol, thirty cubic centimetres. Boas—resorcin, five grammes; saccharum album, three grammes; alcohol (fifty per cent.), one hundred cubic centimetres. For *lactic acid*, Uffelman's test is best for clinical purposes. To twenty cubic centimetres of four per cent. carbolic acid solution add a drop of weak ferric chloride solution. An amethyst-blue results, and upon addition of a few drops of filtered contents this turns canary-yellow or greenish-yellow if lactic acid be present. The test is best carried out by first shaking the filtrate with ether, decanting the ether, evaporating to dryness, and dissolving the residue in a little water. This will contain the lactic acid and lactates, and by using this for the test we eliminate the phosphates, mineral acids, and so on, which may give confusing reactions.

Butyric and *acetic* acids are recognized as present by their odors. The presence of volatile acids is shown, too, by heating a few cubic centimetres of filtrate in a test-tube, at the same time holding a piece of moistened litmus paper in the mouth of the tube. If such acids are present their vapors will turn the litmus red.

The total acidity of the filtrate is determined by titrating five or ten cubic centimetres with decinormal sodium hydrate solution run in from a burette. Phenolphthalein in alcoholic solution is used as the indicator, and with the appearance of a reddish tinge, which persists on stirring, the number of cubic centimetres of sodium solution used is read off. This is then best noted as percentage acidity after the manner of Ewald: for example, if three cubic centimetres of sodium solution are needed to neutralize ten cubic centimetres of filtrate, one hundred cubic centimetres of filtrate will need thirty cubic centimetres of sodium solution. We therefore have a per centum acidity of thirty.

The total free HCl is most satisfactorily determined by Mintz's method. We titrate very slowly with sodium solution, testing after the addition of each few drops with Günzberg's reagent. This is continued to just the point where the test for free HCl fails. The reading of the burette then gives the amount of free HCl by means of the factor .00365, since each cubic centimetre of sodium solution neutralizes that amount (in grammes) of HCl. If then ten cubic centimetres of filtrate need two cubic centimetres of sodium solution to neutralize the free HCl, there are in ten cubic centimetres of filtrate .0073 gramme of free HCl, or in one hundred cubic centimetres .073 gramme,—*i.e.*, there is present .073 per cent. free HCl.

The total amount of HCl may best be determined by the Hehner-Seeman method, which is as follows. Determine the total acidity. To another portion of the filtrate (five cubic centimetres) add a few more cubic centimetres of sodium solution than are necessary for neutralization. Evaporate to dryness in a platinum crucible or in a porcelain crucible on an asbestos plate. Ignite the residue at a red heat. Cool. Add a decinormal mineral acid solution in the same quantity as had been used of sodium solution. Titrate with sodium solution, and the reading, multiplied by .00365, gives the total HCl present in five cubic centimetres. This, multiplied by twenty, gives the percentage of HCl.

The results of this method are rendered somewhat inaccurate by the acid phosphates, which are estimated in the last titration at the same time with the HCl, and it is difficult to prevent losses in incineration. It is, however, a useful and rapid method for clinical work. A rough estimate of the amount of organic acids present may be made by noting the difference between the total acidity and the amount of HCl in terms of sodium solution.

The method of Martins and Lüttke is more exact, but also somewhat more prolonged and complicated. A description of it may be found in any of the leading works devoted to diseases of the stomach.

FUNCTIONAL DISEASES.

PRELIMINARY REMARKS.

The more modern methods of investigating gastric diseases in children, corresponding largely and wisely to those adopted with adults, show that, as in their elders, far fewer derangements can be called functional than was formerly thought. The more exact methods of examining into the size of the stomach and the condition of its secretions, greatly aided by more extended and reliable pathological work, give us much better ground for diagnosis than could be secured a few years since. These demonstrate, too, that many a case which would formerly have been called functional is really associated with organic change, and undoubtedly many, if not all, cases in which, by our present methods of pathological work, we are unable to find changes in the tissue, will be some day demonstrated by more refined methods to exhibit definite lesions. The post-mortem changes effected by the contents of the stomach make it especially difficult to study minute cellular changes, and the recent researches of Schmidt render it probable that our accepted ideas of even the normal structure of the mucous cells at least may have to be changed. It is, therefore, an evil practice to class as functional any class of cases in which there exist even insecure reasons for believing that organic changes are present, but there are certain cases of gastric disturbance which show every evidence of being what we now call functional. Undoubtedly many of these are due to little understood general nervous influences, while others will certainly be demonstrated as due to auto-intoxication and other causes whose real nature and conditions are still but guessed at, and our knowledge of them is as yet not sufficient to allow of their classification with greater exactness than under the general term "functional."

Particularly are many instances of what are called acute or chronic dyspepsia being constantly demonstrated to be dependent upon organic change, and although greater space is devoted in the following descriptions to these conditions than to acute and chronic gastritis, this is not because the inflammatory diseases are infrequent, but because the etiology, symptomatology, and treatment of the two correspond in many particulars. Since the descriptions of the functional diseases precede those of the organic, these matters will be found more fully treated of in the descriptions of dyspepsia. It is to be understood that acute and chronic gastritis are very frequent and much graver than dyspepsia, therefore more important.

ACUTE DYSPEPSIA.

Definition.—Any acute disturbance of the gastric functions in which there are no clinical or known pathological evidences of tissue-change in the stomach.

Etiology.—Predisposition to dyspepsia may be afforded by inherited systemic tendencies, or by these same tendencies acquired after birth, and among the most frequent of these in America is the uric acid diathesis. The children of gouty families, who are so frequently subjects in early life of persistent eczemas, and are apt later to exhibit the whole train of symptoms consequent upon the evil nutrition of the lithæmic, not infrequently evince an early and often lasting tendency to ready disturbance of any portion of the digestive tract. Tubercular subjects, whether the tuberculosis be slumbering in the glands or more active in any of the other tissues, quickly resent the slightest insult to their stomachs. The children, too, of tubercular parents, of parents whose energies are exhausted by laboring beyond their mental or physical strength, or of any parentage in which there is any grave physical fault, suffer in their digestive activity as they may suffer in any other function.

Congenital syphilis is largely aided in its destructiveness by the grave disorders of digestion which accompany it, and if the child does not succumb it often carries with it the sorry legacy of an inefficient stomach. The rickety child has bad digestion not only with his rickets, but often long afterwards, while very commonly acute illnesses cause dyspeptic attacks and leave digestive weakness behind them. Carstens thinks that the dyspeptic symptoms of grippe are caused by nervous influence rather than by organic derangement of the stomach. But upon the sponsors of the child should fall most frequently the reproach that their little knowledge of proper hygiene and diet, or their carelessness of these essentials, is responsible for the predisposition to dyspepsia or gastritis and their active causation.

In less common instances breast-fed babies have indigestion from alterations in the breast-milk consequent upon errors in diet or strong emotion in the mother, and this possibility is always worthy of investigation. A striking example of this occurred in my practice a few years since. The mother had a sudden access of melancholia, and the previously healthy child which she was nursing almost at once became ill with acute dyspeptic symptoms. It afterwards went on into a much debilitated state with an anæmia so profound as greatly to resemble pernicious anæmia, and recovered only after long ill health.

Occasionally the mother's milk or that of a wet-nurse of good constitution and proper stage of lactation disagrees, owing in some cases to peculiarities in the milk, in others to idiosyncrasy of the infant,—though these cases are exceptional. Again, the supply from the breast may be so scanty or of such poor quality that the child wastes, with or without signs of digestive disturbance. If the contrary be true, and an excess is freely delivered from the nipple, the infant swallows it greedily, only to regurgitate it, as a rule, soon after, and before it gives rise to irritative vomiting or fermentation and its consequent evils. But the repetition of this overloading will frequently upset the stomach. The usual causes of dyspeptic affections of sucklings are, however, not in the improper quality of the breast-milk, but, as in older children, in improper administration of the food, in unsuitable food, and in bad hygiene. In the last item the most important inclusions are insufficient or excessive ventilation of apartments, ill-graduated exercise, unwisdom in dress, and improper regulation of bathing, and it cannot be too strongly impressed upon the physician or the layman that hygiene is of the first importance, often the *sine qua non*, in the avoidance of gastric irregularities or their successful management, as well as in infectious diseases, where it has a much more firmly established place. Of greatest causal importance, however, are unsuitable diet and the inexact administration of food.

The lack of sufficient nourishment from the mother or wet-nurse is usually readily supplied by properly diluted and sterilized cow's milk in mixtures suited to the case, but it often is difficult, especially in the untutored classes, to convince mothers that their babies should be fed with even greater regularity than they consider necessary for their own healthfulness, and that the crying of the child is not so often an indication of hunger as of disturbance of its stomach already caused by irregular nursing; nor is the necessity for impressing upon parents the great value of regularity in feeding confined to the lower classes. Even more frequently than from irregular breast-feeding does dyspepsia arise in infants from the unduly large addition of artificial preparations to the diet, and in the lower classes in particular the suckling is not infrequently given food that would be difficult of digestion for the healthy adult stomach, while more intelligent people do not usually go beyond the lesser evil of prepared foods unsuited to the child's age or constitution. In older children the carelessness or thoughtlessness of parents often allows of the frequent use of tea, coffee, and hot spices, of pastry, preserves, and other sweets, or of an undue variety in foods, and with indulgence in candy and in dainties between meals the way is often paved not only for dyspeptic attacks in childhood, but for enfeebled digestion and ill health throughout the whole of a broken life.

Added to these errors, or acting alone, we often have imperfect mastication and insalivation, and the hastily swallowed food itself overtaxes the stomach.

Pathology.—The differentiation of dyspepsia from gastritis requires that the former shall exhibit no post-mortem lesions of the stomach. Lesions of other organs may be present, and upon these may have depended the dyspeptic symptoms during life, but any changes in the stomach itself beyond congestion of its walls or slight atonic dilatation would place the case in the class of organic diseases. It may be well to speak here of gastromalacia, or softening of the stomach, since its occurrence in the body of a child who had shown dyspeptic symptoms might lead to attaching undue importance to it. Both in the adult and in the child this condition was once described as a special disease, and a clinical picture of its symptoms was built up. It most frequently affects the fundus,—the most dependent portion in the cadaver,—which is likewise often congested. Sometimes the mucous membrane alone is soft and gelatinous, while at times the softening may extend throughout the coats, forming an irregularly shaped ulcer with soft edges, through which the contents of the stomach may have escaped into the general cavity of the peritoneum. It is now well established that this is not a distinctly antemortem lesion, but is due to digestion of the stomach-wall by the contents of the organ, which cannot occur in a living stomach. Even if it occasionally occur in the agonal period it is, in Goodhart's oft-quoted words, "the result of an ebbing life, and not a disease which causes death." The true pathology of dyspepsia so far as it is known is a pathology of function, and this may affect the secretions, or, that which is now established in so important a sphere, the motor function. The secretions in sucklings have not been studied with sufficient elaborateness to give us complete knowledge of the variations they may undergo, but from the published knowledge it seems clearly indicated that, as would be expected, the changes may be of the same variety as those in adults, though subjected to different kinds and degrees of exciting causes. In older children the changes must be closely analogous to those in their elders, if we may judge from our small actual knowledge.

The motor power, particularly in ill-nourished, unhealthy, or ill-conditioned children and sucklings, is frequently weak. Post-mortem examination may give proof of this in the relaxed and somewhat dilated stomach though this may not progress to a marked degree nor exist long without th resultant stagnation and fermentation producing inflammatory changes o having reached the stage where it must be classed as gastrectasia. There i no doubt, however, that acute and recurring motor insufficiency, dependin upon a too extreme tax upon the stomach by food difficult of digestio or too large in amount, does often occur, and the same symptoms frequentl present themselves after emotional or physical shock, upon the effects o which in the adult Rosenheim has so recently insisted.

Nervous influence frequently diminishes temporarily the gastric secretions, and certainly many of the cases of acute dyspepsia are caused in thi way. On the other hand, irritation and hypersensitiveness may in sucklings, as in older patients, cause hyperchlorhydria. Oddo and Luna hav shown this almost incontestably in sucklings, while Mensi, Leo, Clopat and others have given less certain demonstration of it. In older childre Rossbach, Lépine, and Snow have recorded instances, while Rosenthal an Fenwick describe the condition in special articles. These authors ref almost exclusively to hyperchlorhydria in chronic conditions, to be sur but there is every reason to believe in its occasional presence in acu attacks from acutely acting causes. Organic acids and other products o fermentation are found in the stomach contents in dyspepsia, and they hav potent local and probably even general effects.

The most authoritative investigations in adults—those of Oppler—sho a close relation between the quantity of hydrochloric acid and of pepsi and all investigations in children, though less exact, accord with this entirely rational result. The milk-curdling ferment seems much more persistently present than either of the other prime constituents of the gastric juic Szydlowski found it always present in fifty sucklings in various stages o digestion and in normal and abnormal conditions of the stomach, and th investigations of von Puteren, Raudnitz, and Leo have reached no greatl varying results.

It is, of course, to be remembered that whichever function—the secretory or the motor—be first at fault, the other becomes implicated almost a once.

Symptomatology.—The usual attack is soon seen to be the result o the disturbed functions that have just been treated of. After some error i diet, exposure to cold or heat, or without any known cause, the infan exhibits some restlessness or languor, grows peevish and irritable, ofte moaning frequently, perhaps crying sharply with pain, and becomes nauseated, the usual manifestations of which in an infant are a pale, contracte face, pronounced languor and relaxation, often with freely perspiring skin and retching. The belly will be somewhat distended, and there may b some epigastric tenderness. Often after a few minutes, and almost alway within a few hours, vomiting occurs, and this frequently terminates the whole attack by expelling the coarse or fermenting food which in many cases has caused the trouble. Or the vomiting may continue for a day or two, excited by any attempt to take food, or occurring spontaneously, and may reach much prominence in the clinical picture. Rarely it may be postponed for twenty-four hours or more, while the infant still exhibits nausea, anorexia, and general discomfort. The emptying of the stomach in young infants is so readily excited and so easily accomplished that the cases in which it does not occur in acute indigestion are rarer with them than in older children or adults, but occasionally the attack is so mild that it does not excite any vomiting. In these cases the offending contents of the stomach are passed on into the bowel and the trouble may end with a mild diarrhœa, and this is often the short sequel of any dyspepsia. When the attack appears, and often preceding it, the bowels are apt to be confined. Fever may or may not be present, but an elevation of temperature to 101° or 102° is usual, and it may for a short time go even higher, but subsides soon, often within a few hours, and in cases where no gastritis ensues it will be normal within two or three days. For some days afterwards the infant will probably take its food badly, have restless sleep and be somewhat irritable, and be liable to further digestive troubles unless they are carefully guarded against, and particularly is this the case in the premature or ill-developed or those with some constitutional imperfection. Older children frequently have the milder forms, with poor appetite, indisposition to play or to any effort, drowsiness, headache, and dizziness, and the gastric origin of these is indicated by nausea, heaviness, and ill-defined discomfort in the epigastrium, with sour or bitter regurgitation, and with their increasing years and ability to make known their symptoms they approach more nearly the manifestations commoner in their elders. The younger the children, nevertheless, the more unstable their temperature, and they are always apt to show at the onset extensive and sometimes alarming temperature elevations, and the severer forms of indigestion with vomiting and much prostration are common, in early childhood particularly.

Such are the usual symptoms and course of this affection; but peculiar and disturbing symptoms may accompany these or replace them.

When the nervous equilibrium is unstable, and especially in those whose family history is one with many instances of nervous disorder, the onset is often such as to direct the main attention to the nervous system. Convulsions may occur and may be repeated, but rarely are of fatal severity. Usually they soon cease, and vomiting, lavage, or free bowel movements are commonly sufficient to dispel the anxious fears of possible organic nervous disease.

On the other hand, errors in diet or indigestion from any cause are especially prone to bring on such attacks in actual epileptics. Other more localized effects upon the nervous system have been observed, such as strabismus, aphasia, even hemiplegia. Of aphasia the remarkable and much-quoted example observed by Henoch is a notable instance, in which almost complete inability to articulate came on suddenly with no gastric symptoms, and vanished as suddenly after a number of undigested cherries were expelled from the stomach by vomiting.

The respiratory and cardiac symptoms may be prominent and even completely veil the dyspeptic. All are familiar with the asthmatic attacks common in the adult and with their analogues in children, which are so manifestly dyspeptic asthmas, and children are sometimes seen in whom the extremely rapid respiration, the rapid pulse, the cyanosis, and the cold extremities point to grave organic lesions of the heart or lungs until these are excluded by careful examination, and our subsequent suspicions of a gastric origin are confirmed by the entire well-being of the patient after the stomach has been relieved. Cardiac arythmia, tachycardia, or bradycardia may also be manifestations of a dyspepsia, largely by reflex influence. Mayer and Pribram long ago demonstrated in animals slowing of the pulse and rise in arterial pressure upon irritation of the stomach.

Diagnosis.—In the milder forms of acute dyspepsia there can be little doubt or confusion. The trouble is often so evidently dependent upon error in diet, and the symptoms are so directly gastric alone, that other questions scarcely arise. When vomiting is present, as it so habitually is, however, and especially with fever and much languor, many possibilities are at once evident, and are excluded only by complete physical examination and the repetition of this procedure until danger of the acute infectious diseases in especial is past. An onset with vomiting and flushed, dry skin, together with variably high temperature and mild stupor, must at once arouse fears of the appearance of scarlet fever, which should be quieted only by the subsidence of the symptoms, the lack of a characteristic rash and of sore throat and enlargement of the tonsils and of the glands at the angle of the jaw, and the absence of albuminuria. The fleeting erythema which is common in gastric disorders has not the punctate appearance of the eruption of scarlet fever, and is more evanescent. In infants below six months scarlet fever is so rare that it may almost be denied consideration. Pneumonia and tonsillitis are the next most important questions. The rapid respirations and expiratory moan occasionally marked in dyspepsia may, on the one hand, simulate a pneumonia, and, on the other, pneumonia may be ushered in with gastric distress and vomiting as a prominent feature; and the latter is true of tonsillitis, in which young children and infants are especially apt to give no evidence of the true seat of the trouble. Errors in diagnosis in these two diseases are in almost all circumstances due to incomplete or careless physical examination, and it cannot be too often or too strongly emphasized that all cases of acute illness necessitate, if one would avoid blunders, the careful and repeated examination of the chest and the throat. Any of the other eruptive fevers may arise with gastric symptoms, and I have repeatedly seen rötheln show vomiting and fever for twenty-four hours, with a subsidence afterwards of all active symptoms, and so slight a rash that only deliberate examination discovered it. But in all these disorders the concurrent manifestations, such as the extensive glandular enlargements in rötheln, and the weeping eyes and nose in measles and the discovery of an eruption if it be looked for, make the diagnosis easy.

In nephritis one avoids a blunder and establishes the diagnosis by the examination of the urine, which should, with a complete physical examination, be part of a routine.

Lastly, the diagnosis of acute dyspepsia from acute gastritis is clinically to be based principally upon the duration of the attack, and, in especial, the duration of the vomiting and of the fever. If the vomiting persist for more than two or three days, and particularly if it grows more severe and much mucus is expelled, and with this, perhaps, occasional small blood-clots, we must recognize a gastritis. And if the fever does not vanish in the same time, but, while pursuing a lower course, still holds on, an inflammatory change is to be accepted. These are, however, border-land cases, and in such no strict differentiation can be made. The attacks of dyspepsia which are quickly past, and exhibit to us in the vomit the whole cause, are readily separated from gastritis.

Prognosis.—The prognosis of acute dyspepsia is almost invariably good. It is only in the irregular manifestations of gastric disturbance, or when the latter occurs in those who already have a weak tenure upon life, that fatalities ever result. In complications such as convulsions fatalities are not common, but the prognosis should not be rashly stated. Nor is the prognosis as to complete recovery from the effects of the attack a less happy one if the management of the case be sufficiently conscientious. Recurrent attacks of indigestion are due in very many instances to the thoughtlessness or indifference of the medical attendant or elders.

Treatment.—The initial treatment should depend upon the previous occurrence or absence of free vomiting. In any case, however, in infants lavage will do good by calming the irritated stomach, and harm will not result from its use unless there be grave cardiac disease or other such distinct contra-indication. If vomiting has not occurred, or but small amounts of the contents of the stomach have been with difficulty ejected, lavage offers the most ready method in children of less than two years of unloading and quieting the stomach. When for any reason lavage may not be employed with infants, and in most cases in older children, other means of emptying the stomach must be resorted to. Infants may be given lukewarm water from their bottles, and older children may take one or two glasses, this being followed by introduction of the finger into the throat to excite retching. If this simpler method is not sufficient, it may be reinforced, especially in older children, in whom vomiting is less regularly an initial symptom, by one of the milder emetics, such as ipecac or sulphate of zinc; but care must always be taken that the emetic itself, from its nature or its quantity, does not make the irritation greater.

On the other hand, we frequently find, in infants especially, that free vomiting has already occurred, and everything may have quieted down, so that all that is necessary is to keep the child at rest upon the lap or in bed, to apply a cloth wrung out of hot water or a mustard plaster well weakened with flour over the epigastrium, or to substitute for these latter a spice plaster or painting with diluted tincture of iodine, and to direct that the patient shall for several hours, at least, receive no food. Small pieces of cracked ice may be given to quiet thirst, or teaspoonful portions of cool soda or Vichy water may answer for this purpose. Mixed with equal parts or less of milk these are less apt to excite vomiting again, and the same small portions of cold albumen water are often satisfying and non-irritative. If the stomach is quiet, after six or eight hours the baby should, if on the breast, be nursed, but at longer intervals and for a shorter time for a day or two. In others the return to artificial preparations or table-foods should be gradual, and the amounts and variety of food be so increased that only after a few days do we return to its settled diet. If the bowels were confined at the time of the attack, or some abdominal distention and distress occur afterwards, with constipation or a mild diarrhœa, small repeated doses of calomel (one-twentieth to one-twelfth) and bicarbonate of sodium, or some of the other unirritating laxatives, such as castor-oil in emulsion, or magnesia, will unload the bowel and often put a stop to all discomfort. Frequently constipation is as well or better relieved by a simple or glycerin enema, and all other active laxatives should be withheld, as they are apt to irritate the stomach.

When matters are not so simple, and we are called to control continued vomiting or reduce the fever, we should insist that the child be kept at rest in a quiet, darkened room, and that food by the mouth be absolutely withheld so long as vomiting occurs upon its ingestion or spontaneously. The latter may require special emphasis and watching, for even temporary starvation frequently arouses much determined opposition in the elders, but it is essential for a rapid cure and the prevention of exhaustion that the already overburdened stomach shall not be further taxed. While the vomiting continues, nothing may be allowed by the mouth that is not calculated to allay the irritation or, by gentle measures, to relieve thirst, and of these, as already mentioned, cracked ice in small pieces or carbonated waters in minute portions are admissible. If these are rejected or the vomiting does not cease, and the irritation and restlessness be severe from the start, it is well to give an enema of warm water containing deodorized tincture of opium in small dose and suited to the age of the child, or the same drug in smaller amount may be given by the mouth, and minute doses of aconite with it will at the same time lessen the fever. The latter symptom is usually best controlled by a cool or, in infants, tepid bath or sponging with water to which alcohol may be added. In older children, if these measures are not sufficient and the stomach is not specially unretentive, small doses of the antipyretics may be given, of which the best is phenacetin. The temperature, however, rarely remains high many hours, and its persistence indicates, as stated, an inflammatory lesion.

When, as so often happens, the bowels have been inactive, the most successful medication may be laxatives which have at the same time sedative action upon the stomach. With any of these measures abdominal applications of mustard or spice plasters or hot cloths may be used with advantage, especially if there is much pain.

The nervous symptoms not infrequently demand action, and often the first necessity is to combat these. Suggestive signs or complaints, such as extreme restlessness, jerking of the face or general muscular twitching, grating the teeth, or, in older children, severe headache, may not be neglected without danger of convulsions, and a warm bath should be used at once. An enema of chloral from two grains upward, according to the age and strength of the child, of large doses of the bromides, or of proper amounts of laudanum or deodorized tincture, or combinations of these, will be sometimes needed, and will often reduce the fever, which is not infrequently largely from nervous irritation. If there be much fever, with marked nervous symptoms, antipyretics should be used by the mouth if the stomach is retentive, by enema if necessary.

If convulsions occur and the stomach be suspected as the cause, the main indications are to empty this organ at once by lavage or by ipecacuanha or other emetics, and to control the nervous system by baths and increased doses of chloral or the bromides, or both, with the use, if necessary, of chloroform and morphine. The less common nervous manifestations, such as the aphasias mentioned, are most rapidly and surely relieved by treatment of the cause, which lies in the stomach.

Exhaustion may at times be so great as to demand active stimulation by alcohol, nux vomica, or other remedies adapted to the individual.

The management of the case up to perfect convalescence should not suffer the neglect that is often shown. The rest should be continued until all symptoms have subsided, and the usual diet should be re-established tentatively and gradually, and should be properly modified if there be reason for holding it in any way responsible for the attack. When the stomach has become retentive, digestive aids, such as hydrochloric acid and pepsin, may be wisely used, and when the irritation has left its frequent result in a tendency to chronic gastric insufficiency, the stomach functions should be stimulated with bitter stomachics, such as gentian, and particularly nux vomica, given in small doses at first, and these stopped if there be any evidence of irritation.

CHRONIC DYSPEPSIA.

Definition.—The continued or repeated expression in clinical symptoms of abnormal gastric functions without evidence of organic lesion of the stomach. Still more difficult is it in chronic conditions of the stomach than in the acute to state that no organic lesion exists, and it is becoming constantly more evident that the existence of chronic gastric disturbance means in most instances change in the stomach itself. But there remain cases in which neither the repeated examination of the stomach and of its contents nor the symptoms presented serve to convince one that an organic process is at hand. Especially is this true of those children who are disposed to nervous instability, and in the recurrent attacks of dyspepsia that become so frequent as to deserve the name chronic we must often look to functional disorders rather than to gastritis for the cause.

Etiology.—The etiology of chronic dyspepsia is largely in a continuation or repetition of the causes of acute attacks.

In those who are predisposed to ready disturbance of any function by evil inheritance or by bad constitution in their progenitors, errors in diet or lack of hygienic care may suffice to induce an acute attack, and after being once pushed beyond endurance the stomach may never be able completely to fulfil the normal demands, or the affection may appear gradually from unnoticed strains upon it or from an especial local predisposition. All children of overworked parents or who come of tubercular families are apt to exhibit this predisposition. In the tubercular the family tendency to pulmonary involvement frequently manifests itself only after the general health and nutrition have suffered from poor digestion, and the children from a stock that exhibits its nutritive disturbance in gout and rheumatism are very likely to show this heritage first in faulty digestion. The first dentition, though a physiological process, is an irritating one, and may suffice to set up gastric derangement in a predisposed child or one ill cared for.

Again, these predispositions are made active by insufficent bodily care in diet and hygiene and by their unhealthful surroundings. The impure air of large towns and cities and the ill-ventilated dwellings of the city conduce to dyspepsia, and improper clothing and exposure are factors of the greatest importance. The onset of acute illnesses, of the infectious diseases in especial, in disturbance of the stomach may find its sequel in chronic dyspepsia. But, as in the acute conditions, the imperfect regulation of the quantity and quality of the diet and its improper preparation are the most important factors in the etiology. The irregular nursing or feeding of infants and the unfortunate thoughtlessness of parents of older children in allowing them all kinds and quantities of foods at their meals and between meals, particularly the eating of candy and other sweets, pastries, and hot breads, are frequently the sole causes, and too seldom recognized as such before the damage is done. Imperfect mastication is almost the rule in these youngsters eager for their sport, and this should be firmly and persistently corrected. Carious and ill-cared-for teeth prevent proper mastication, and may directly cause gastric irritation by giving rise to local inflammations of the mouth or by the putrefying and bacteria-laden discharges from them.

Pathology.—The pathology of chronic dyspepsia is but the pathology in function that occurs in acute dyspepsia extended over a considerable period or recurring with frequency. The organ itself may exhibit after death some relaxation.

But if atony of the stomach has resulted in a distinct enlargement, we have gone beyond a simple dyspepsia, and have the much more serious condition of gastrectasia. The other organs are very frequently diseased, and catarrhal or ulcerated conditions of the bowels, pulmonary changes, or other lesions may have given rise to the purely functional gastric derangements, though their more frequent accompaniment is a gastritis.

The functions of the stomach may suffer in any of the varieties of changes. Especially the nervous and emotional among children may, as in adults, show unsatisfactory motor power, and it must become fully recognized that this is often the only faulty function, or that the secretions have suffered secondary change from the irritation induced by the consequent extension of the labors of the stomach. We should be more willing to accept a muscular weakness of this organ, as we have long been willing to admit such weakness in other organs whose function is largely dynamic. Jaworski is somewhat immoderate in attributing to the stomach so little individual part in the actual preparation of the food for its absorption and in relegating the viscus principally to the sphere of a reservoir, but he has done good service in leading to the recognition of the evil that results if the normal chemical changes in the stomach are not accompanied by proper propulsive force.

Of the secretory changes those in the hydrochloric acid are the most important in their value as indications of subnormality or over-secretion and in treatment. That hyperacidity may occur in infants has been demonstrated by the investigations of Oddo and Luna and others which have been already mentioned. In older children the difficulties encountered in the investigation of the secretions by means of the stomach-tube are such that our knowledge is very limited, but the fact that hyperchlorhydria does occur is now well recognized. Mensi, Friedeman, and a good many others have found this in some of their cases, while Rosenthal, Leyden, Boas, and others recognize a distinct form of hyperchlorhydria in children which arises from over-study. The occurrence of hypersecretion as a separate entity is a matter of serious doubt at any age, since in most instances it is caused by some irritation of definite source or a general hypersensitiveness of the stomach. General secretion in excessive amount does occur in childhood, as in adults, however. Rossbach, among others, has demonstrated this; von Noorden has found it in chlorosis at the period where childhood verges into womanhood, and I have confirmed his results. In all these cases it is apparently due to nervous irritability of the stomach. That it occurs from actual irritation cannot be doubted by one who has frequently administered test-meals in cases of dilatation, particularly of the obstructive variety, the large amount of very acid fluid that one expresses with the meal, often exceeding in quantity that originally given, being sufficient proof of a hypersecretion from irritation.

Hypochlorhydria which is continued and constant is usually a sign of inflammatory lesion. It is not pathognomonic of such, however, and hydrochloric acid and the ferments may be entirely absent over long periods of time without any other discernible cause than nervous influence. These are rare cases, however, and the most characteristic thing about the acidity of the stomach contents in nervous dyspepsia is its entire irregularity, being sometimes high, sometimes low, and again normal.

The amount of pepsin follows pretty closely the amount of hydrochloric acid. The milk-curdling ferment is more persistent and rarely entirely absent.

Symptomatology.—The establishment of a chronic dyspepsia is very frequently preceded by one or more attacks of acute indigestion. After the stomach is once upset it may for long afterwards be constantly subject to disturbance upon the slightest cause, or, again, the acute attacks may be so frequently reinduced that, with the consequent depression of the general system and the continued irritation of the stomach, the latter becomes so weak or so sensitive that the ordinary demands upon it are a burden, and increased demands are met by open revolt.

Generally the infant gradually loses some fat, its muscles become flabby, and it grows pale and puny. The amount of loss in flesh is largely dependent upon the quality of its diet and whether the amount is sufficient. When the food is unsuited to the child, and especially when the natural breast-milk or its congener, cow's milk, is largely replaced by artificial preparations in which starch is usually so important an ingredient, the emaciation may be less dependent upon the degree of interference with its gastric functions than upon a species of starvation, for the babe is not receiving that which is assimilable, but the ingested material is cast out by the bowel, unused. Especially is this true of the offspring of the poor and the uninstructed, whom we sometimes see put upon solid diet in the first week of life.

With the loss in strength and weight come changes in temperament. The happy temperament of the healthy babe is replaced by peevishness, and it grows languid and irritable, pays less attention to its surroundings, and is less easy to amuse. Sleep is restless and often broken, and particularly in the night does it require attention and soothing to quiet the attacks of crying, which are without apparent cause or evidently due to abdominal distress. While the healthy infant is always ready for the breast or the bottle, the appetite of the dyspeptic varies. It may at times suckle ravenously and take large quantities of food, which frequently results in colicky pains or the sweating skin and drawn face indicating nausea in infants, and this may be followed by vomiting; or the appetite may be poor, and it soon drops the breast or leaves the bottle half emptied, and this may reach the grade of absolute anorexia, so that it refuses food for long periods and may even require gavage, or forced feeding. Not infrequently anorexia of varying degree alternates with voracious appetite. The tongue may not show any abnormalities, but is usually pale, large, and lightly coated. Vomiting occurs in many cases, but is less prominent than in gastritis. It may be excited only by overloading the stomach, by irregular feedings, or by unusual neglect of hygienic care. Many exacerbations, with vomiting, fever, and prostration, are brought on by imprudent exposure to cold and dampness.

With these symptoms there is usually some distention of the belly, which often grows very protruding and tympanitic. Attacks of colic frequently occur in these infants; the abdomen becomes more tense and distended and somewhat tender. The bowels are apt to be constipated, and the stools are often dry and hard and of clay-like or other unhealthy color, and their expulsion is often attended with pain that is occasionally distressing. Diarrhœa frequently alternates with the constipation, often lasting only a short time, to be again replaced by constipation. The diarrhœa may persist, and in these cases it usually soon dominates the whole scene. When the nutrition has suffered severely the temperature is often subnormal. Fever may occur with acute exacerbations.

The train of symptoms in older children approaches that in the adult.

The general nutrition suffers in one way or another. Some dyspeptic children lose no flesh, or may put on fat, but the muscles grow soft and there is a general lack of systemic tone. In others there is moderate emaciation and flabbiness, while still others, much rarer instances, exhibit very extreme loss of flesh, though these latter must often have inflammatory changes in the stomach. The stature of the child may be small and its appearance stunted, but children who have grown more rapidly than normal very commonly have dyspeptic troubles. With this evil nutrition, languor develops, and the child appears weak and debilitated, is pale or sallow, and its temper and disposition are uncertain. It no longer takes its former interest in play, loses the elasticity of youth and is only fitfully energetic. The temper of healthy, unirritated children is almost invariably good, but the dyspeptic become irritable upon little provocation, and at times they are sullen and almost melancholic. The perversions of temper when no active symptoms of disease are present often lead to unjust punishment by the elders, who readily attribute them to faults of the child. The sleep is disturbed and unrefreshing, and after heavy, deep slumber the child awakens stupid and unrefreshed. During sleep it often grates its teeth, rolls and tosses about the bed, its muscles twitch, and visions of unearthly beings and wild beasts or climaxes of horrible nightmares awake it, startled and affright, and many dyspeptic children fear the night because of the frightful dreams that so often disturb them. The nervous element is often prominent in dyspepsias of childhood, as in adults, and pronounced nervous phenomena may appear. Acute exacerbations may be announced by convulsions or the rarer local signs, and attacks of hysterical excitement occur, or the child may become chronically hysterical. Syncopal attacks bearing close resemblance to *petit mal* are at times observed, and the disturbances of the heart's action or of the respiration noted under acute dyspepsia are very frequent. The appetite shows even more variation than in infants, and perversions of appetite are common. Little desire is usually shown for suitable food, or this may be partaken of with but fitful voraciousness and at irregular hours, or dainty and highly seasoned articles, candy, and the like may be the only things to tempt it to eat. Anorexia may become so pronounced as to produce extreme emaciation, and it may refuse food to dangerous limits, though these are rare occurrences. Such a case I saw recently in a young girl who was ill with subacute rheumatism, and whose stomach was irritable. Forced feeding was made necessary by her refusal of food, and even then she for some time brought on vomiting immediately afterwards by introducing her finger into her throat. Only after she had become dangerously exhausted did the moral impression of persistence in the forced feeding and rectal alimentation induce her to take her food naturally.

Pain is frequently present. Commonly the only complaint is that of fulness and distress from distention after eating, with occasional turns of acute colicky pains a few hours after food. Burning in the epigastrium, the so-called heartburn, occurs frequently, usually some hours after food or when the stomach is empty, and sometimes becomes severe, while gastralgia of diverse grades of severity up to the most extreme occurs at times.

Nausea is a frequent complaint of older children, and those too young to give verbal expression to their ills show this in the manner already described. It often comes on after meals, or may precede them, and a meal is frequently begun with a good appetite which soon veers round to nausea and disgust for food.

Vomiting takes a less prominent place than in the habitual indigestion of infants. It may occasionally occur when the stomach is overloaded or after unsuitable food, and acute exacerbations with vomiting are constantly apt to interrupt the usually less stormy course of the affection. Eructations of small quantities of sour or bitter stomach contents are common. The cases that exhibit a distinct hyperchlorhydria usually have a good deal of pain, which is worse when the stomach is empty and decreased by taking food. Often the epigastrium is distinctly or even excessively tender. Vomiting is not infrequent in this class, and this may relieve the pain at once. When the excess of hydrochloric acid comes on periodically there is usually severe prodromal headache, soon followed by severe pain in the epigastrium, and to this vomiting is commonly added, the vomit being very rich in hydrochloric acid and often containing bile. After emptying the stomach vomiting may cease, or it may continue for some days, sometimes with serious exhaustion. After the attack passes off the child will be in comparatively good health, though milder symptoms often persist until another outbreak occurs.

The bowels show such variations that no regularity of their condition can be expected. The commonest is constipation, which may be marked, and the passage of the hard stools may cause so much distress and pain as to lead the children to suppress the desire for defecation and thus exaggerate the constipation. Little fissures about the anus are not uncommon, and are acutely painful. Frequently constipation does not exist, but is replaced by one or more daily stools of mushy consistence containing much half-digested food. Some children with irritable stomachs have constant tendency to diarrhœa, and those cases in which loose movements occur soon after meals, often with colicky pain, are, I believe, frequently due to an irritable or insufficient stomach, which hurries the contents onward before they are properly macerated and digested.

The actual signs of dyspepsia are few and irregular. The tongue is no certain indication of the condition of the stomach, but it usually shows variations from the normal. In the nervous and atonic cases it inclines to be large, pale, and flabby, with marks of the teeth along the edges and a variable amount of white or dirty coating on the dorsum. In many cases it is covered with a yellowish, thick fur, and the breath is heavy and unpleasant or foul. A red and beefy tongue occurs frequently in hyperacidity, but bears no constant relation to it. Caries and unclean teeth, enlarged tonsils, and chronic inflammations in the naso-pharynx or the pharynx are causal factors rather than signs of dyspepsia itself, but should always be looked for in examination.

The epigastrium may be slightly tender to pressure, and usually shows some degree of prominence. Often the protruding belly is very large from the distention of the intestines as well as of the stomach, and in ill-nourished children the contrast between the full abdomen and the thin chest and limbs and pinched face is very striking, and may suggest a dilatation of the stomach.

In infants, and in older children when it can be made, an examination of the stomach contents after a test-meal will usually show a larger quantity than normal, and the stomach will not empty itself quite within normal limits. In less frequent cases, where the stomach is very irritable, the contents are driven hastily on into the intestine. The hydrochloric acid may show variations such as indicated, and will often be found to exhibit all the conditions from normal to hyperacidity or to irregular and inconstant subacidities that are met with in atonic and nervous dyspeptics of more mature years.

Diagnosis.—The symptoms are usually distinctly gastric, and the chief difficulty is in eliminating organic affections of the stomach.

When vomiting is a very prominent or constant symptom, and much mucus is expelled, or when pain is marked and epigastric tenderness is present most of the time, it is certainly safe to consider the cause a gastritis.

If several examinations of the stomach contents show free hydrochloric acid absent in infants, or in very small amounts in older children, and the total acidity very low, this is very strongly in favor of gastritis, particularly if much thick mucus be present. Irregularity and lesser severity of symptoms and variable conditions of the stomach contents are the characteristics of dyspepsia.

Dilatation of the stomach must be ruled out by the methods of diagnosis to be found in the section on that disease, and it is always well to be on the lookout for milder grades of this affection.

Intestinal parasites and renal and constitutional diseases must not be overlooked. Parasites of the intestines frequently cause dyspeptic symptoms, and if they be suspected unirritating vermifuges may be administered to clear up doubts.

Renal disease and diabetes are excluded by repeated examinations of the urine. Tuberculosis may be long feared, and excluded only by physical examinations of the chest and abdomen and careful temperature records, together with failure to find the tubercle bacilli in the stools or, in older children, where this can be obtained, the sputum.

Congenital syphilis may be readily overlooked in infants if its characteristic signs be not present. These should, however, be repeatedly looked for and the parental history inquired into, and if doubt still remains the therapeutic test may be applied,—best by inunctions of mercury, as such drugs by the mouth are apt to increase the gastric difficulties.

Chronic dyspepsia is always a slovenly diagnosis until careful examination and observation of the case convince one that no organic disease exists.

Prognosis.—With properly careful management a simple dyspepsia with no lesions of other organs may almost always be cured. With associated change in other organs the prognosis of the dyspepsia is that of the then more important disease. Otherwise the prospects of recovery depend largely upon the conscientiousness of the physician in his inquiry into the causes and his regulation of diet and hygiene, and upon the care which the child receives from its elders, though there are cases in which the natural frailty or the unhappy and unalterable surroundings make betterment impossible.

The only direct way in which dyspepsia may end life is by the inducement of the rarer nervous phenomena, of which convulsions are the only ones in which danger is to be feared, or by adding to the strains of a system which has already reached its limit.

But all digestive troubles have a strong tendency to recur, and require long after-treatment to insure permanent recovery. If the difficulty persist for any time by reason of inefficient management or constitutional tendency the resulting depression or irritation makes the way for organic disease of the stomach itself or of other organs. Tuberculosis, catarrhal pneumonia, or nephritis may bring the end, and it is always necessary that one be on his guard against the development of complications.

Treatment.—The first point necessary is an investigation of the diet and its careful adaptation to the individual case. Space does not permit of elaborate consideration of this most important point, and more complete information must be sought in the articles on dietetics; but certain leading suggestions must be brought into notice.

In infants still at the breast a continuation of dyspeptic symptoms beyond an acute attack should always call for an examination, if possible, into the quantity and quality of the breast-milk, and if this be found at fault, or if it continue to disagree without any distinct reason therefor, a suitable wet-nurse should be procured when available. If, however, a wet-nurse cannot be had, and the mother cannot nurse the child, or her milk continues to disagree in spite of well-regulated nursings and proper medicinal treatment, we must seek a suitable artificial food, which is usually most nearly reached in a well-selected cow's milk diluted with water, lime water, or barley water, to which milk-sugar is added to a degree suited to the age and stomach of the child, and administered at exact intervals. The latter must, with the quantity, be determined for the individual case. For infants but a few months old dilution to at least one-half should be at first advised, and often more of the diluent must be added. Frequently mixtures of milk, cream, and diluents do better by more nearly approaching the mother's milk or the special needs of the child. The so-called strippings from the cow, properly diluted, are at times more digestible than the first milk. The mixture of gelatin, milk, cream, and arrow-root recommended in Meigs and Pepper on "Diseases of Children" has often given me good service in these cases, and I have much confidence in again advising its use. Completely or partially peptonized mixtures may be the only way in which milk is retained, or the resort to gavage may be a means of keeping it down when not retained after swallowing. When milk agrees in no form, chicken, veal, or beef broth is very useful, and albumen water is likewise acceptable and often soothing to the stomach. These may be often continued over a considerable period with much improvement, and raw beef juice used with other preparation, or even alone, frequently gives admirable results. However the milk be prepared, it should always be well selected. Milk prepared in special laboratories of any desired strength of ingredients is available to many of us, and we may sometimes procure the needed combination in this way. But the sterilization in the laboratory is never to be substituted for that done at home. Sterilization, or, better, pasteurization, is almost always necessary for infant foods in the city, and in frequent cases in the country, and I much prefer to have this done in the house.

With proper diet and exact feedings most infant dyspepsias disappear, but the investigation of the clothing and bathing of the child should always be made, and it should be invariably seen to that woollen underclothing covers the whole of the body and limbs, and a light flannel binder over the abdomen should be always worn. With these precautions in clothing the skin should be kept acting well by daily sponging with water, which may contain a bit of salt or some alcohol, and babies whose strength is good may be placed in their tubs. Inunctions with olive oil following the bath are very useful in preventing colds, and seem to improve the nutrition. Cod-liver oil is sometimes thought more beneficial, but its persistent and offensive odor makes it difficult to secure persevering use of it. By these means we prevent many colds, and the room may be kept aired and at almost 70° F. without danger of chilling the child.

In these main principles of diet and hygiene lie our most certain means of cure.

Fleiner's forcible remark, that "the stomach is not a retort allowing of the introduction and removal of substances at will," applies to the use of the stomach-tube at all ages, and lavage should ever be used with discrimination. If there be much irritability of the stomach, lavage, practised once, twice, or at most three times a week, may be of much use in infants, but can rarely be used in older children. Atonic stomachs are sometimes much benefited by washing occasionally with water of a temperature a little below that of the body or with warm water. But the use of lavage should depend upon the results of a trial. If the appetite and general nutrition improve and the distress decrease, the procedure should be continued. If good results are not secured or things grow worse, it can only do harm, and should be stopped.

Medication should be limited as far as possible, and used only for some well-defined purpose. If there be much irritation and tendency to vomiting, minute doses of nitrate of silver are more useful than any other remedy. Occasional fractional doses of gray powder may be serviceable when there is tendency to constipation, and smaller doses of calomel with sodium bicarbonate are often better, or, if the bowels are loose, bismuth quiets both stomach and bowels. When the inorganic acidity is subnormal, hydrochloric acid may be used and may quiet an irritable stomach by lessening its labors. With this pepsin may be combined. When there is atony rather than irritation, the last-named digestive principles are frequently valuable, or some of the bitter stomachics, such as nux vomica, may be used as stimulants; and, although Reichmann has been unable to find experimental proof of its stimulant action, I feel convinced that sodium bicarbonate given with bitters increases their effect.

If there be hyperacidity from any cause, the alkalies are indicated, the choice between these laxatives and others depending upon the state of the bowels.

Fermentation and flatulent distention of the stomach and bowels may often be much relieved by the digestive aids just mentioned or by creosote in minute doses in emulsion. Carbolic acid and other antiseptics furnish very useful means of controlling this to some degree, though sometimes lavage is much more important than any of them. Marked loss in general nutrition may be met by administration of cod-liver oil, which is much more frequently received kindly by the stomach than one would expect. It is best given in an acceptable emulsion.

Arsenic often gives good results if well borne, but is exceedingly apt to irritate the stomach, and the same may be said of iron; and none of these remedies should be persisted in if there is any sign of increased irritation. They have a wider usefulness when convalescence is well established. Begun then in small quantities, and increased slowly to fair doses, they often increase the progress most satisfactorily. Alcoholic stimulants may be required during the course of the trouble in the very weakly, but their use should be stopped as early as possible, as their effect upon the stomach itself is always bad, and surprisingly small doses are sufficient, if continued for a considerable period, to cause changes in the other organs of an infant.

Chronic indigestion in older children needs much the same manner of treatment, with details varied for the changed conditions. Again, the clothing next the skin should be woollen, of a weight suited to the atmospheric conditions, and bare legs and arms should not be allowed. Low slippers or shoes may not be worn. The ankles and feet need careful protection from draughts by high shoes and woollen stockings.

Bathing should be done daily unless there is much depression. With a stolid temperament cool sponge-baths, with lively rubbing afterwards, are often grateful and beneficial. If cool water be depressing or the child be of nervous temperament, we should use tepid or warmer water instead and gradually reduce the temperature, if no ill results follow, until it is about that of the sleeping-apartment. Innunctions are frequently of benefit, and the addition of salt or alcohol to the water will be often useful. The method of bathing should always be adapted to the case, and the objects to be kept in mind beyond cleanliness are a good skin circulation and avoidance of depression. If after the bath the child appear languid or depressed, another temperature should be tried, and if a cool bath result in cold extremities or a little cyanosis it should be made warmer.

It should be seen to that the child never exhausts itself in play, and with the dyspeptic in especial exercise soon after meals should be prevented. If the child show the contrary tendency and be languid and indisposed to play, it should be encouraged to join its fellows in mild sports, and regular exercises should be as far as possible instituted. With the exercise abundant sleep must be always allowed, the child retiring early and resting after meals. During any exacerbation, and at any time if fever be present, rest in bed is to be enjoined.

The consideration of the diet for older children is so broad that it must be merely touched upon. Regularity in meals must be exact, and they should be partaken of slowly and with thorough mastication. Four meals daily is a good rule for most cases, while some do better on three, and greater frequency and a correspondingly smaller amount at each meal will at times prove best. If acute exacerbations occur, they should be managed as acute dyspepsias, and the food restricted as may be necessary, even to its complete withdrawal for a time.

The variety in foods should be limited as far as possible without disgusting the palate, and the variety at each meal should be very limited. A dietary written for each case, with the times of meals and the kinds and amounts of foods, will do much to prevent unthinking departure from the rules. The kinds of food desirable in each case must usually be determined by the study of the symptoms and by trials, though the determination of the condition of the stomach secretions will give much aid in diet when it can be carried out. If the acidity of the stomach contents is determined and found high, farinaceous foods must be restricted. They by no means always agree well with low or normal secretion, however, and if fermentation be present, as it very commonly is, starches should be limited. Potatoes particularly are apt then to disagree. Bread may be borne only well toasted, and the greatest improvement comes from a diet of very soft-boiled or poached eggs, finely minced or scraped lean meats, raw oysters, the white meat of fowls or of fish of fine fibre, with vegetables limited largely to spinach, asparagus-tips, stewed celery, cauliflower, and sometimes raw tomatoes. A little rice may often be given, while fresh or hot bread must be prohibited, and only stale and, best, well-toasted bread allowed. Soda-crackers are sometimes acceptable and well digested in place of bread, and may also be toasted. Milk should be taken in goodly quantity, and to it some of the prepared foods may be added. If necessary, it may be diluted with plain or effervescing waters or lime water, or it may be peptonized. Some variety may be added by light desserts, such as rennet, custards, or milk puddings containing little starchy ingredients, and not made too sweet.

Not infrequently, however, eggs in particular disagree, and milk cannot always be taken, or albuminous diet in general may be insufficient for the child's nourishment or may increase the symptoms. In the latter case more starches must be added, largely in the increased amount of stale bread and of potatoes, baked or mashed, with the meats restricted somewhat and confined to those readily digested, such as have been already mentioned. Tea and coffee should not be allowed in any young children, and in dyspeptic children beverages should be restricted to milk and water, taken quite warm or moderately cold. Immoderate drinking of ice-water and other cold fluids is a common cause of dyspepsia in this country, and is not to be allowed, but the quantity of liquids may have to be increased. If the urine is scanty and concentrated or the bowels costive, liquids should be increased, while with diarrhœa it may often be necessary to decrease them greatly.

The medicinal treatment is far better too limited than too free. Much irritability of the stomach is often best allayed by silver nitrate in doses of from one-forty-eighth to one-tenth of a grain, depending on the age, or, temporarily, opium may be added to this. Calomel, bismuth, and other sedatives may be used as conditions call for them, but irritation is very frequently concurrent with general nervous irritation or depression, and then general measures to tone up the system will have much better results than local medication. The more purely nervous forms of dyspepsia are best treated by general tonics, with baths, massage, and perhaps electricity to the epigastrium. Mental rest should be enjoined where there has been over-study.

For hypersecretion, belladonna may sometimes be tried with good results, but the cure of the local or general irritation is the only effective means against it. As antifermentatives, hydrochloric acid, sodium hyposulphite, carbolic acid, and creosote are the best. The latter has given me the best results used either in capsules or in emulsion with sodium bicarbonate, magnesia, or bismuth salicylate, depending on the state of the bowels.

The alkalies also may prevent fermentation or relieve the distress from it by reducing the acidity of the stomach contents.

The digestion may be aided by the administration of hydrochloric acid in large doses, and pepsin may be given with it. The stomach may be whipped up to increased action with bitter tonics, but only when it is not already irritated.

The nutrition may be much improved in suitable cases, and especially in convalescence, by cod-liver oil, strychnine, or nux vomica, and, when the stomach bears them well, by iron and arsenic. Malt preparations are very valuable in many cases. The use of alcoholic stimulants should be restrained to short periods, when weakness requires them, or to those cases in which the actual vomiting is relieved by small doses of iced champagne or weak brandy and water.

With the treatment of the stomach should go always the regulation of the bowels if, as is almost constantly the case, they are disordered. The constipation that is usual may generally be overcome by the careful addition to the diet of food which leaves some fecal residue, together with the institution of a proper amount of physical recreation and toning up the often relaxed abdominal muscles by their special exercise. Friction, massage, and electricity will often give good service. The increase of liquids ingested, or even a glass of water drunk hot before breakfast, will be sufficient in some cases.

If these measures fail, gluten or glycerin suppositories or occasional enemas are to be much preferred to laxatives by the mouth. In the rare cases in which we are obliged to use medication, small doses of Carlsbad salts in hot water or cascara in the least dose that will act are to be preferred.

NERVOUS VOMITING.

Definition.—Vomiting which is not due to either functional or organic disease of the stomach.

Etiology.—The causes are to be divided into organic or central and reflex. Of the former meningitis is of most importance in gravity and frequency, while tumor and abscess of the brain are the other organic changes which may be wisely sought for in children. Disturbances of the cerebral circulation are not uncommonly followed by vomiting, and it is probable that concussion of the brain often acts in this way, though unknown cellular changes can scarcely fail to have a part. Vomiting caused by strong emotions, of which fright is the most active, is probably due to circulatory changes, and heat and cold seem to produce this occasional effect in the same way. Some toxic drugs cause centric vomiting, and that well-defined union of symptoms known as migraine, of which vomiting is so prominent a member, appears frequently to be due to the action of toxic products of perverted metabolism. The same is true of the vomiting in many acute and chronic diseases.

The reflex causes are without end. Of especial importance among them are those acting through the special senses. The rapid movement of objects before the eyes, as when travelling upon railways, or watching monotonous repetition of movement, as exampled by the waves of the sea, is a frequent cause of vomiting, and sea-sickness may be classed here as probably due to disturbance of our sense of equilibrium resident in the semicircular canals; but children are fortunate in being less susceptible to this disturbance than adults. Smelling certain odors may result in emesis. Reflexes from other organs when diseased or irritated frequently cause vomiting. The irritation of intestinal parasites, affections of the peritoneum, and appendicitis are common among these, and intestinal obstruction causes most grave emesis. Of the other organs, those of the respiratory system have considerable prominence in causing this affection, and especially in those of nervous mould, irritability of the nose or larynx, or their disease, occasionally causes vomiting by reflex disturbance, and an irritable pharynx is even more active. But to complete the enumeration of the reflex causes were an endless task.

General states, such as hysteria and neurasthenia, are sometimes accompanied by nervous vomiting, and the especial form of the affection described as cyclical, periodic, fitful, or recurrent vomiting must receive a short special description.

Symptomatology and Diagnosis.—These two divisions of the subject may be treated of together, as the vomiting is merely a striking symptom of some extra-gastric condition that must be sought out.

But a few words on the regurgitation from an overfilled stomach that is so frequent in young infants. This is a purely physiological act that occurs after too large a feeding, and simply returns from the stomach, slightly or not at all curdled, the excess of milk. It is unattended by any further symptoms unless the overfeeding be continued for some time, and the restriction of the amount taken at each feeding is all that is needed for its future prevention.

Real nervous vomiting may be met with of all grades of severity. It may occur but once and be soon over, as in cases depending upon powerful emotional attacks, or it may recur frequently with the recurrence of the cause. Again, it may be continued for so long a time or be so gravely exhausting in its severity as to endanger or take life. Profound hysteria or organic disease is usually present in these rarer instances. Between these extremes are found all grades.

That it is nervous vomiting is determined by the absence of evidence of local affection of the stomach with signs of organic disease elsewhere situated or of depraved general nervous states, and in this we are aided by the peculiar characteristics commonly exhibited by the vomiting itself.

Short attacks in which the cause is evident are recognized at once. Thus, sea-sickness can cause no doubts, as a rule, and emotional vomiting and that due to rapid movement on railway and other conveyances ordinarily exhibit distinct dependence upon their special causes, as do many others of the reflex forms.

Under the title of "vomitus acidus" Rosenthal has described attacks of nervous vomiting occurring in school-children of from seven to eleven years of age which seemed entirely dependent upon excessive mental strain in study, and Leyden and others have observed like attacks from the same cause. General dyspeptic symptoms are usually present. Headache is frequent, especially in the attack, and vomiting is the most striking symptom, which may be periodic and recurrent or may occur daily. Rosenthal has observed extreme pallor of the skin, a slow pulse, which may fall to sixty in the minute, and exaggerated nervous excitability or depression. These attacks are incontestably the result of the nervous overstrain in school, but are more distinctly nervous dyspepsia than simple nervous vomiting. The suspicion of the dependence of nervous vomiting upon organic disease at once gives an element of gravity to the case, and the elicitation of positive grounds for diagnosing disease of other organs is often extremely difficult. The diagnosis of hysteria and allied states is very dangerous without careful attempts, at least, to eliminate all connection with actual disease of the stomach itself or other organs.

The absence of tenderness and distention of the stomach and of marked changes in its size, its secretions, and its motor power will often turn our attention to other causes of the vomiting. A suspicion of a nervous origin will be furnished by the peculiarities of the vomiting. Functional cases are frequently accompanied by a surprisingly slight effect upon the general strength and nutrition, and are dependent rather upon even the slightest mental and moral causes than upon either the quantity or the quality of the food. Not uncommonly all rational food is rejected, and only the most bizarre and seemingly impossible diet is acceptable. A curious selective faculty is sometimes shown, certain special articles of diet being rejected from several ingested at the same time. The vomiting likewise often occurs when the stomach is empty. Frequently the act is accomplished with the utmost ease, and it is apt to be without nausea or retching. In such cases we find no fever. Prostration and exhaustion are dependent upon the severity of the vomiting only, and there are often evident signs of hysteria or of affections related to it. The diagnosis of purely functional nervous vomiting must, however, be put aside until we have established the non-existence of disease. The exclusion of organic centric vomiting from tubercular meningitis will frequently give greatest anxiety. The vomiting here is usually projectile, sudden and forcible, and without nausea or retching. Fever is present, of irregular type, but continuing, and if we find an irregular pulse or respiration, and apathy broken by frequent moans or shrieks of agony, so striking a picture is presented that it is not readily overlooked or forgotten. The examination of the eye-grounds should be always undertaken in suspicion of this disease or of brain tumor or abscess, and the special mental and localizing signs of the latter affections will usually appear upon sufficient examination.

The possibility of the onset of acute infectious diseases must be foreseen, and dismissed only after repeated examination has failed to demonstrate them.

Abdominal affections other than those of the stomach are so important as to demand much more earnest endeavor towards their elimination than is usually accorded them, and signs of intestinal obstruction and appendicitis are to be particularly looked for by palpation of the abdomen and rectal examination. Intestinal parasites and irritation of the genitals must be excluded, and urinary examinations are always essential.

Prognosis.—Nervous vomiting depends upon such varied causes that a prognosis can never be safely stated until the origin is discovered. It is then usually included with the prognosis of the causal condition. Occasionally without organic disease the vomiting itself is so persistent or so violent that it may induce the gravest exhaustion, or even death. No rule can be established in such cases, and the practitioner must depend upon his observation of the individual resisting powers and of the gravity of the vomiting.

Treatment.—The main indication is to find the cause as soon as possible and, when feasible, prevent its further action or recurrence. But this may not be at once possible, and the vomiting, if continuing, must be stopped to prevent exhaustion or resultant inflammation of the stomach.

Local sedative measures, such as ice, small portions of hot or cold drinks, or applications to the epigastrium, often fail, as does medication with calomel, bismuth, and the like. More successful are nervous sedatives, such as the bromides of sodium, strontium, or potassium, and chloral is often superior to bromides. Laudanum in infants or morphine in older children is sometimes indispensable as a last resort, or should be used early if the indications are urgent. These are all usually best given by rectum, or, in case of morphine, hypodermically, but sometimes their direct action on the stomach is most useful. Used for this purpose, menthol also is effective. If exhaustion be sufficiently marked to require stimulants, iced champagne and dilute brandy and water, the latter given hot, are often well received by the stomach and may act as sedatives. If their use in this way is impossible, stimulation may be given either by rectum or hypodermically.

That no food should be administered while its ingestion prolongs or aggravates vomiting is a good general rule. When the vomiting is continued for but a short time food should be entirely omitted, and when more extended rectal alimentation is to be used if possible. But the exhaustion from lack of food may become so severe, especially if the rectum reject nutritive enemas, that we are occasionally obliged to risk irritation of the stomach in the hope that some food taken may be retained even when it is evidently irritating. The vomiting is sometimes continued by the exhaustion when food would relieve it. That peculiar food is not infrequently received by the stomach also makes a certain amount of experimentation in diet sometimes allowable, especially where the cause is found in functional states. When the stomach has been much irritated by the vomiting, subsequent carelessness in diet is very apt to lead to gastritis, and in all cases this sequel should be guarded against. The general management after vomiting has ceased is to be guided by the cause. In instances due to over-study the indication is clearly to take the child from school for a time.

CYCLICAL VOMITING.

Cyclical vomiting is a special form of nervous vomiting in which attacks of limited duration occur at irregular periods from no definitely determined cause. The attacks may be not distinctly cyclical in their occurrence, but their regularity is so frequently pronounced as to allow of the general application of this term. Some cases show no marked periodicity. The disorder is by no means limited to children, but occurs at all ages.

Etiology.—That the disorder is not primarily dependent upon disease of the stomach, either organic or functional, I am convinced, though observers as able as Fenwick describe it as a recurrent catarrh. The entire absence of gastric symptoms between the recurrences, and the frequent lack of any reason for accepting the existence of an inflamed mucous membrane during the attacks, together with the common evidences of derangement of the nervous functions, are to me sufficient proofs of its dependence upon influences beyond the stomach, though what these influences are cannot be stated. Some cases which have been published under this title may perhaps belong to another class of neuroses of the stomach; they are not improbably examples of periodic hypersecretion. There are others, however, of which I have seen several examples, in which there was apparently no abnormality of secretion. For these cases we have no adequate explanation. Nervous depression is the rule in the subjects of the affection, and exposure to cold, emotion, or other well-defined causes may bring on attacks, but in what way the result is produced is not known. Leyden's original explanation of the condition as a neurosis of the vagus is attractive, but leaves us in want of a knowledge of the nature of the neurosis. The vomiting may be eliminative of some ptomaine recurrently produced or accumulating in the system, and the increased toxicity of the urine observed by Herter in one of Holt's cases is an important support for this view, but the origin of such a possible ptomaine is unknown. Malaria in rare cases seems to give rise to it, and it may follow other acute diseases. Thus, one of Gee's cases could be connected with no cause other than an attack of measles which occurred twelve months previously, and I have seen it follow typhoid fever. Holt has noted the frequent coexistence of a uric-acid diathesis.

Symptomatology.—The attacks are very frequently preceded and accompanied by headache, which is at times of great severity. Pain in the abdomen, most marked in the epigastrium or around the navel, likewise often precedes the outbreak of vomiting; this may also persist throughout. The pain may be so severe either before or during the attack that its relief is the all-important necessity. Commonly, too, for a period of a few hours to a day before the vomiting comes on there are languor and mental depression, and the bowels are often constipated, though they may be loose or normal. The temperature is frequently found to rise preceding the vomiting, and rapid elevation during the attack, with often quite as sudden fall, may or may not occur. To these variable general symptoms efforts at vomiting are soon added. These occasionally, as in one of Gee's cases, result only in violent retching, but usually the contents of the stomach are expelled, followed by bile-stained or watery fluid, sometimes containing a little mucus, and the violence of the straining may be such that the vomit contains a little blood. The acidity of the vomit has no constant characteristics. There are great variations in the severity of the attacks, both in different individuals and in the same patient in different seizures. Thus, vomiting may be rather mild and continue but a few hours, or it may be of great violence for but a short time, while other cases go on for several days with more or less exhausting emesis. Food sometimes aggravates it greatly, though it cannot ever be said to start it. In other instances it is entirely independent of food, and retching persists with an empty stomach. More rarely taking nourishment into the stomach acts soothingly. The irritability of the stomach to food may vary at special times in the day. Thirst is usually very great in severe cases, and is difficult to relieve while the attack lasts.

When the expulsive efforts last for more than a part of a day the resultant exhaustion often grows alarmingly dangerous, and the patient may lie almost unconscious, with profoundly depressed expression and weak, irregular, or almost impalpable pulse.

The cessation of active symptoms is usually rather sudden, though the vomiting may have been lessening somewhat before it finally ceased. All that then remains is exhaustion, severe abdominal tenderness, and aching from the violent muscular effort, and gastric symptoms cease until the disturbance recurs. The one remaining evidence of derangement of the digestive tract that has been observed with some frequency is an abnormally light or clay-like color of the stools.

Diagnosis.—This is almost entirely included in the consideration of the diagnosis of nervous vomiting in general. Meningitis and nephritis are especially to be excluded. The examination of the urine and a little time spent in watching for other brain symptoms will do this. Acute indigestion is distinguished by the dependence of the attacks of this disorder upon some cause acting upon the stomach, usually errors in diet, and by the lesser severity and generally shorter duration. Gastritis has a longer course, with evidences of changes in the functions of the stomach, and epigastric tenderness and pain are frequently continuously severe. It, too, is dependent upon abuse of the stomach, and rarely as severe as cyclical vomiting often is. The latter has commonly a well-defined, self-limited course which often comes to a sudden stop.

Prognosis.—The prognosis of the attack depends upon the resulting exhaustion. As to recurrences, it can only be said that when definite causes are unknown a definite prognosis cannot be given. Speaking broadly, the outlook for escape from future attacks is commensurate with the improvement of the general health.

Treatment.—The vomiting should be controlled during the attack by some of the means suggested under the treatment of the general subject of nervous vomiting. To these I would add that antipyrin is especially worthy of a trial in this particular disorder, and that the bowels are so frequently constipated that their condition should always be investigated at once, and an overloaded bowel relieved by enema or quickly acting laxatives or mild purgatives. Indeed, if the vomiting be anticipated from the occurrence of prodromal symptoms, it may be sometimes aborted by administering Epsom or Rochelle salts or calomel in sufficient quantity to cause free purgation.

Recurrence must be striven against by the endeavor to increase the nervous power of the child and correct faults in metabolism. For both purposes daily spongings with brisk friction following are always to be used and proper habits of exercise instituted. Iron is indicated in anæmia, and often arsenic in this and other conditions of nervous depression, and strychnine and bitter tonics are frequently valuable. Lithia and alkalies or waters containing these improve the lithæmic, and in this constitutional tendency diuresis should be promoted by drinking water freely. The diet should include no sweets and but little starches, while liver, kidney, oysters, and other articles rich in nuclein should be eschewed.

GASTRALGIA.

Definition.—A neurosis characterized by lancinating pain in the epigastrium which occurs without known local cause.

Organic lesions of the stomach are at times found in cases that have had gastralgia, but they are not necessary to its causation; most of the cases of true gastralgia have come to autopsy showing no lesions, and when such are present it is necessary to show that it was a true gastralgia according to our definition of the affection, for in many such cases it is the pain of involvement of the nerves by organic disease. Disturbance of the gastric functions may be present or absent, and the synonyms which imply existence of such disturbance are inaccurate and should be discarded. Thus, "spasm of the stomach" is a name which arises rather from the patient's sensations than from the real existence of spasm. In spasm of the pylorus there is probably always some local cause. Cardialgia is a term more generally used for the burning pain felt under the ensiform cartilage, and due ordinarily to hyperacidity of the stomach contents,—the "heart-burn" of the laity. Gastralgia is opposed to this in having no constant relation with changes in the gastric acidity, and the pain in the neurosis is more extended and may affect any part or the whole of the epigastrium. Stomach-colics from indigestion are entirely separate, and the term colic with any adjective has no place here. Of the English synonyms used none are acceptable but neuralgia of the stomach and gastrodynia.

True gastralgia is rare in children, as are other forms of neuralgias, but its occasional occurrence makes a description of it necessary to prevent confusion with other affections.

Etiology.—The causes of this symptom are as various as are the causes of other neuroses.

A neurotic ancestry is undoubtedly the usual history. When subjects of gastralgia have not sprung from a stock exhibiting much disease or irregularity of the nervous mechanism, depressing overstrain or enervating excesses may usually be traced in their elders. An unstable temperament results in the child, which is often intensified by the indulgences allowed by parents or by their poor judgment in rearing their offspring. Primary anæmias or those of secondary nature and the debility of disease or ill nutrition frequently exist. Girls are more disposed than boys to gastralgia, as they are more liable to all functional nervous affections.

One infectious disease in particular has a definite relation with it, for the paroxysms of a malaria may, as Niemeyer has pointed out, be marked by neuralgias in lieu of a chill.

Emotional scenes and shock may inaugurate gastralgia of all grades from the most fleeting to those of extreme persistence, and it is at times a form of traumatic neurosis. The abuse of alcohol, while frequently active in adults, is too rare in children to need attention, but the immoderate use of tea and coffee is not uncommonly the cause, particularly in the lower classes, who so often allow large quantities of these drugs even to very young children. In the later stages of childhood, as in adults, I have frequently found gastralgias due to tobacco, more commonly when used in the form of cigarettes, and Stillé makes this abuse the most common cause in adult males in this country. In persons predisposed to the affection there is no doubt that the ingestion of indigestible food may cause attacks. Whether continued dietetic errors can by local irritation cause the affection is with difficulty decided, as the pain from indigestion is hard to eliminate in coming to such a decision. Such a result is probable, but certainly not common.

Symptomatology.—The supreme symptom is the pain, and to this are due all the other manifestations in the usual form of the attack. With the stomach either empty or full, without apparent cause, sudden pain shoots through the epigastrium, of such severity that with a scream of severe suffering the child writhes about, or, doubling up the body, presses its hands forcibly against the epigastrium, the countenance expressing its torment. The pulse at this time is always tense and usually slow, probably from stimulation of the pneumogastric. Sometimes it is excessively rapid. This picture may be continued for hours almost without variance, or there may be successive darts relieved by deceptive intervals of apparently final cessation. Again, it is very evanescent, and there is but a scream and a flash of pain, with which the child stops its pastime, and all is then over but the start and the fright. With any severity of pain vomiting occasionally occurs. With the continuance of the suffering comes exhaustion, with pallor and cool, relaxed skin, and flagging pulse. The pain may radiate to the chest, and in uncommon examples to the shoulders and arms. Very neurotic children not uncommonly show dyspnœa and irregular heart-action before or during the attack, and with an hysterical tendency any manifestation of that disorder may appear.

Physical examination of the abdomen will reveal nothing more than retraction, or, with exhausting protraction of pain, relaxation. Tenderness is not usually present. There is at times, especially in the evidently hysterical, an excessive superficial hyperæsthesia, but pressure commonly relieves the pain.

Attacks of gastralgia frequently recur, but neither its reappearance nor the time between attacks can be foreseen. It is usually of very irregular recurrence, though occasionally almost periodic.

Diagnosis.—The exclusion of other affections of the stomach is comparatively easy.

Gastric ulcer bears greatest resemblance in the character of the pain, but this disease is almost always accompanied by other characteristic symptoms, such as hæmatemesis, more constant, severe, and localized tenderness, pain depending on ingestion of food, peculiar vomiting, and disturbance of the gastric functions between attacks. Gastritis and dyspepsia have far less severe pain, as a rule, are less sudden in onset, and have fever and physical signs and symptoms of disturbed function.

Other diseases than those of the stomach are often much more difficult of exclusion.

With fleeting attacks the resemblance to *petit mal* may be great, and the exclusion of the latter is of extreme importance in prognosis and treatment. Such cases must be decided by continued watching, with the comforting reflection that this especial form of pain with minor epilepsy is very uncommon. I have known of but one such case. In this the child would suddenly cease its play, give a scream of pain, and return to its toys. It afterwards passed into major epilepsy and died.

Intercostal neuralgia is differentiated by the different location of the pain, the interference with respiration, and the presence of hyperæsthesia or painful points along the course of the intercostal nerves. Hepatic colic has usually icterus, some considerable prolongation of the pain, tenderness in the hypochondrium, clay-like stools, and often gall-stones in the fæces.

Epigastric hernia, on which Vulpius, Roth, Kuttner, and others have so strongly dwelt, is usually distinguished by the hernial protuberance.

Treatment.—The relief of the pain, if still continuing, is the first necessity. Hot applications often relieve greatly. If there be a pronounced hysterical element, vigorous counter-irritation with the actual cautery may be wonderfully effective, while most cases are benefited by hot mustard plasters, and in protracted pain by tincture of iodine. Hot drinks without medication may be sufficient internally, but small portions of whiskey or brandy given hot, or a few drops of chloroform, will do better. Acetanilid, phenacetin, exalgin, and like preparations will often relieve continued pain, while laudanum, codeine, or morphine may be used in severe or protracted cases, though, as in all conditions liable to recurrence, they are to be avoided when possible.

If suspicion exist that the attack is due to indigestible food, it is well to empty the stomach by lavage or emesis, or to administer a brisk purgative.

To prevent recurrences, tea, coffee, and tobacco should be prohibited and errors in diet corrected, and especial attention should be given to hygienic details.

Of drugs arsenic is perhaps most useful, and iron should be added in anæmia. Cod-liver oil, with hypophosphites, occasionally gives even better results, alone or with the conjoined use of arsenic and iron. Subnormal gastric functions should receive attention, with hydrochloric acid, pepsin, and stomachics, and irritability reduced by silver nitrate, bismuth, or other sedatives. The tendency to gastralgia is sometimes overcome by lavage, douches, and electricity.

Only in highly neurotic subjects and when other means are unsuccessful is it wise to resort to bromides, valerian, and the like, and then they should be used only temporarily and omitted as soon as possible.

ORGANIC DISEASES.

ACUTE GASTRITIS.

Definition.—An acute disturbance of the gastric functions arising from inflammatory lesions of the stomach and accompanied by symptoms of inflammation of that organ.

Etiology.—The same causes that bring on attacks of acute dyspepsia are active in producing gastritis. In such case they are usually either brought to bear upon less resistant subjects or are themselves more violent. The same predisposing causes are found in physical imperfections of ancestors, or in acquired frailty of constitution of the child, induced by disease or bad hygiene.

The active cause of simple gastritis is commonly found in errors in diet or exposure to cold. Bad feeding is undoubtedly the ordinary cause in infants, and acts with great frequency in older children. The influence of damp and cold is, however, too frequently overlooked, and most cases may be traced to these exciting causes or to excessive heat, if palpable errors in diet cannot be found. Nervous influences rarely cause gastritis directly, but the dyspepsias which they frequently set up may produce inflammatory changes secondarily.

When the influence of dietetic errors, cold, and high temperature is excluded, the most common causes are the acute infectious fevers, among which influenza has recently gained an extremely prominent place. Some specific germs, as the diphtheria bacillus, may directly invade the stomach and give rise to a special variety of inflammation. The ingestion of toxic substances causes gastritis which may be indistinguishable either clinically or post mortem from the so-called simple form. Usually, however, such causes produce peculiar symptoms and readily recognized lesions.

Pathology.—In the simple form the mucous membrane is covered with a thick, sticky layer of mucus. The color may be even whiter than usual, or there may be much congestion, and very frequently there are small extravasations of blood and hemorrhagic erosions. The surface of the mucous membrane looks dull and opaque.

The microscope shows many cells and some blood-corpuscles in the mucus. The epithelium on the surface of the mucous membrane itself is frequently in great part lost, and the cells that remain are filled with mucus, cloudy or shrunken. The tubules have the lumen filled with a granular débris, the glandular cells are ill defined and cloudy, and the nuclei stain badly. The tubules themselves are but poorly outlined the one from the other. The interstitial tissues are filled with small round cells to a greater or lesser degree. Sometimes this interstitial infiltration is more pronounced than the lesions of the mucous membrane, or the contrary may be true. Usually the two have a nearly equal share in the picture.

Toxic gastritis may give the same appearances if the causal agent be non-corrosive. Some non-corrosive drugs, as alcohol and phosphorus, cause only fatty degeneration of the epithelium, which may not be evident without the microscope. Non-corrosive poisons, such as copper or arsenic, when present in such concentration as to give rise to very violent action, cause an intense, fiery injection of the stomach; corrosive poisons cause usually the same intense hyperæmia and more or less destruction of the mucosa, with formation of sloughs or dark eschars.

Phlegmonous gastritis may be present in one of two forms,—as a localized abscess or as a diffuse infiltration of the walls with pus. It is rare in either form.

Membranous gastritis has been found with diphtheria of the fauces, thrush, favus, scarlatina, variola, and other acute and chronic infectious diseases, and occasionally with a violent simple gastritis. At times there seems to be little involvement of the mucous membrane. Then the false mem brane alone is discovered. In other instances there are cellular changes and much infiltration of the interstitial tissue, and the membrane is strongly adherent to the stomach-wall. The favorite site for the membrane is the pyloric region.

Symptomatology.—The beginning of an attack of acute simple gastritis cannot be satisfactorily differentiated from acute dyspepsia. In infants dietetic errors, exposure to cold or to excessive heat, usually precede the onset with the symptoms that have been described in treating of acute dyspepsia. Often at the start the vomiting is more severe, and the vomit contains much mucus and even streaks of blood, but the vomiting of dyspepsia may be very violent. Tenderness in the epigastrium and pain are often quite severe in gastritis, less frequently so in dyspepsia. Only, however, after a day or two has passed do distinctive signs of gastritis appear. Then, instead of convalescence, we have a continuation of the symptoms. The vomiting persists, with perhaps less frequency, or, it may be, growing worse. Much mucus is found in the vomit, free hydrochloric acid is absent, and the combined acid is present in but small amounts, if at all. Lactic and volatile acids are usually found if food is being taken, but often none can be taken and the vomit consists only of mucus and watery bile. This inability to retain food persists much longer than with dyspepsia, and vomiting may be for days excited by any attempt at ingestion of food. In other cases it occurs with the stomach either full or empty, from the violence of the inflammation alone. Fever rarely remains very high for more than a day, though I have seen it touch 104° F. frequently for nearly a week. Its usual course is to drop to 101° or 102° F. after the first rise and remain about that point for a few days, dropping slowly to normal as the symptoms disappear. At times its course is very irregular, and some mild cases show even almost normal temperature, but the height of continued fever is a pretty good indication of the severity of the lesion in the stomach. Pain and tenderness are, as mentioned, apt to be marked from the beginning, and are often quite severe. As the case progresses and convalescence begins they decrease, and the pain does not usually continue long. The tenderness is inclined to persist longer in slighter degree, and a little tenderness or discomfort often remains even for some time after all active symptoms have disappeared. Epigastric distention is present from the beginning, though often not marked, and is apt to persist some days after vomiting and fever have ceased.

The more violent, at least, of the symptoms will commonly disappear within a week or ten days, and frequently the vomiting and fever in especial may not be continued longer than four or five days. There is always some weakness, however, and if the attack has been violent a good deal of exhaustion will remain. The irritability of the stomach will continue for some time, with few exceptions, and the least impropriety in feeding will suffice to cause distress, and, perhaps, to excite another pronounced attack. Many cases of acute gastritis go on into a chronic form, particularly if not managed with care during convalescence, active symptoms subsiding, but leaving behind them milder attacks of pain and distress after food, and irritability of temper, with a constant tendency to acute outbreaks.

Often before the acute attack the bowels have been constipated. During the attack, in infants, this frequently gives way to diarrhœa, and in the very young gastritis is not usually met with alone. The involvement of the intestine is apt to become the more persistent and serious part of the trouble, and many cases that begin with acute gastritis run on into chronic entero-colitis. This is not so common in older children, in whom the actual attacks are often less severe and diarrhœa as a troublesome sequel is less common. The vomiting and pain are with them the most marked symptoms, the pain being, of course, made more evident through their verbal complaints. They are less subject to elevations of temperature with slight cause, and the fever is, as a consequence, more regularly in accord with the degree of inflammation of the stomach. Many mild cases have but a degree or two of elevation after the first twenty-four or forty-eight hours, while severe cases may run as high as 103° or 104° F. a good part of the time for as long as a week. They are often somewhat stupid, particularly when the temperature is high, and, with the flushed face, the coated tongue, oftentimes diarrhœa, and the belly usually moderately distended and a little tender, it may be impossible for some days to distinguish the case from one of typhoid fever. After the attack has passed they are more apt to exhibit a strong tendency to further gastric disturbance than to bowel-involvement.

The complications, such as disturbances of pulse and respiration, which have been noted as occurring in dyspepsia are just as much to be expected in gastritis, and severe nervous symptoms are more common, as there is, as a rule, more violent cause. Convulsions occur with more frequency than in dyspepsia, infants being of course oftener the subjects than older children. The examination of the vomit or stomach contents in acute gastritis will show that there is some retention of food beyond the normal time. Chemically, organic acids, lactic, butyric, and acetic, will be found, while usually the total amount of hydrochloric acid will be low. With infants there is usually no free acid present, while with older children there will generally be but little.

With toxic gastritis, if the cause be a drug, there will, of course, be special symptoms with the gastritis pointing to the nature of the poison. With corrosive poisons one will find eschars of the lips and mouth, violent retching, and vomit containing blood and perhaps corroded bits of mucous membrane. Pain and tenderness in the abdomen will be severe and thirst excessive. Extreme prostration goes on to collapse and death almost invariably in young infants when the poisoning is from a corrosive agent or very severe from any poison. In older children recovery ensues in uncommon cases of corrosive poisoning, but with greater frequency when the toxic agent was non-corrosive. After the subsidence of the more violent symptoms there remain those of acute inflammation of the stomach, and often of the bowel, frequently accompanied by albuminuria and jaundice. In any case the convalescence is protracted; very often there remains a troublesome chronic gastritis which may strongly resist treatment, and recovery from corrosives usually results in contractures, which may be of no serious consequence or may go on to dilatation.

Membranous and phlegmonous gastritis are very rare, and cannot usually be diagnosed. The former often presents no symptoms, and may be discovered only in the course of an autopsy when death has occurred from other cause.

Usually it causes mild symptoms of gastritis, and more rarely a diagnosis may be made probable by the discovery of membrane in the vomit. Thomson has reported a case of tuberculosis in which a cast of the entire stomach was vomited.

Phlegmonous gastritis causes the symptoms of violent simple gastritis with those of septic intoxication. Occasionally in these very rare cases pus may be found in the vomit in large quantities, or a fluctuating tumor may be felt in the epigastrium if there be a localized abscess.

Prognosis.—Simple gastritis usually results in recovery. Very rarely the disease itself may be of such violence as to carry off the child, and with somewhat greater frequency the same end ensues more from the previous weakness of the child than from the actual severity of the acute disease. Convulsions may cause fatalities, as they may in dyspepsia, and any prominence of nervous symptoms or the actual occurrence of convulsions makes the prognosis grave until these are subdued. In the absence of severe nervous symptoms, if the infant or child be of fair or good constitution, the prognosis may almost always be stated as favorable so far as the immediate outcome is concerned. But any severe attack leaves a tendency, often for long afterwards, to recurrence of acute gastritis or of milder dyspeptic attacks, and every case is in danger of becoming chronic if care be not used in managing it to entire convalescence. Toxic gastritis is generally fatal in infants, as a sufficient amount of any non-corrosive toxic agent to give rise to a gastritis is usually more than they can bear, and the gastritis from such causes is a violent one. If the poison be almost at once removed from the stomach, they may recover. Corrosive poisoning is with them almost invariably fatal, but it occurs rarely. In older children the prognosis depends largely, of course, upon the nature and amount of the toxic agent and the violence of the symptoms when the child is seen. If recovery from the immediate effects be secured, one must always expect the usual course of a severe gastritis, followed by a protraction of the less severe symptoms over a long period, perhaps leaving a permanently inefficient stomach, or resulting in that gravest of all non-malignant stomach affections, a severe dilatation.

If phlegmonous gastritis be diagnosed with some degree of probability, the prognosis is at once very unfavorable. The membranous form of the disease will depend in prognosis upon the disease in which it has its origin.

Diagnosis.—The diagnosis of simple gastritis from acute dyspepsia has already been given. It depends upon the continuance of those symptoms whose persistence would denote inflammation,—*i.e.*, fever, vomiting, pain, and tenderness. If any of these be still present after two or at most three days, the case should be considered a gastritis, particularly if the fever or vomiting persist.

Tonsillitis and pneumonia are eliminated by repeated examination, and the eruptive diseases by the failure of appearance of a rash and of their other special symptoms.

A suspicion of tubercular meningitis may give anxiety for a longer time than with acute dyspeptic attacks, but the case has not the characteristic expression of this disease, the pain in the head is rarely tormenting, the pulse is usually regular, paralyses do not appear, and in a few days the symptoms, with uncommon exceptions, are much improved or have entirely subsided.

Typhoid fever is the most difficult disease to eliminate, owing to the irregular or abortive course it frequently pursues with children. With typhoid prodromal headache, languor, and nose-bleed are frequent, and vomiting is less common than in gastritis, though it does occur with typhoid. The symptoms are ordinarily more directly of intestinal origin, and diarrhœa and considerable general abdominal distention are the usual accompaniments. Stupor is more frequently marked with typhoid. Fever continued in the usual typhoidal course, enlarged spleen, and rose-spots would give firm diagnostic ground. The Widal reaction is very useful in cases that are protracted beyond a few days, if the reaction appear early. Unfortunately, however, the test is often positive only at a time when the course of the case would have settled the diagnosis. Still, remembering the frequent short course of the fever in children, subsequent application of the test will sometimes clear up a diagnosis that has remained doubtful even into convalescence.

Treatment.—The early treatment of simple gastritis is the same as for dyspepsia. Lavage should be used if possible, unless free vomiting has occurred. If this is not available and the stomach probably still contains irritating substances, free emesis should be induced by warm water or ipecac. After the stomach is empty, the child, who should be already in bed, is to be kept at rest and food withheld until the stomach grows entirely quiet. Further vomiting should be treated by bits of ice or by effervescing cool drinks, albumen water, and the like, already mentioned. Calomel in minute doses will be often useful, especially if there be constipation or offensive diarrhœa. In other cases bismuth or oxalate of cerium will sometimes do better, or we may be obliged to resort to morphine, codeine, or opium, given by mouth, by rectum, or hypodermically. Infants usually get over vomiting better after withdrawal of all food for a day than with any medication, and this is always the most important direction to be given. But with the continuance of vomiting for a week or more we are often driven to try many sedative drugs in the endeavor to stop it and the resulting exhaustion.

Fever is best treated by sponging with cool or tepid water or alcohol and water, or by wet packs. But if it be very high or persistent, the full tepid bath should be used. Antipyretics are to be used very exceptionally.

The pain should be as far as possible controlled by the application of mustard or spice plasters, of stupes, or of dilute tincture of iodine. In older children a couple of leeches to the epigastrium may give much relief. Opium and its derivatives are to be used only when the severity of the suffering demands their administration.

The immediate treatment of toxic gastritis consists, of course, in the administration of the physiological or chemical antidote. Lavage should be done at once if it be known that the poison is not corrosive. If the nature of the poison be unknown, excoriations of the lips or mouth contra-indicate lavage. When corrosive poisons are known to have been taken, the stomach-tube is absolutely contra-indicated. After the antidote or lavage soothing drinks, such as milk and albumen water, should be administered, and the shock treated by stimulation, while the pain must be controlled by opium. The subsequent treatment is that of severe acute gastritis. The administration of food during any attack of gastritis is to be regulated after the manner suggested under Acute Dyspepsia. Frequent and small feedings are to be continued until all active symptoms have disappeared, and the amount increased only as it is evident that the stomach will permit of this. Older children must first add semi-solids to their liquid diet when convalescence has begun, and the return to full diet should be postponed for ten days or a fortnight at least. When matters have begun to mend, the common deficiency in secretions may be supplied by hydrochloric acid and pepsin, and after all irritation has ceased bitter stomachics and general tonics may be tentatively administered.

Complications during the attack, such as convulsions, should be treated as when they occur in acute dyspepsia.

CHRONIC GASTRITIS.

Definition.—A chronic derangement of the gastric functions which, with the accompanying symptoms, depends upon continued inflammatory changes in the stomach.

Etiology.—Any of those causes that have been given as active in chronic dyspepsia may result in gastritis. This may occur through the intermediate production of dyspepsia, in which the altered functions cause such severe or continued irritation that inflammatory changes ensue, or the immediate severity of the exciting cause may be such that a gastritis arises without preceding dyspepsia. In subjects possessing but slight resisting powers inflammation is brought about by causes which would in their stronger fellows give rise to only functional changes. Thus, those children whose ancestors have shown serious ills are apt upon the action of but slight causes to acquire gastritis, even though they exhibit no other disease themselves, and the premature or those of weak constitution are more ready subjects than those who begin life with normal powers.

Acquired disease in the child is very commonly the direct cause. Acute gastritis is very frequently subdued for the time, only to leave behind a chronic process, and many of the acute diseases have chronic gastritis as a sequel, either by first inducing an acute attack or by setting up slow and at first unnoticed changes. Influenza has recently added itself to the many others, and its influence is daily seen in the histories we obtain of symptoms of gastritis persisting after an attack of that disease.

Chronic diseases may cause simple functional troubles, but they usually produce tissue-change of a lower grade, and protracted cardiac or pulmonary disease commonly leaves post-mortem evidences of a distinct gastritis, while this ensues with even greater regularity upon nephritis or disease of the liver, and gastritis, as has been mentioned, is by some thought to be a specific lesion of constitutional syphilis. Improper dress and exposure are of still greater importance in chronic gastritis than in dyspepsia. If the child be well nourished and of good constitution, moderate exposures may result only in functional disturbance of its digestive tract, together with bronchitis and the like; but if such trials upon it be more severe, or act upon a more susceptible subject, the greater number will suffer by the production of gastritis. Naso-pharyngeal or tonsillar disease is sometimes the primary result, but these affect the stomach secondarily. But, as in all stomach affections, it is that which directly irritates or overtaxes the stomach which may be most commonly found at fault, and errors in diet are the usual excitants.

In infancy the pernicious tendency of many parents to give foods irregularly or to use such as are irritating and indigestible will be usually found, while in older children eating between meals, hurried mastication, or imperfect mastication from bad teeth, and indulgence in candy and other dainties are frequent causes; too large a variety at meals, including hot breads, pastries, and spices, together with drinking of tea and coffee, is common.

Pathology.—The lesions found will vary with the cause and the stage to which the process has advanced. Generally the stomach will appear of about normal size, and the walls often of normal thickness. The mucous membrane is usually dirty gray, and covered with a layer of tough mucus that is with difficulty removed. Small old extravasations of blood are scattered about over the surface, and especially is this the case when there has been much obstruction to the circulation from disease of other organs, as of the heart or lungs. In such case the vessels of the walls are deeply injected with dark blood and the whole organ is of a purplish color. With this there may be so much œdema or interstitial overgrowth as to mak the walls much thicker than normal. Some cases are of hypertrophic form, with walls of abnormal thickness; and in these, if the process be in the early stage, the hypertrophy will be largely caused by thickening of the glandular layer, which will be of pale-red or bluish-red color, of considerable density, and rolled into thick rugæ or projecting into little hillock-like or polypoid excrescences, with sometimes small projecting cysts from accumulation in the glands. The lymphoid follicles will be enlarged, and, according to the time through which the process has persisted, the interstitial tissue will be to a greater or lesser degree thickened. At a more advanced stage with atrophy of the glands, which is a rarer condition, the surface looks smooth and white, and here and there are white, fibrous-looking streaks. With this there may be much thickening of the other layers, and the whole wall may still appear thick, or the tissues may be all atrophied and the walls very thin and easily ruptured.

Microscopically one sees in earlier stages the same changes that have been described as occurring in acute gastritis. The glandular cells become cloudy, and the parietal and central cells are indistinguishable, while the surface cells are lost, or those remaining are distended with mucus or shrunken. The tubules beneath become in general less well defined, and have the lumen choked with débris and blood-corpuscles. Later the glandular cells undergo fatty degeneration, and fat-drops appear in the cellular remains. Finally the glands disappear, to be replaced by small shrunken sacs containing some débris and a few epithelial cells. Interstitial infiltration with round cells is present to some degree in the early stages, and later fibrous tissue forms between the glands, often compressing them and resulting in their atrophy from pressure or the formation of the cysts or papillæ mentioned. When the process has advanced to atrophy the glandular cells will have disappeared, being replaced by a layer of fibrous tissue upon the submucosa. These appearances will vary in different parts, in one place being far advanced, in another in an earlier stage, and atrophy in particular is rarely so general as to affect the entire surface. The parenchymatous changes, too, will be in some places the most marked, while in others the interstitial tissues will have suffered most. In some cases the process in general will be largely either parenchymatous or interstitial.

Symptomatology.—In the earlier stages the manifestations of the disease will be of somewhat irregular occurrence and of mild degree, depending often directly upon dietetic or hygienic errors, frequently decreasing, again growing more intense. Infants become pale and wan, and are frequently irritable. Their food disagrees and is often vomited, usually some time after its ingestion, the milk coming up sour and ill smelling; colicky pains occur at frequent intervals. They begin to lose flesh and grow flabby, and the vomiting becomes more frequent. Vomited matters or test-meals will show evidences of some retention of food in the amount ejected or obtained by expression and in the unpleasant sour odor and ill-digested appearance.

Free hydrochloric acid will be absent, and the organically combined acid will be present in reduced amounts, while lactic acid will be found in some quantity. Butyric and acetic acids will be often noticed in the odor and found by chemical tests. There is usually much sticky mucus, which prevents rapid filtration and lends a glairy consistency to the whole mass. If the stomach be examined after some hours' fast, as in the morning, it will still contain some curds of milk, mucus, and a little watery fluid. The condition grows worse, and slight elevations of temperature are often found at intervals, while the loss of flesh and of muscular tone continues. The appetite at the start is usually capricious, and the infants sometimes nurse or take their bottles with avidity, while again they will not take food or will soon cease nursing.

The tongue is coated with a dirty yellow fur as a rule, the epigastrium is somewhat tender, and the infant cries upon palpation of its abdomen. The belly protrudes from distention both of the stomach and of the intestines, the latter becoming filled with gas which is often regurgitated or passed as flatus. The bowels are more commonly confined than loose in the earlier stages, but very often the imperfectly prepared food that is supplied the intestine soon causes diarrhœa, and this frequently persists. The bowels rarely escape organic involvement in long-continued gastritis, and, if the intestinal change becomes seriously extensive, the emaciation proceeds to an alarming degree, the face becomes old and pinched, the fontanelle sinks, and the child falls into a marasmic state, unable to digest any food that is given it, and, with its strength slowly slipping away, finally dies almost a skeleton. If, however, the process is earlier arrested by careful management, the vomiting grows less, the child retains some suitable food and slowly regains a little flesh and color, and more sufficient secretion will be found in the stomach contents. The favorable course will be often arrested by new outbreaks, unless the greatest care is used in feeding and clothing and in the details of the child's life, and prolonged recurrences are frequently caused by seemingly slight deviations from a rigid routine. If the symptoms all disappear, and the hydrochloric acid in the contents of the stomach regains its normal percentage with the disappearance of organic acid, excessive mucus, and fermentation, careful watching will still be necessary for a long time, to prevent some exposure to cold or to high summer temperature, or a departure from proper diet, from setting up a recurrence of the old trouble. A child who has once had in infancy a chronic gastritis retains for years, and perhaps for life, an especial susceptibility to renewal of its gastric symptoms.

Older children begin with languor and depression after eating, often accompanied by some pain and distention and a feeling of weight in the epigastrium. In the mornings, especially, they have little appetite and are often nauseated. Their nights are restless, and sleep is unrefreshing and often accompanied by frightful dreams. In the morning hours they are depressed and stolid or irritable, while later in the day they regain their appetite and vivacity, and often indulge heartily at their mid-day or evening meal, frequently bringing on abdominal pain and distress. They often have attacks of palpitation with considerable prostration after eating. They lose strength and become pale and thin, and their tempers change. They frequently have headache and giddiness and are disinclined to play. Vomiting is not so regular and frequent a symptom as with infants, but occurs at intervals, usually after heavy indulgence in food, or in the more pronounced stages often coming on in the early morning or after breakfast. Eructations of sour or bitter material are common. Thirst is usually excessive, frequently extremely so, and the child indulges in large draughts of water whenever permitted to do so.

The breath becomes foul or heavy, the tongue is coated, and there is a more or less constant bad taste in the mouth. The epigastrium is usually a little tender and swollen, and the whole abdomen is often protruding and tympanitic.

The bowels are frequently constipated and the movements often hard and painful and frequently suppressed for several days, thus adding to the gastric and intestinal symptoms. Diarrhœa sometimes occurs, and a few daily semi-solid stools are often passed, or there are short attacks of more severe degree, but prolonged diarrhœa is not so frequent as in infants.

From time to time the symptoms become aggravated, and acute outbreaks are apt to occur upon very slight cause.

The examination of the vomit or stomach contents after a test-meal will show particles of poorly digested food in considerable amounts and exhibiting little change but maceration since their ingestion. Unless the food contained much lactic acid, this will be found in but small amounts, and the volatile acids will require for their formation a considerable degree of retention of food. Starches will be often well digested, since the salivary secretion is not usually deficient and the gastric acidity is subnormal, and for the latter reason the albuminous foods are commonly but imperfectly changed. Hydrochloric acid will be below the normal in total amount, and the free acid at times present in small quantity, at times absent. Pepsin is present in amount largely proportional to the amount of hydrochloric acid. Mucus is very frequently found in excess and very stringy and thick, and if the stomach is examined in the morning, instead of its being found empty, mucus will often be removed in some quantity. In very advanced cases that go on to atrophy the hydrochloric acid disappears entirely, as do the ferments, and mucus is no longer found.

Diagnosis.—The symptoms cannot be attributed to a simple chronic dyspepsia when such as have been related are present with the constant changes in the secretions of the stomach. The constancy of the failure in health, and greater regularity in the symptoms, with occasional vomiting, continuous subacidity, and excess of mucus, are sufficient to distinguish the organic disease from the functional.

As the severity of the disease is greater than that of dyspepsia, so is the necessity for eliminating a dilatation greater and more usually brought to mind, and this must, when possible, be done by the methods of examination of the stomach and its contents which will be detailed later. Organic diseases of other organs, as of the lungs, the heart, and the liver, will be excluded by repeated physical examinations of those organs, and the examination of the urine will rule out nephritis.

Prognosis.—In general the prognosis is good in infants if it is possible to institute proper hygiene and diet. Many cases will require for their improvement removal from their usual surroundings to more healthful locations, and particularly city babies will often continue ill or grow worse unless country air can be secured for them, and the prognosis thus becomes in many cases dependent upon the length of the family purse. Generally in the earlier stages they will with proper management recover, needing constant watchfulness and care until all symptoms have, after slowly decreasing, long disappeared. Cases of moderate severity and protraction will usually recover if they are given the necessary care, and if, when necessary, change can be secured, but it will require great exactness in following rules and judgment in establishing a routine to secure continued improvement. When the bowels have become much involved and the infant has grown marasmic, the outlook is bad. Only most conscientious treatment and a long struggle will suffice to secure a slow return to health, with the course of the improvement frequently interfered with by the return of vomiting and diarrhœa over longer or shorter periods.

In older children there is rarely any danger of a fatality from the disease itself. It always renders them more liable to other diseases, and such disease occurring in a child already weak and ill nourished is of course more apt to be severe, perhaps fatal. When the parents and the physician act in accord in regulating the diet and other details of the child's life, entire recovery may be confidently expected as a rule, though often protracted. Generally, however, little indulgences will be allowed from time to time, and it will be difficult to secure complete good health. Unless the disease be entirely overcome in early childhood, it very frequently persists in causing more or less discomfort and ill health throughout a good part of life.

Treatment.—The first things to be seen to are the regulation of the diet and the general care of the patient.

With chronic gastritis there should be no departure from the rule that woollen clothing be worn next the skin of both the body and the limbs, the thickness being varied, of course, for varying temperatures. The usual bath may be supplanted by daily sponges with infants, using water of about the body-temperature. Cooler water is usually beneficial for older children. The sponging should be followed by gentle massage and friction. All exposure to draughts and to dampness should be avoided, and if the dwelling be unhealthful a change should be advised. Infants in particular often show no tendency to improvement until they are removed from towns or cities into the country. The diet of infants should be regulated upon the principles which may be found in the articles devoted to that subject. Artificial foods must be more dilute and readier of digestion than for babies of the same age who are in good health, and, whether they are artificially fed or at the breast, they must receive their food at exact intervals. The time between meals will in some cases be prolonged with advantage, while others will do better if the meals be smaller and more frequent. If artificially fed, and dilute milk or milk and cream mixtures do not agree, these should be peptonized for a time, and if acceptable in this form continued, but predigested foods should not be persevered in longer than necessary. Often the milder dyspeptic symptoms will persist for some time when such foods are being taken, while they disappear when undigested milk is substituted, as the latter provides the natural stimulus for exciting stomach-functions.

The diet of older children is to be based upon the principles cited in dealing with chronic dyspepsia. If possible, our schemes for food should have the support of previous examinations of the stomach contents, and if, as is usual, the acidity be found low, all forms of meats that are slow of digestion should be entirely excluded, and only moderate portions of the lighter kinds of fish and poultry and a little rare scraped beef allowed, while the more digestible starches, such as toasted bread, mashed and baked potatoes, and well-boiled rice, may be allowed in larger amounts, well chewed before being swallowed. Light custards and milk-puddings will aid in variety. All sweets, pastries, and hot breads must be excluded, as must tea and coffee.

In chronic gastritis, lavage is of great importance with infants and where it can be used in the older patients. It will not do good in every case, and its effects should always be watched. Sometimes actual harm ensues, when it must be stopped, but usually, by removing the mucus and food remnants and by its tonic effect upon the stomach, it very distinctly relieves the symptoms, and subsequent examination of the stomach contents will demonstrate the improvement of the secretions. The frequency of its use is best governed by the effects. Usually once in two days acts rather better than greater frequency at first, though if there be much accumulation of mucus it is well to undertake it daily. With the improvement in the symptoms, once or twice a week will be sufficient, and it should be stopped when indications for its use cease. It is often well, when there is much mucus, to add about one per cent. of sodium bicarbonate to the wash-water. Older children who will not allow lavage may use a poor but more agreeable substitute in a glass of hot water, best containing a little baking soda.

Medicinal treatment is usually too freely depended upon, and should be limited to that distinctly indicated. Much vomiting and painful irritation are commonly best overcome by small doses of silver nitrate, with or without minute portions of opium. Occasionally other sedatives, like hyoscyamus and the bromides, do better; or when vomiting depends, as it not infrequently does, upon depression rather than irritation, minute doses of ipecac or capsicum, with bitter stomachics, will be more useful. When irritation is not evidenced, the stomachics before meals are valuable, best given with moderate doses of bicarbonate of sodium. Hydrochloric acid and pepsin are very often indicated to supply deficient secretions, and their administration may give much relief. Frequently, however, bitter tonics, such as nux vomica, do more good. Fermentation is best treated by lavage. When this may not be used or the fermentation is not very troublesome, creosote, carbolic acid, large doses of hydrochloric acid, or other antiseptics may take its place.

The bowels, whether loose or confined, should be kept regular by some of the means already suggested. The use of laxatives for any length of time is rarely permissible when the stomach is chronically inflamed, and proper diet, massage, and exercise will often regulate constipation. If they do not, enemata or suppositories should be tried first.

SIMPLE GASTRIC ULCER.

Definition.—Simple ulcer is a localized tissue-destruction in the stomach, tending towards perforation, occurring without dependence upon any specific disease, and probably due to the action of the gastric juice upon an area insufficiently nourished.

Its occurrence in children has been but rarely described. Undoubted cases are, however, on record. The difficulty in eliciting symptoms in children, the frequency with which, at any age, it shows no characteristic signs during life, and the occasional discovery, at autopsies upon children, of the results of unsuspected and cicatrized ulcers make it extremely probable that the disease is overlooked with much more frequency than is usually admitted.

Etiology.—The causes that may produce the proper conditions for the formation of a gastric ulcer are in many cases quite well recognized. In chlorosis its occurrence is more frequent than with comparatively normal blood-conditions, and in various other forms of anæmia it is especially apt to develop. Insufficient nourishment and improper foods may cause it. Of the latter, hot spices and food in general taken too hot seem especially active, and hot tea and coffee have the same evil local effect, with their added results upon the nervous system. Injuries are likewise causative. Accidental injuries or burns of any part may be thus active at any age, while injuries from occupation are much more common in adults.

Sex is a factor in its causation, as females are much more commonly subjects of it than males, and children have seemingly a somewhat protective factor in their age. Both the latter are certainly largely due to differences in the conduct of life in the two sexes and at different ages, and the same is true of the influence of race. Among remaining causes acute infections are of considerable importance.

The actual pathogenesis is uncertain. Virchow's theory that the cause is obstruction of an artery from thrombosis or embolism by a clot is upheld in some cases by the presence of such obstruction, but not in all. Microbic embolism and the direct action of micro-organisms upon the tissues cannot be usually established even as probabilities. Inflamed lymphoid follicles and ruptured abscess in the follicles may result in ulcer. This is evidently an occurrence readily possible in acute infections. All these causes are probably active at times, but even then will not result in progressive ulceration unless certain other conditions are present. Such conditions may be at times found in the lessened alkalinity of the blood in anæmias or in the hyperacidity commonly present. Neither of these latter is, however, constantly present, and probably mechanical or other interference with the nutrition or continuity of the mucous membrane is first necessary, followed by the effect of hyperacid gastric juice, or of subalkalinity of the blood, or by both the latter in most cases, while some instances still need a complete explanation.

Morbid Anatomy.—There is usually but one ulcer, and in no case in children where the nature of the lesion was well established have more than two been described. Barlow has reported a case in which there were five ulcers, but there is strong probability that they were tubercular. The size in those reported has varied from one-eighth of an inch to two and a half inches.

In both adults and children their usual location is the pyloric region, frequently on the posterior wall near the lesser curvature, though the examples which have been observed in children show somewhat more irregularity in location than is frequent in adults.

Recent ulcers have usually sharp edges and look somewhat as if cut out with a punch. The sides slope slightly, giving a somewhat funnel-like shape, and the base is commonly smooth. Older ulcers have indurated edges which are often terraced, and the character of the base is determined by the depth to which they have gone, as the underlying organs are often adherent.

Simple erosions are distinguished by their usual small size and little depth, with no appearance of chronicity or advance. They are often multiple, and frequently over the fundus.

Gastromalacia must not be confounded with this lesion. I have recently seen at post-mortem a perforation of the wall of the stomach which at first sight was exactly like that from ulcer, but was characterized, as this post-mortem change is always characterized, by the pulpy and gelatinous condition of the mucous membrane and the lack of inflammatory reaction. It is, too, usually at the fundus, and the perforation irregular.

Symptomatology.—When the characteristic symptoms of ulcer appear, they are much like those in the adult, and need not be elaborately described. They are pain, tenderness on pressure, hemorrhage, and vomiting, the latter occurring with peculiar relation to the pain.

The latter symptom varies from a mere gnawing or burning to grades of a severity resembling violent gastralgia. It is more commonly severe, and is usually made worse by pressure or by food and relieved by vomiting. Frequently it is localized in some one constant and definite spot. In mild degree it is sometimes almost constant, but usually there are violent attacks after meals and other exciting causes, with little or no suffering between.

Tenderness is more apt to be absent than is pain. When it exists at all it is more persistently present than pain, and so great that neither palpation nor the pressure of tight clothing can be borne. Its location is usually the same as the pain. There may be a very tender area in the back near the last two dorsal vertebræ, such as Boas has described in the adult.

When hemorrhage occurs, the blood is usually seen in the vomit, but may appear only in the stools. It has several times been the first distinct intimation of the existence of gastric ulcer in children, as it often is in adults. The amount is frequently enough to be alarming, and it is a common cause of death in ulcer or of severe resulting anæmia. Only very small amounts of blood are lost in some cases, but then the hemorrhage is not distinctive of ulcer. When vomited, the blood is bright red, unless it has remained in the stomach some time. In the latter case it is dark red or nearly black. When it appears in the fæces, it is always dark and tarry. Hemorrhage is very prone to recur.

The vomiting is somewhat characteristic in that it does not occur with the beginning of the pain nor directly after meals, but usually comes on at the height of the paroxysms of suffering, and after the stomach is emptied it generally ceases. The pain is at least much relieved and often over for the time. The vomited matter commonly has a high acidity, in greatest part due to hydrochloric acid.

In infants and young children these four distinctive symptoms are often absent, or those that are subjective cannot be elicited. There are, however, always dyspeptic symptoms of varying kinds and degrees, and the appearance of any one of the more certain signs should always make us extremely careful in prognosis and treatment. Hemorrhage in any considerable amount, unless from some other discoverable cause, should at once call for treatment for ulcer. In the very young, pain after the ingestion of food that is not otherwise evidenced may be shown by persistent refusal to take food even when the child is plainly hungry. In chronic cases where adhesions have taken place the formation of a palpable tumor may give opportunity for a correct diagnosis. Other cases may give suspicion of a preceding ulcer only when stenosis of the pylorus and the resulting gastrectasia appear, as they sometimes do. Such a case I saw recently at autopsy. There had been from early childhood an indefinite history of dyspeptic symptoms, and the patient died at about twenty from dilatation of the stomach. At post-mortem we found the pylorus almost completely obstructed by the cicatrization of an old ulcer and an enormous gastrectasia. Chvostek has reported a similar case.

At any time the case may terminate by the occurrence of fatal hemorrhage or perforation. The latter will be recognized by the sudden abdominal pain, tenderness, and distention, with, usually, disappearance of the liver dulness, associated with which symptoms are the evidences of collapse in the pinched and anxious countenance, fall of temperature, and coldness of the extremities. Subnormal temperature may be replaced later by fever. The skin is often bathed in cold sweat and the pulse always rapid and weak at first. Subsequently perhaps the tense pulse of peritonitis replaces this, to grow soft again as death approaches.

Diagnosis.—This must rest upon the occurrence of one or more of the four characteristic manifestations with coexistent dyspeptic symptoms. Hemorrhage, as stated, is of itself sufficient to necessitate a diagnosis of ulcer and the use of the appropriate treatment when there is no other evident source of the bleeding.

The diagnosis in the absence of hemorrhage is much less certain, even though pain, tenderness, or vomiting be present. The pain of gastralgia is separated by its peculiarly nervous character and less definite location, its irregularity of occurrence, and its entire independence of other gastric symptoms or the ingestion of food, as well as by the common evidences of a perverted nervous system.

In gastritis and dyspepsia there is much less pain, the vomiting is of different character, and the tenderness less severe, and they usually occur with some variation of temperature, while uncomplicated ulcer causes no fever.

Biliary colic has been sufficiently considered under gastralgia.

Prognosis.—Death from hemorrhage is always to be feared, as is perforation, which latter is always fatal unless an early operation be undertaken, when the outlook is still gloomy in the very young, but more hopeful in older children.

The possibility of these fatal endings must never be put aside, and an exact prognosis cannot be given. In adults, fifteen per cent. or more of all cases diagnosed die, and such an average in children can scarcely be too high. Pyloric stenosis and contractures and adhesions of other portions of the stomach, with their usually grave sequelæ, are apt to occur, and it can never be promised that they will be escaped until long after all symptoms have disappeared.

Treatment.—The most active occurrences demanding treatment are hemorrhage and perforation. In the latter all treatment except operation is but palliative, and when a diagnosis of perforation is made surgical intervention is the only rational advice. Hemorrhage calls for absolute rest, the withdrawal of food by the mouth, and the application of a light ice-bag to the epigastrium. Hypodermics of ergotin and of morphine should be given in older children, while the same in very small doses, or astringents, like tannic acid, administered by the mouth, are in place with infants. If the hemorrhage be not controlled by these, small doses of well-diluted Monsel's solution may be used.

If hemorrhage has been severe, no food should be allowed infants by e mouth for twenty-four hours, and then the breast or bottle taken once hour for but a few moments. Food by the mouth should not be allowed der children for four or five days at least, alimentation and liquids by the ctum only being permissible. Small pieces of ice may be taken by the outh both for the thirst and for the hemorrhage.

After this time small portions of liquid food may be administered at equent intervals and a gradual advance made to a semi-solid but very and diet, which should be persisted in for some weeks, or longer if neces-ry for getting rid of all active symptoms.

Other cases which have but slight hemorrhage or none, but in which an lcer is suspected or diagnosed, should always be put upon liquid diet for few days at first, and unirritating semi-solids gradually substituted. It s often well, if any symptoms are severe, to precede this by rectal feeding or a few days.

Medicinal treatment should not be freely used. Silver nitrate given ver a period of several weeks is the most satisfactory. It should be taken n an empty stomach, and opium given with it if there be much pain.

Fleiner's treatment by very large doses of bismuth may be tried with much benefit in some cases. In others it causes severe constipation or diarrhœa, and at times extremely unpleasant feelings of pressure and weight in the epigastrium.

The bowels should in all cases be kept well opened. One of the best ways of accomplishing this is the administration each morning of a proper dose of Carlsbad salts. Sodium phosphate may, too, be readily administered in milk. Magnesia in various forms is useful both for its laxative and its antacid effects.

Special symptoms, such as severe pain and frequent vomiting, may need treatment. For either, silver nitrate, with small doses of opium or alone, is very useful. The pain when acute is better subdued by chloroform water, menthol, or exalgin: in more severe cases, morphine or codeine should be used by hypodermic injection.

The vomiting usually depends largely on the amount of pain. It is controlled by treating the pain or by local sedatives, such as calomel, oxalate of cerium, and sometimes cocaine, or, better, by liquid diet or rectal alimentation.

PYLORIC STENOSIS.

Definition.—Any decrease in the calibre of the pylorus which prevents the passage of the stomach contents into the intestine within normal limits of time and with normal muscular effort.

Etiology.—All the causes that may act in adults are likewise effective in children, and do produce this result, though more infrequent in early life. Cancer alone must be denied consideration, since its occurrence is so excessively rare. Ulcer has several times been known to cause severe stricture in childhood, acting thus either by means of the resulting cica-

At any time the case may terminate by the occurrence of fatal hemorrhage or perforation. The latter will be recognized by the sudden abdominal pain, tenderness, and distention, with, usually, disappearance of the liver dulness, associated with which symptoms are the evidences of collapse in the pinched and anxious countenance, fall of temperature, and coldness of the extremities. Subnormal temperature may be replaced later by fever. The skin is often bathed in cold sweat and the pulse always rapid and weak at first. Subsequently perhaps the tense pulse of peritonitis replaces this, to grow soft again as death approaches.

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The possibility of these fatal endings must never be put aside, and an exact prognosis cannot be given. In adults, fifteen per cent. or more of all cases diagnosed die, and such an average in children can scarcely be too high. Pyloric stenosis and contractures and adhesions of other portions of the stomach, with their usually grave sequelæ, are apt to occur, and it can never be promised that they will be escaped until long after all symptoms have disappeared.

Treatment.—The most active occurrences demanding treatment are hemorrhage and perforation. In the latter all treatment except operation is but palliative, and when a diagnosis of perforation is made surgical intervention is the only rational advice. Hemorrhage calls for absolute rest, the withdrawal of food by the mouth, and the application of a light ice-bag to the epigastrium. Hypodermics of ergotin and of morphine should be given in older children, while the same in very small doses, or astringents, like tannic acid, administered by the mouth, are in place with infants. If the hemorrhage be not controlled by these, small doses of well-diluted Monsel's solution may be used.

If hemorrhage has been severe, no food should be allowed infants by the mouth for twenty-four hours, and then the breast or bottle taken once an hour for but a few moments. Food by the mouth should not be allowed older children for four or five days at least, alimentation and liquids by the rectum only being permissible. Small pieces of ice may be taken by the mouth both for the thirst and for the hemorrhage.

After this time small portions of liquid food may be administered at frequent intervals and a gradual advance made to a semi-solid but very bland diet, which should be persisted in for some weeks, or longer if necessary for getting rid of all active symptoms.

Other cases which have but slight hemorrhage or none, but in which an ulcer is suspected or diagnosed, should always be put upon liquid diet for a few days at first, and unirritating semi-solids gradually substituted. It is often well, if any symptoms are severe, to precede this by rectal feeding for a few days.

Medicinal treatment should not be freely used. Silver nitrate given over a period of several weeks is the most satisfactory. It should be taken on an empty stomach, and opium given with it if there be much pain.

Fleiner's treatment by very large doses of bismuth may be tried with much benefit in some cases. In others it causes severe constipation or diarrhœa, and at times extremely unpleasant feelings of pressure and weight in the epigastrium.

The bowels should in all cases be kept well opened. One of the best ways of accomplishing this is the administration each morning of a proper dose of Carlsbad salts. Sodium phosphate may, too, be readily administered in milk. Magnesia in various forms is useful both for its laxative and its antacid effects.

Special symptoms, such as severe pain and frequent vomiting, may need treatment. For either, silver nitrate, with small doses of opium or alone, is very useful. The pain when acute is better subdued by chloroform water, menthol, or exalgin: in more severe cases, morphine or codeine should be used by hypodermic injection.

The vomiting usually depends largely on the amount of pain. It is controlled by treating the pain or by local sedatives, such as calomel, oxalate of cerium, and sometimes cocaine, or, better, by liquid diet or rectal alimentation.

PYLORIC STENOSIS.

Definition.—Any decrease in the calibre of the pylorus which prevents the passage of the stomach contents into the intestine within normal limits of time and with normal muscular effort.

Etiology.—All the causes that may act in adults are likewise effective in children, and do produce this result, though more infrequent in early life. Cancer alone must be denied consideration, since its occurrence is so excessively rare. Ulcer has several times been known to cause severe stricture in childhood, acting thus either by means of the resulting cicatrization of the wall of the stomach or by the formation of external adhesions. Adhesions from any other cause may so bind down or angulate the pylorus as greatly to decrease its lumen. Growths external to the stomach, but in the neighborhood of the pylorus, may compress the latter, with resulting stenosis. Displacements of the stomach itself tend to cause stenosis by angulation, and some degree of this is probably not very uncommon in children, though the duodenum suffers more largely. Contractures of the stomach-wall other than those from ulcer may be the cause. Drinking corrosive poisons and injury from foreign bodies swallowed are possibilities of this kind. Polypoid growths projecting into the cavity of the stomach may be forced into the lumen of the pylorus at each peristaltic effort, with more or less complete blocking of the passage. Atresia of the orifice has been very rarely present. But the cause of stenosis which has been most frequently noted in infants is hypertrophy of the pylorus, which in early life has been usually found as a congenital condition, though this is by no means necessary.

The condition of congenital pyloric stenosis, though one seldom met with, and usually difficult of recognition during life, has of recent years excited much interest, since its existence renders the case at once one of the greatest gravity; and in the consideration of stenosis of the pylorus in children attention is more especially engaged by the congenital variety. Although the case published long ago by Williamson as "scirrhus" was probably an unrecognized example of congenital hypertrophy, the first good description was given twenty years ago by Landerer. Thirty-one cases, most of them probably authentic, were collected by Mayer some time later. Since then cases have been reported with considerable frequency; and among writers upon the subject, Hirschsprung, Finkelstein, Grau, and Thompson have furnished much of our knowledge of this grave condition. Very rarely, as in Neale's case, there exists complete congenital atresia, and the pylorus may be represented by a solid cord alone. Usually, in either congenital or acquired hypertrophic stenosis the pylorus is enlarged, owing to fibrous or muscular overgrowth, the surface is smooth and somewhat conical, and its tissue firm. The opening may be almost completely closed, and has been found very small in most congenital cases reported, though, as in one of Hirschsprung's, death may result from other causes, and the stenosis be insufficient of itself to prevent the child from reaching adult life. It is not improbable that the condition has existed in some cases of dilatation which are discovered in later life, or that a mild grade of stenosis may be completely overcome. The microscopic examination usually shows much thickening of the muscular coats, of which either the circular or the longitudinal may be most affected. Mayer states that the congenital form is distinguished from the acquired by the sharp localization of the hypertrophy to the pylorus itself. Even when there is a coexistent hypertrophy of the walls of the organ in general, this is said to be easily distinguished from the congenitally thickened portion, while in acquired cases the two blend. In congenital stenosis, too, the pylorus is very firmly fixed, owing to the thickness and inelasticity of the lesser omentum and the duodeno-hepatic ligament.

Stenosis soon causes dilatation, which is preceded, if time be given, by hypertrophy of the walls in the endeavor to compensate for the difficulty at the pylorus. These changes, together with the various conditions found in stenosis from causes other than atresia or hypertrophy, are best considered under dilatation, as they can be but rarely suspected before this has developed.

Symptomatology.—Except in those cases that live long enough to result in dilatation, the condition is often only suspected, and grades so mild that they are overcome without serious results can hardly be recognized. In congenital atresia or marked stenosis the predominant symptom is the vomiting. This ordinarily does not appear until several days after birth. When it does begin it occurs after every meal and continues until death, uninfluenced by any medicinal or dietetic treatment. Finkelstein considers this persistent vomiting after each feeding a pathognomonic sign. With the vomiting there is pronounced constipation, and the child wastes and becomes exhausted, dying usually within a few weeks, and always, in high grades of stenosis, within a very few months. The gastric secretions have been examined by Finkelstein, Grau, and Fenwick with varying results in the three cases, and investigation of them gives no aid in diagnosis.

Such indefinite symptoms point only to obstruction in the digestive tract, though the vomiting is somewhat peculiar. Finkelstein and Fenwick have felt the thickened pylorus during life, and the search for such a tumor in the position it would occupy and the endeavor to exclude other sources of obstruction must be our main aids in reaching a diagnosis. The only outcome is death, unless an operation be undertaken.

The symptoms of pyloric stenosis when it has persisted to the production of dilatation become but a part of those of the latter disease, and we will consider in the next section the clinical manifestations of acquired stenosis or of a possible congenital stenosis that does not result in almost immediate death.

Treatment.—The cure of a high grade of pyloric stenosis can be undertaken by the surgeon alone, using the same procedures that are in place in treating dilatation; and the methods of alleviating the symptoms are the same as for gastrectasia.

In congenital stenosis of grades high enough to cause early death but one faint hope of saving life is offered in operation. The usual dietetic and medicinal means for quieting vomiting should be employed, but if the diagnosis be sustained such treatment will be without result.

Rectal alimentation should be instituted, and nutrition and stimulants provided in this manner as long as possible. If a tumor be felt along with the usual symptoms, operation is the only rational advice, but the hope of success in these young infants is small.

DILATATION OF THE STOMACH.

Definition.—Abnormally large size of the stomach which is accompanied by inability to propel its contents into the intestine within the physiological limit of time and of muscular effort.

A stomach which is alone abnormally large as compared with ordinary measurements is by no means necessarily a dilated stomach, since we know well, through the work of Ewald and Riegel in particular, that a stomach may have dimensions much beyond those usual and yet exhibit no abnormality of function. And yet the term *enlarged* stomach as distinguished from a *large* stomach is not, as Ewald would make it, sufficient, for if the motor power of a naturally large stomach fail, the condition is at once a dilatation in no practical sense differing from that existing with an enlarged organ. Undoubtedly the usual distinction between a stomach that is simply unusually large and one that is dilated is that the latter is unable to rid itself of its contents within the time due it. Still there are cases in which there is enlargement without actual stagnation, and in these the added muscular effort which the walls bring into play is sufficient to overcome any obstacles to the onward flow of the contents. Such cases are, to be sure, of little clinical importance at that stage, but, like heart-cases in which hypertrophy compensates a dilatation, they are apt to break down with excessive strain, and the possibility of the presence of such a condition must be reckoned with.

Etiology.—Dilatation is brought about by causes of two main types, which may act singly or in conjunction. These two types are the atonic and the obstructive. In children the former seems more active in contradistinction to adults, in whom some cause of obstruction can be determined in the majority of cases. The relative frequency of these varieties of causes in childhood is, however, not well determined. In either type of cause the action may be acute or temporary, or, by far more common, repeated or persistent.

The causes of atonic type are such as may give rise to atony and relaxation in any tissue. The rarer cases in which acute dilatation takes place are largely of such origin, though some may be due to spasmodic stricture of the pylorus and the violent effort to overcome the acute obstruction present. That acute dilatation takes place at all may be doubted by some, but its occurrence in the adult is indubitably proven by cases published by Erdmann, Fraenkel, Boas, and others, and there is absolutely nothing peculiar to childhood which might prevent such an event. Furthermore, I have myself seen cases in which this took place in children. It may be excited by anything which causes a violent nervous shock. Sudden and powerful emotions cause profound relaxation in any muscular organs, and the stomach suffers at times with its fellows or alone if peculiarly susceptible. Shock from injury acts in a like manner, whether it be, for example, a direct blow in the epigastrium or injury applied to any other part the shock from which is severe. Rosenheim has recently brought this clearly before the profession. Overfilling of the stomach is an occasional cause, particularly sudden swallowing of ice-water or other very cold liquid in large amount. Acute dilatation may result in death, or be very rapidly overcome and escape observation, or it may last a few days and then completely disappear; but there are several cases recorded in which it has persisted for years, with no prospect of cure, either from the violence of the first effect upon the stomach itself or from the impression upon the general nervous system. The condition is in marked degree rare, but milder examples not improbably occur frequently.

Causes of atony which act over long periods and produce their effect gradually are very numerous. All causes of general atony may be included. Bad hygienic conditions, exposure, insufficient nourishment, anæmia, and constitutional diseases may give rise to it. But there are certain special causes which need mention. The most frequent of these in infants is overfeeding. The frequent ingestion of food in excessive amounts must result in abnormal distention of the stomach-walls and undue muscular effort. When long continued this necessarily causes overstrain and relaxation of the muscle, and further continuance of the cause means certain dilatation. A mild degree from such cause is often found in overfed infants, or indeed in any child whose food or drink is not kept within reasonable limits, and serious cases may have like origin.

Continued dyspepsia or chronic gastritis may result in dilatation, as muscular strain is almost constantly present from the irritation of imperfectly digested food, and is frequently increased by exacerbations of the disease. With many a case of dilatation will be elicited a history of long-continued dyspeptic symptoms preceding those pointing to dilatation.

Rickets is a disease of special importance in the etiology, from the frequency of occurrence of dilatation in its subjects. Two important factors exist here,—the general flabbiness so usual with the disease, and the almost constant disturbances of digestion. In an extremely large number of rickety children mild grades of gastrectasia may be found, and occasionally much more severe cases seem traceable to this affection.

Many other causes may have to be sought for atony, and some of them may be found in the stomach itself. Ulcer elsewhere than at the pylorus may be met with, and this may act by its interference with peristalsis by interrupting the progress of the wave and preventing normally concerted action of the various portions of the muscle. Similarly, the traction of inflammatory adhesions following ulcer or adhesions from any other cause may give rise to overstrain, and hour-glass or other contractions from corrosive poisons have a like effect.

In speaking of all these conditions I have used almost synonymously the terms overstrain, atony, and dilatation from atony, and these are indeed nearly synonymous. When a hollow viscus is overtaxed it dilates, and the stomach is no exception. In such condition the performance of its functions with normal relations is too severe a task, and the cause usually persists, so that the necessary result of overstrain is dilatation beginning almost at once, and from being very slight it progresses directly in proportion to the severity of the strain and the time of continuance of its action. Any "atony of the stomach" in which there is enlargement of the capacity of the organ with retention of contents is a dilatation.

Most of the obstructive causes have been mentioned in speaking of pyloric stenosis. Of the acute obstructive causes only one seems likely to occur,—*i.e*, spasm of the pylorus from hyperacidity, from contents otherwise exceedingly irritating, or from the irritation of painful ulcers. This cause is not extremely uncommon where these conditions present themselves frequently in the same individual.

Children have a special cause of obstruction in congenital stenosis, and this condition has been found with sufficient frequency to warrant its habitual consideration, especially in very young subjects. As has been stated, most of these cases which are known to be such die early, and often before any marked obstructive dilatation has taken place. But there is every reason to think that the stenosis may be frequently of milder grade, as it has been in some cases examined. The overgrowth, though usually congenital, may occur after birth with like results. Simple ulcer may give rise to obstruction from the mildest grade to the most severe, acting by means of contractions in cicatrization, or by formation of constricting or angulating adhesions. Tubercular ulceration may give like result, or, as in one case reported, the swelling of the mucous membrane about tubercular ulcerations partly surrounding the pylorus may effect some considerable obstruction. Cicatrices from corrosive poisons may also be so situated as to cause obstruction. Adhesions from other causes of inflammation may be expected with greater frequency than from ulcer. Pediculated tumors within the stomach may act as ball-valves, and any growth without may compress the pylorus. Displacements of the organ sometimes cause angulation at the pylorus, though this deformity is, as mentioned, more frequently situated in the duodenum. Its exact location matters little in its effects, however, as the result is obstruction to the outflow from the stomach, and any other variety of stenosis of the duodenum has the same influence on the stomach, whether it be due to congenital narrowing, to the rare tumors of this part of the gut, to ulcer, or to pressure from without by growths. The etiological importance of a movable right kidney in adults has been much talked of, but, whatever its value in those who have passed puberty, it is certainly an unusual factor in children, owing to the rarity of its occurrence in early life.

Pathology.—The dilatation is rarely saccular or irregular, and in such cases most frequently is caused by pressure of foreign bodies or by traction from adhesions. The usual form is cylindrical, the whole extent of the stomach being enlarged, though this commonly affects the cardiac region more than other portions. The size varies greatly. Enlargement to triple the normal capacity has been found in a new-born child dead of digestive troubles. Holt mentions a dilatation to the capacity of seventeen ounces in an infant two weeks old, and has been able during life to determine a capacity of twelve ounces at the age of four months. Any life measurements are, however, subject to error. In a ten-months baby a stomach holding twenty-five ounces has been found, and in later childhood all degrees have been met with up to such enormous dilatation that upon opening the abdomen the stomach covered nearly all the other organs in the cavity, as in Chvostek's case. But such instances as those mentioned are unusual, and a lesser degree of enlargement is more commonly found.

The lesions found in causal relation to the gastrectasia need little description after their mention in the etiology. In obstructive cases the pylorus may be found compressed or contracted in varying degree, the orifice appearing of slit-like form or of its usual shape with the size diminished. The radiating scar of a healed ulcer may present itself as the explanation of this condition, or there may be no other lesion than adhesions, of which the actual cause may or may not be evident. Compression from tumors and the like would explain itself on sight.

The atonic forms may be associated with adhesions from local peritonitis, the traction causing dilatation directly and by the extra strain this throws upon the stomach-muscle. General chronic peritonitis, of whatever nature, has like results. A chronic ulcer somewhere in the general body of the organ may be found with indurated edges and surroundings increasing the labor in peristalsis. But commonly there is no localized lesion explanatory of atonic dilatation, and the condition is then found to be a general relaxation of the walls, which are in such cases usually stretched and thin. In either form of the trouble the walls may be of normal thickness or vary on either side of this, but hypertrophy of the coats is frequent only with the obstructive variety and is most marked in the pyloric region. The mucous membrane often shows evidence of chronic gastritis, either the cause of the dilatation or secondary to the stagnation which the latter induces.

Microscopic examination will show increased or diminished thickness of the muscular layer, as the case may be, usually with overgrowth of the interstitial tissue and some infiltration with round cells. When the walls are thin the muscle-cells appear fewer in number, owing to their separation by spaces filled with areolar tissue. Fatty degeneration of muscle-cells is often seen. The mucous membrane commonly exhibits some stage of chronic gastritis, varying in portions taken from different regions.

Symptomatology.—Mild cases of dilatation often show no subjective symptoms that are distinctive of the condition. There are the general symptoms common to most gastric troubles. Languor and depression of spirits or irritability is often present, and the infant or child has not its usual interest in diversions or play. Corresponding with the extent of the interference with nutrition, the muscles are flabby and waste, the fat disappears, and anæmia develops. When the dilatation is but slight, sufficient nourishment is furnished the body to prevent these general symptoms from attaining any great gravity, but when the food passes into the intestines in but small amounts, ill digested and fermenting or putrefying, and very little is absorbed by the stomach itself, the general bodily nutrition suffers to such a degree as to suggest at once the existence of some grave constitutional disease. The child emaciates greatly, its look grows haggard and wan, and the skin is pale or sallow and forms wrinkles or folds. The circulation is sluggish, and the extremities are often cool and dusky. In these advanced cases a frequent and peculiar symptom is the dryness of the skin, often accompanied by a brawny desquamation of the superficial epithelium, which is to be referred to the insufficient supply of fluid to the tissues. Von Mering has taught us that the healthy stomach absorbs little water. Hence little absorption can be expected from a dilated organ, and, since the ability to propel sufficient fluids into the intestine is absent, dryness of all the tissues results. To this insufficiency of fluid and the poor circulation are to be referred the numbness of extremities, formication, and muscular cramps often complained of in severe cases.

Older children, like adults, are very apt to complain of headache, which is often very persistent and distressing, and vertigo is common, coming on in distinct attacks or becoming so constant that uncertainty accompanies all movements. This is by no means limited to the graver cases, but may be very distressing with slight dilatation. Thirst is a very troublesome symptom and one difficult to relieve, since water that is taken by the mouth is but poorly absorbed at best, and large amounts of fluid only increase the labors of the already overtaxed stomach. Thirst increases directly with the amount of stagnation, and the dryness of the tongue is well given by Boas as some indication of the grade of the dilatation.

Appetite runs directly contrary to thirst. When there is only moderate retention of food the appetite remains fairly good, and may be even excessive. As more pronounced retention comes on, the desire for food decreases, until, with actual stagnation, it is never enjoyed, and often is taken only after coaxing. Epigastric discomfort and fulness after eating are always complained of when the child is old enough to give voice to its sensations, and the appetite is in inverse ratio to this distention. If the amount remaining behind from the last meal is large, so is the feeling of fulness and satisfaction of the appetite sooner attained; and when the stomach is already overloaded there is no appetite. Gas is formed freely, and eructations are frequent, even in mild cases. Rapid distention with gas may have grave results in young infants, and Holt records a death from such cause. The gaseous distention may give rise to pain, which is very commonly present in some degree. The distress after eating may be only slight and soon over as the stomach partially empties itself, or it may be nearly or quite constant and of itself amount to actual dragging pain; and with old or recent ulcers or adhesions the pain after food may grow severe. Burning pain in the epigastrium is extremely common, and often the most actively distressing of all the symptoms. It is largely dependent upon the amount of hydrochloric acid or organic acids present, but may be due to the irritation of other retained products of fermentation or decomposition. In mild cases it is usually worst some hours after taking food, and often troublesome in the night, though its degree and constancy are in almost direct proportion to the gravity of the dilatation. Advanced cases frequently have almost constant dull, burning pain, commonly worst in the early part or the middle of the night, so that sleep is often disturbed or scarcely procurable, and the only relief is usually in vomiting or lavage. Acute, sharp pain is not frequent. Vomiting is in pronounced cases a highly characteristic symptom, both in its manner of occurrence and in the appearance and constituents of the vomit. Mild cases may lack the symptom entirely, or it may occur but rarely, and then have no special characteristics beyond evidence of a moderate amount of retention from previous meals and ordinarily a high acidity, in the production of which both hydrochloric acid and the organic acids are concerned. In such cases the starches are badly digested, unchanged particles appear, and on standing a slight frothy layer usually collects on the top of the stomach contents.

In advanced cases vomiting is an almost constant symptom, and occurs in a sequence that is very characteristic. It has usually no relation to individual meals, but is commensurate in frequency of occurrence with the amount which remains from numerous previous meals, and becomes a regular symptom only when the stomach finds itself unable to rid itself completely of its contents in any other way. Then it occasionally exhibits an almost cyclical regularity of occurrence, though exact regularity is uncommon, since it all depends upon the accumulation of contents. Some time elapses between the attacks of vomiting, usually two or three days, though at times they occur daily or are much less frequent. The epigastric fulness, discomfort, and burning have been meanwhile increasing and the appetite disappearing, until at length the overburdened stomach rebels, and vomiting comes on, with repeated straining and retching. Aiding the overstretched stomach-walls with the abdominal muscles and pressure beneath, the patient brings forth often an immense mass of liquid and solid contents, in which there are frequently particles of food taken many days before. Butyric acid is recognized at once from the odor, and with this acetic and other organic acids, and the various products of the stagnant food give forth an almost unbearably foul and sickening smell. After the exhaustion from the vomiting the child often brightens a little, and has some temporary desire for food until the same course of events is once more begun.

Constipation is a regular symptom, sometimes alternating with more or less violent attacks of diarrhœa with very offensive stools, set up by the passage of foul stomach contents into the intestine. Small, infrequent, and hard stools are the usual accompaniments, the frequency and character of the stools depending largely upon the amount of fluids which the stomach is able to propel into the intestines. In advanced cases constipation, with the intestinal fermentation and distention which accompany it, is one of the most troublesome and intractable symptoms. Urination is infrequent, the amounts passed small, and the urine often concentrated, a heavy deposit of phosphates frequently forming on standing.

Any of the complications that have been described as occasionally arising in dyspepsia or gastritis may occur in dilatation, and the child is very prone to intercurrent disease, such as catarrhal pneumonia, nephritis, and the like. Tetany is of special interest in this connection, since a dilated stomach of some degree is not infrequently present when this peculiar train of symptoms appears, and some authors make gastrectasia a highly important factor in its etiology. The exact mode of origin of tetany is not yet clear, and its discussion here is not pertinent, but it certainly sometimes arises in connection with a dilated stomach, and then the most probable explanation is either auto-intoxication or possibly reflex action from irritation. Its occurrence with marked degrees of dilatation is of ill omen.

The symptoms of acute dilatation may be briefly noted. There is sudden distention in the region of the stomach, attended by pain, which is often very severe, and usually with violent vomiting, which ceases if the stomach become paralyzed.

The distention later is often so great that the pressure much disturbs the heart-action, and irregularity of the pulse, or tachycardia, is common. Dyspnœa from the same cause is a usual symptom. With these there may be severe collapse with its usual signs, and the case may end in death, or the symptoms decrease and partial or complete recovery ensue.

Diagnosis.—The physical signs of gastrectasia are so intimately related to the diagnosis that the two are best considered together. Inspection is of great importance in cases of marked severity. The abdomen appears more or less full in the upper and left-hand portion. The greater curvature of the stomach is often marked by the depression beneath this fulness, and is then seen as a line running curvedly downward, reaching near the umbilicus, or perhaps far below it. Often comparative flatness of the abdomen alternates with the appearance of a rounded and elastic tumor swelling out the belly from the ribs to the umbilicus or below, and this sudden distention, followed by collapse, is often complained of by older patients. In many cases the collection of the contents in the most dependent parts makes the epigastrium seem even abnormally flat, while about the umbilicus or below there is a distended area. When dilatation is extreme the whole abdomen may be much distended, but this is more pronounced in the upper left-hand portion, and points thus to enlargement of the stomach. In such cases the prominence of the belly, with the skin drawn tightly over it and the superficial veins enlarged and prominent, is in strong contrast to the emaciated extremities and chest, over which the dry and rough skin hangs loosely or in folds.

Peristaltic waves are frequently seen as rounded prominences moving from the edge of the ribs on the left downward and to the right towards the pylorus. Reverse peristalsis may also be seen, and is of considerable importance when observed, as it is highly suggestive of organic obstruction at the pylorus, though I have seen it caused by spasm at that orifice.

Palpation often aids greatly, as the surface and lower outlines of the stomach when distended with gas or other contents may be felt with much distinctness, as may also the peristaltic waves; and palpato-percussion, or quick pressure, elicits a splashing sound which with the exercise of proper precautions in eliminating error is of considerable importance in the determination of both the existence of dilatation and its degree, the latter by determining the limits of the succussion-splash. This sign has been too freely relied upon by many, however, especially by numerous French writers, and before according the presence of the splashing sound any importance one must assure himself that it is not due to fluid contents of the overlying colon. Its absence, too, when gastrectasia is suspected may be due to tension of the abdominal walls or to the temporary lack of fluid in the stomach.

Percussion gives most important physical signs, and in children these sometimes furnish our strongest diagnostic support. Percussion must be practised very lightly, as a rule, as the change in note when one passes from stomach to intestine or *vice versa* is often very slight, and sometimes no recognizable change occurs. Often, however, one obtains more knowledge of the outlines by percussing several times with varying force of stroke. In all cases the limits of the organ in all directions should be determined with as much accuracy as possible. The knowledge of the position of the lower curvature is not sufficient, as malpositions may cause this to vary, and the more vertical position normal to the young child makes the pyloric region relatively lower. The note upon percussion varies according as the organ is filled with fluid and solid contents or with gas, and the amount of distention with the latter causes change in note. When gas is present in not too excessive amount, as is usually the case, the note will be lower than that of the neighboring intestine, and of dull-tympanitic quality. If other contents are present in large amount, or if there is extreme distention with gas, the note will be flat. If uncertain results are obtained in this way, auscultatory percussion is often a very useful and valuable aid. Auscultation alone is of little importance, and the bubbling and sizzling sounds often heard, or the succussion-splash on shaking the body while auscultating, only tell one of the fermentation going on or of the presence of gas and liquids in abnormal amounts. Occasionally spurting or gurgling sounds may be heard over the pyloric region, suggesting by their character greater or lesser calibre of its orifice. But when auscultation is combined with percussion we often obtain the limits much more satisfactorily, owing to the greater readiness with which changes in the note are appreciated.

A valuable method in children is that of Penzoldt and Dehio. Percussing in the upright position of the child before and after swallowing several portions of water, the lower curvature is often brought out distinctly from the intestines by the dulness that results, and the distensibility of its walls is to some extent indicated by the depth to which the greater curvature descends after the several amounts taken.

But the most satisfactory results in determining the size and conformation of the stomach are obtained by inflation. This is most safely and reliably done by inflation through a tube when the tube can be used. When the tube cannot be introduced without severe excitement and opposition, gas may be generated in the stomach by swallowing separately proper amounts of sodium bicarbonate and tartaric acid dissolved in water; but this gives much less certain results and causes more distress than inflation, and is not free from danger, especially with very young patients. The introduction of air through the tube can be done with safety if gentleness and care be used and the air pumped in slowly and not to extreme distention. One should always stop at once if any signs of distress from the distention appear. By this method the stomach may be seen distending, and any distinct changes in size or form are readily recognized by palpation or percussion. Inflation is not so entirely satisfactory in infants as in older patients, owing to the readier escape of air along the tube, but it gives much more reliable results than any other method.

The study of the functions will then decide finally whether the large stomach is a dilated stomach. This is best done by the administration of a test-meal and its subsequent extraction, when this can be accomplished, since only in this way can we gain any clear idea of the actual condition of the functions. In infants and small children this is done by administering a proper amount of milk after the stomach is emptied by a short fast or, when necessary, by a previous washing. Any remnants of the meal after three and a half or at most four hours show that there is distinct mechanical insufficiency. In older children the Leube test-dinner, in amounts appropriate to the age, should be used, and the stay of these constituents in the stomach may not be prolonged beyond six hours without indicating motor weakness. In severe cases a test-meal left in over-night will be found wholly or in large part still in the stomach.

The acidity of the contents is usually high in the early stages of atonic dilatation and often for a long period in cases of obstruction, owing to the severe irritation present. In young children and infants the gastric secretions fail early more commonly than in adults, and free hydrochloric acid is often found absent and the combined acid in but small amount even in early stages. With advancing fermentation the acidity is often excessive from the presence of organic acids and their salts. Lactic acid is often present when the hydrochloric acid is low, and butyric and acetic acids are frequent. When the stomach contents after a test-meal cannot be obtained, vomiting will afford us opportunity in advanced cases of learning what is going on in the stomach, and the usual large amounts of offensive material ordinarily brought forth by these cases can leave no doubt of the mechanical insufficiency. Evidences of stagnation and fermentation will be further found in the demonstration of large quantities of lactic, acetic, and butyric acids and the discovery upon microscopic examination of many particles of undigested food and of sarcinæ and yeast fungi in large numbers. Inflammable and other noxious gases and ptomaines have been found in the vomit.

The diagnosis of dilatation is not sufficient, however, but an effort must be made to learn the cause. This can by no means be always done, but the following points are helpful, and sometimes a correct conclusion can be reached. A previous history pointing to ulcer, such as hemorrhage and localized pain, or the persistence of such localized pain or tenderness, a history of ingestion of corrosive poisons or of localized peritonitis or causes leading thereto, such as trauma or difficult passage of gall-stones, discovery of peristaltic or antiperistaltic waves, and a tendency to onward progress little influenced by treatment, all speak for obstructive origin. The absence of these, tendency to improvement, frequent presence of bile in the stomach contents, an atonic constitution from general disease or other cause, and a history of taking excessive amounts of food or drink, are all in favor of atonic dilatation. Vomiting, too, is usually somewhat characteristic in advanced cases, as in obstruction the motor power is often good owing to hypertrophy of the muscle, though it is unable to overcome obstruction. Therefore these stomachs soon resent overloading, and vomiting occurs often and before an extreme amount of retention has occurred between the attacks, while the weakly muscle of a much dilated atonic stomach has not the energy to empty itself until stagnation becomes so great that it is finally irritated into vomiting tremendous masses of disgusting contents. The action of the stomach in lavage is, in the same way, somewhat distinctive. In both the water runs in freely and quickly, but in the outflow, if the dilatation be from obstruction and the motor power good, the stomach expresses itself at a lively rate and with forcible flow. An atonic stomach even but slightly dilated returns the water with a lazy flow, and advanced cases of this kind are often difficult to empty at all by siphonage.

In mild cases other disorders of the stomach are excluded by the existence of enlargement and retention. Severe cases have such pronounced manifestations that their recognition is easy if any care be used.

Enlargements of the liver or spleen and abdominal growths may have a superficial semblance to dilatation, but the use of the methods of examination which have been described will be sufficient to eliminate them and to show the abnormalities of the stomach. Ascites sometimes gives an analogous appearance of the abdomen, but in this the effusion sinks to the flanks and lower abdomen and distends those portions, while the region of the stomach is comparatively flat; the stomach is found of normal size, and no stagnation of food takes place. Slight dilatation is not uncommon, however, in diseases causing ascites, and examination will then demonstrate both conditions, with the atony of the stomach usually distinctly subsidiary. Enlargement of the colon, either congenital or acquired, presents the most serious difficulties, but Dehio's method or inflation, the characteristic vomiting, and the demonstration of mechanical insufficiency will show a faulty stomach; contrary to this, a greatly enlarged colon shows a fulness with its lower limit concave downward, the ballooned gut can usually be felt running down into either flank, auscultatory or direct percussion shows an outline corresponding to the colon rather than the stomach, and these signs can be intensified by gentle inflation by the rectum.

Prognosis.—In the commoner chronic cases the outcome depends upon the cause and the degree to which the condition has progressed. If an organic obstruction be diagnosed, even mild cases can be only partially relieved by treatment other than surgical, and the condition is almost certain to progress with a rapidity depending upon the degree of obstruction and the lack of tone of the patient, unless the surgeon remove the cause. When the dilatation is atonic, all but advanced cases may be very greatly relieved by proper and persistent treatment, the probability of actual cure depending largely upon the general constitution and the absence of neurotic tendencies. Even very high grades of dilatation, which would be hopeless in adults without surgical intervention, may be remarkably benefited and sometimes entirely cured after the improvement of the general health and the relief of the stomach by diet and lavage.

The prognosis is in such cases more favorable than in adults, but in organic obstruction and in advanced cases of atonic type, unless treatment relieves them very distinctly and over a considerable period of time, the prognosis must be considered unfavorable unless operation is undertaken, when great relief and even entire cure may be hoped for. The greater prospect of recovery when the diagnosis has been made early, whether treatment be medical or surgical, makes it strongly to be wished that more attention were devoted to the early manifestations of the disease.

With the rare acute cases the prognosis cannot be stated before they have been watched for some time. Death has occurred from this cause, and acute dilatation of extreme degree is necessarily a dangerous condition from the shock and pressure resulting. If the immediate outcome be favorable, severe cases are apt to persist as chronic dilatation, and only their subsequent course will settle the prognosis. Much the same may be said of cases of moderate severity. These and the mild cases usually recover, but this cannot always be promised, and a neurotic constitution, again, makes an unfavorable ultimate outcome more probable.

Treatment.—The main principles upon which the treatment of gastrectasia in children is to be conducted are in no way different from those suitable in adults, though the details must vary. The important indications for the physician in all cases are to support the general system and in particular to heighten the tone of the stomach, to provide nourishment in such form and by such means as are least apt to increase the labors of the already overworked viscus, and, as far as possible, to prevent the accumulation and stagnation of large quantities of food and drink.

The latter indication is best met by lavage. Rectal alimentation is a partial substitute and often a valuable aid, but it often gives rise to much complaint, and can seldom be exclusively used or persisted in for any great length of time, as the bowel soon rebels against this unnatural way of receiving food.

Lavage is always indicated if there is stagnation from day to day,—in other words, if there are still notable remains in the morning of a test-meal taken the day before, or if the vomit shows remains of food in large quantity, or portions of meals taken many hours before. It must then be practised with persistence, washing the stomach if possible until the return flow is almost or quite clear, and using ordinarily plain warm water. Most antiseptics that are often recommended to be added to the water are irritating to the stomach, and their action is but temporary. The main indication for lavage is to wash out the decomposing contents and to empty the stomach of their bulk. Antiseptics may be tried, however, and if their use give relief they may be continued for such purpose. Sodium bicarbonate and salicylate or resorcin, in one per cent. or two per cent. solution, boric acid in three per cent. solution, or lysol, from ten to fifteen drops to a quart of water, are best. Such solutions should always be followed by plain water to wash out any portion remaining from them. One is guided by the degree of stagnation in deciding with what frequency lavage is to be used. In most bad cases once a day or once in two days will be best, though sometimes two washings daily may be necessary, and less severe cases need it but twice a week. Improvement of the appetite, decrease of the stagnation and constipation, and increase of the amount of urine excreted, together with improvement in the bodily nutrition and increase in weight, usually follow, and the amount of continued improvement under lavage is a good indication, as a rule, of the curability of the case. So important is this procedure that in bad cases of dilatation alone, if persuasion fail to reassure the child and the tube cannot be introduced by gentle measures, I think it admissible to use the gag and assistance if necessary, to see if the relief afforded by a few washings will not lead the little patient to submit afterwards without fear and a struggle. In cases of moderate severity or mild degree lavage is to be used but once or twice a week, and not too long continued, with the object of securing a tonic effect rather than the cleansing, which is here less required.

In these cases the stimulating effect of lavage is often increased by using cool water or alternating this with fluids of higher temperature; and when secretion is subnormal, solutions of bitter stomachics like quassia and gentian may stimulate the gastric functions to greater activity.

Quite as important as lavage is the endeavor to prevent as far as possible the accumulation of food in the stomach and its further overstrain by giving only readily digested foods, and those in as concentrated form as possible. Liquids by the mouth should be allowed in but small quantities when they can be largely done away with. In infants liquid diet is, of course, necessary, and in older children the stomach sometimes bears other food badly. But in any case these should be used as food only, and the drink limited. Excessive thirst should be relieved as far as possible by supplying water to the tissues per rectum and by wetting the mouth with small sips of water or iced drinks. Large draughts of liquid are not to be permitted. Food in all cases is to be taken in small portions, and the nutrition sustained by decreasing the intervals between meals, and in bad cases by rectal alimentation. For the latter purpose from one to three enemas may be used daily, containing milk, albumen solutions, or derivatives of albumen, either ready made or prepared by peptonization, or beef preparations and eggs may be used, any of these in quantities suited to the age. Any food is often better borne by the mouth or the rectum if previously peptonized. Nutritive enemas can rarely be wisely continued at one time for more than a fortnight, and should then be stopped temporarily, and if necessary used repeatedly at intervals. Alimentation exclusively by the rectum for any considerable time will be needed only in bad cases.

The food taken by the mouth in very young patients should be milk or special preparations which are to be found in the chapters on infant feeding, given in small amounts and at more frequent intervals as may be necessary to preserve the nutrition. Greater concentration cannot be borne by their stomachs, as a rule. But when the child can take mixed food it will usually do better on dry diet, such as bread toasted dry, eggs, scraped meats, and in milder cases a larger variety may be found in dried or smoked meat and vegetables of which water is not a very large constituent, and best given in the form of purées. Fats can be allowed only in small amount, or not at all, as they are not digested in the stomach, and only decompose.

When there is little retention, the diet should be such as is suitable for chronic dyspepsia or gastritis, using only readily digested forms of food, and as far as possible regulating the relative amounts of starchy and albuminous foods by determining the acidity of the stomach contents and by trials of their digestibility in each case. Examination of vomited matters will often show which of these classes of foods is less easily digested.

In all cases there should be absolute rest after each meal, and in bad cases the child should remain in bed until there is distinct improvement. At the same time, general measures should be used to improve systemic tone. Tepid sponge-bathing, followed by friction of the skin and massage, is very useful for its general effect, and epigastric douches may stimulate the stomach. Cold should be guarded against by woollen underclothing, plenty of fresh air provided, and the child diverted by restful amusements. Massage of the abdomen in general, or that directed towards the stomach itself, may have useful results, while electricity applied to the epigastrium sometimes seems to aid appreciably in improvement. A firm flannel binder both protects from cold and supports the overstretched stomach. Medicinal treatment is of limited value in bad cases. Most often then nux vomica or strychnine may be of use in as large doses as are allowable. Given by the mouth when there is much dilatation, little of the drug is absorbed and good results are rarely apparent, but used hypodermically it sometimes acts very happily as a tonic both to the general system and to the gastric walls. Antiseptics, such as hydrochloric acid (when there is subacidity), resorcin, creosote, menthol, and sodium hyposulphite, may be used to relieve the distress from fermentation. Some lessening of the symptoms often ensues, but brilliant results from such treatment alone cannot be expected.

The violence or persistency of vomiting may require sedatives, when menthol or chloroform may be tried and often result well, but frequently small hypodermic injections of codeine or morphine with atropine will do much better.

Sleep may be occasionally so much disturbed by pain as to require the use of the latter drugs. Insomnia without pain that is not controlled by general measures is usually overcome by small doses from time to time of sulfonal, trional, or the bromides.

Cases of moderate or lesser severity rarely require hypnotics or antemetics, and derive much more benefit from medicinal treatment. Strychnine or, in the very young, nux vomica, given by the mouth, is of great importance as a muscular and nervous tonic and at the same time acts as a stomachic, increasing secretion and the local muscular action. Antifermentatives are here more useful and more effective, and the treatment in general in such cases is largely that of chronic dyspepsia, with especial regard to the improvement of muscular tone.

Constipation in cases of any degree of severity should call for purgatives only after the entire failure of other measures. Abdominal massage and douches may relieve it, and in milder cases the diet may be arranged so as to leave a considerable fecal residue. When such measures are insufficient, the employment of enemata is useful and is often previously indicated to supply water to the tissues. Plain water may thus answer for both purposes, or soapsuds, castor oil, and other laxative injections or suppositories may be needed. Only in the rare cases in which these fail to produce evacuations should mild laxatives be used by the mouth; and it is to be remembered that the bland diet usually taken by even older children makes daily stools unnecessary.

The treatment of acute dilatation would consist in the evacuation of the stomach by lavage if free vomiting had not already occurred, the application of a firm binder, and treatment of the collapse and the special cause that may be found to exist. Excessive vomiting would require sedatives.

Recovery or great improvement in health is more common in children with extreme chronic dilatation than in adults, but surgical aid must always be kept in mind in severe cases. In any bad case, if after reasonably extended medical treatment there be no lasting improvement, or the health fail, operation should be advised, and if in any case a reasonable probability of organic obstruction be established and the progress be unfavorable, or serious interference with the general health persist, the same course will be advisable. The choice between gastro-enterostomy, pyloroplasty, resection or divulsion of the pylorus, and Bircher's operation must be made by the surgeon, and in this he will be largely influenced by the conditions found after opening the abdominal cavity.

HEMORRHAGE FROM THE STOMACH.

Definition.—The escape of blood from the walls of the stomach.

Etiology.—The hemorrhage that occurs in the new-born may be dependent upon special causes other than those usually observed with the symptom in adults. Thus, it may be due to difficult labor and imperfect respiration, with resulting engorgement of the stomach with blood, to hæmophilia, or to small erosions or follicular ulcers of the stomach. In some cases a syphilitic liver or congenital obstruction in the portal venous system is found as a cause. The one cause that has been found with considerable frequency is sepsis, and the origin of this is probably connected with the bacillus discovered by Gaertner, which has many points of resemblance to the bacillus coli communis, but does not seem to be identical with it. In some cases no cause can be satisfactorily determined. All the causes that are active in adults may, however, give rise to gastric hemorrhage in children, cancer being unworthy of consideration owing to its rarity. Trauma or corrosive poisons, ulceration, congestion from pulmonary, cardiac, or hepatic disease or from acute inflammation, may cause it. It often occurs in yellow fever, and frequently in acute yellow atrophy of the liver. Malaria produces it in infrequent instances, and it is sometimes a symptom of hemorrhagic measles or small-pox. Hæmophilia may cause it at any age, as may purpura hæmorrhagica and scurvy, and sepsis of any variety may be accompanied by it. In young girls approaching puberty it occasionally appears as a form of vicarious menstruation.

Symptomatology.—The hemorrhage is itself only a symptom, but when blood is lost in large amounts the secondary symptoms of acute anæmia develop. These are the same as those in the adult, the most important being rapidly increasing pallor, weakness and rapidity of the pulse, sighing or gasping respiration, and anxious expression, with cold extremities and sweating skin, ending, in fatal cases, with syncope and collapse. The hemorrhage itself may be entirely internal and no blood appear, or it may exhibit itself in hæmatemesis, or vomiting of blood, or appear in the stools. In the latter case it is very dark and of tarry look. When vomited the blood is dark, often coffee-colored if it has been long in the stomach. If there is rapid bleeding and the blood is quickly vomited, the color will be bright red.

When but small amounts are lost the whole trouble may escape observation, as no secondary symptoms develop and the quantity of blood may be too small to arrest the attention.

The hemorrhage of the new-born usually begins within about thirty-six hours after birth, but the bleeding may occur much later.

Diagnosis.—The chief point in diagnosis is to exclude sources other than the stomach, as the blood may have been swallowed. With suckling infants, the mother's breasts should be examined for fissures. In all cases the nose and pharynx should be examined. If there be suspicion that the hemorrhage is from the lungs, and not gastric, the discovery of disease in the lungs and the bright, frothy appearance of the blood will confirm such suspicion.

Blood in the stools is with more difficulty recognized as of gastric origin. This may be from the intestines, and produced by the same causes as gastric hemorrhage, or due to hemorrhoids or fissures about the rectum or anus. Examination should be made in these cases to exclude the latter causes at least.

The actual cause of the hemorrhage, when its gastric source is determined, is often hard or impossible to find. When difficult labor has preceded or congenital heart or liver disease is found to exist, these may be accepted as the cause, as may sepsis and the more rarely acting infectious diseases if these are present. The presence of symptoms of ulcer would at once suggest this as a source. Erosions of the mucous membrane, malformations of the portal system, and the like, could be only suspected.

Prognosis.—In general this is bad in young infants, in ulcer, and in grave diseases. Profuse hemorrhage is always of bad prognosis.

Treatment.—Absolute rest and no food by the mouth are essential until the hemorrhage has ceased for at least a day, and in older children rectal alimentation should take the place of that by the mouth for several days at least.

To stop the bleeding Monsel's solution in small and well-diluted doses should be frequently administered if the hemorrhage be profuse. In older children hypodermics of morphine may be used. Tannic acid and other astringents may be given by the mouth. If collapse appear or be imminent, cardiac stimulants should be pushed and transfusion practised, but these are not to be used unless strongly indicated, as they may cause renewed hemorrhage.

SYPHILIS, TUBERCULOSIS, AND MALIGNANT GROWTHS.

Syphilis.—Affections of the stomach that are definitely due to this disease are rare. Gumma is a very uncommon form, but undoubtedly exists, and ulcers may form from a broken-down gumma. Chiari has found gumma of the stomach twice in one hundred and forty-five autopsies upon cases of hereditary syphilis. The observations of many observers, among whom may be prominently mentioned Lancereaux and E. Wagner, make syphilitic gastritis a probable frequent accompaniment of the general disease. Atrophy of the glandular layer and its replacement by fibrous tissue with thickening of the submucous and subserous layers and of the intermuscular interstitial tissue have been found, but their actual syphilitic nature is not demonstrated. Congenital syphilis of the liver may cause hemorrhage of the new-born, and the hemorrhagic diathesis that occasionally causes this accident is sometimes an accompaniment of congenital specific disease.

The diagnosis of syphilitic affections of the stomach can be but rarely probable, and never established ante mortem.

Tuberculosis is extremely rare in the stomach, in marked contrast to its frequent occurrence in the intestines. It occurs as ulcers, usually one or two in number, though they have once or twice been found in large numbers. The size varies from the smallest erosions to a diameter of beyond an inch, though usually they are about midway between these dimensions. The appearance is often that usual to tubercular ulcers, but they always require microscopic study to make their nature clear. They may be from broken-down tubercular follicles or, probably, from infection of previously existing erosions or follicular ulceration.

The symptoms, if symptoms appear, would be those of simple ulcer. Death from hemorrhage or perforation has occurred, though but one instance from each cause seems known. In one case some pyloric obstruction was caused by an ulcerated ring about the pyloric opening, the swollen and infiltrated mucous membrane about the ulceration blocking the passage of contents into the intestine.

Malignant Growths.—These are of such rarity in childhood that the possibility of their occurrence is all that needs mention.

MALFORMATIONS AND MALPOSITIONS OF THE STOMACH.

Absence of the entire stomach has been found only with acephalic monsters, but in numerous instances the viscus has been found of such extremely small size that its diameter was scarcely greater than that of the duodenum. Hour-glass contraction is occasionally found as a congenital condition. Neither of these anomalies could well be diagnosed during life. The importance of congenital stenosis of the pylorus or of atresia of this orifice has been mentioned. In much rarer cases atresia of the cardia has been found.

Acquired malformations are due to corrosive poisons or ulcers and their resulting contractures, to external adhesions, and to the causes of dilatation already mentioned.

Among congenital malpositions may be mentioned transposition of the viscera, in which, by the usual methods of examination, the stomach may be found occupying the right side of the upper abdomen. Umbilical or diaphragmatic hernias may have this organ among the contents of the sac.

Acquired anomalies of position may be various, and are usually due to traction of adhesions or of other dislocated organs within the abdomen or to injurious pressure from without by means of unhygienic clothing or of some bodily deformity or disease, such as curvature of the spine. Any new growth may, of course, push the stomach from its normal situation. Gastroptosis is not of the same importance in children as it is in adults, since the more usual causes, such as tight lacing and pregnancy, are absent. Cases may be met with, however, when they may be recognized by percussion and inflation.

THE DIARRHŒAL DISEASES.

BY JOHN N. UPSHUR, M.D.

AN increase in the alvine discharges, characterized by increased peristalsis and more or less profuse, watery stools, constitutes diarrhœa. But so diverse and complex are its causes and the conditions under which it occurs that an intelligent consideration of the affection requires a practical classification. To make this classification is difficult, because the most serious cases, proving most rapidly fatal, occur when very slight lesions are found post mortem. On the other hand, the presence of grave lesions sometimes manifests itself by such slight symptoms that no apprehension is excited. Thus we are met at the outset by the difficulty of an established basis of classification. Cases alike clinically may depend on different pathologic conditions. Cases dying within three or four days may show no lesion, or there may be only slight desquamation of epithelium. Those which last longer may show inflammatory change, enlarged follicles, mucous membrane swollen, villi prominent. When the attack lasts more than ten days, ulcerative changes may take place, and these may exist to any degree or extent. It will be of most practical value to classify these various forms upon the etiologic basis.

The classical article by Holt, in vol. iii. pp. 61 to 162, is as apt to-day as it was when written eight years ago. In the main its teachings are those which are generally accepted at the present time, so that this supplemental article will deal only with the forms of diarrhœa in which our knowledge has advanced sufficiently to warrant consideration.

Acute Milk Infection.—Acute milk infection occurs in only two or three per cent. of breast-fed infants. It is in the artificially fed that the disease is most common, and during the hot weather, when all the conditions for the growth and multiplication of bacteria are at their best, the milk forming the most suitable culture-medium, especially in cities where milk is served under conditions that cannot be controlled. The milk drawn at a time of the day when the cows are overheated, without proper regard for cleanliness in milking, the milk agitated by being jolted over the streets, and often containing a pre-formed tyrotoxicon, the case becomes one of acute poisoning, the presence of bacteria in the bowels developing poisonous toxines as dangerous as acute chemical poisons. The researches of such bacteriologists as Booker and Jeffries in this country and Escherich and Baginsky in Germany have failed to discover any bacteria peculiar to the affection (Vaughan, in "American Text-Book on Diseases of Children"), but nevertheless it is now well known that the streptococcus pyogenes becomes highly pathogenic in company with certain other organisms in milk. Rotch tells us that in a large series of cases studied by Nivens, Guillebeau, Kruger, Escherich, Stokes, Tonarelli, Bullock, Taval, and particularly in our own country by Booker, severe diarrhœas and acute gastro-enteritis in infancy were shown to be due to the presence of this organism. Stokes has shown the relation between streptococcus infection of milk and an epidemic of diarrhœa among seventy-five girls in a most convincing manner. Rotch hopes to have diarrhœal cases so differentiated that they will be fed and treated by a reduction of the especial constituent of the milk which appears to offer a culture-ground for the development of especial flora in the intestines. Lesage and Theircelin,[1] in a report of the study of the bacteriology of acute gastro-intestinal infections in infants, found that the bacterium coli commune is the most common organism, and that next in order of frequency are the tyrothrix and the bacillus pyocyaneus. Booker in a very scientific communication has shown that he has been able to isolate nineteen different organisms in the diarrhœic stools of young infants. Baginsky, who also recognizes the frequency of the bacterium coli commune and the bacterium lactis aërogenes, has, in addition, seen a white liquefying bacterium rapidly fatal to mice. While the studies of these two observers have shown only the organisms mentioned, still they are convinced that the pyogenic organisms may favor antecedent digestive disturbances, and thus be important factors in the cause of acute intestinal infections.

Czerny and Moses[2] have examined the blood in nursing infants with dyspepsia and with gastro-enteritis. They offer additional evidence that gastro-enteritis is a generalized infection originating in the intestines and due to various micro-organisms; they have found staphylococci, streptococci, colon bacilli, bacillus pyocyaneus, and bacterium lactis aërogenes. In twelve out of fifteen cases of gastro-enteritis one or more of these varieties of organisms were found in the blood; in eleven cases of dyspepsia examination of the blood yielded negative results.

Allyn,[3] Northbridge,[4] and Engelman[5] have recently contributed valuable papers upon the bacteriology and pathology of the so-called summer diarrhœas.[6]

Symptoms.—Sometimes preceded by the existence of a mild dyspepsia; oftener sudden in its development in a previously healthy child. The child begins to vomit and purge, the surface of the body becomes cool, the face grows pallid, and then pinched, death ensuing sometimes in a few hours. The eyes sink in the sockets, and the expression of the face indicates anxiety and alarm. The stools are at first fecal, containing undigested food, then become more watery and copious; at first yellowish or green, they finally become serous and almost colorless, and of a peculiar musty odor. Emaciation is rapid and progressive, more so than in any other disease except Asiatic cholera. The skin is pale and pinched, but the thermometer in the rectum indicates great bodily heat, registering from 102° to 104° F., and before death may rise as high as 108° F. Respiration is shallow and irregular, the pulse feeble, thready, and frequent. The child cries at first, then moans, then becomes restless, delirious, rolling the head from side to side, and may die in coma or in convulsions. Sometimes the vomiting and purging cease suddenly, raising false hopes of improvement, but this is only a precursor of death. Sometimes the child passes rapidly into the algid state, coma supervenes, the temperature is subnormal, and death soon closes the scene.[1] In this form of trouble the child lies with its eyes half shut, the corners of the mouth retracted, fontanelle depressed, respiration irregular, pulse weak, urine scanty or entirely suppressed.

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[1] Rev. Mens. des Mal. de l'Enfance, November, 1894.

[2] Jahrbuch für Kinderheilk., Bd. xxxviii. S. 430; Year-Book of Medicine and Surgery, Gould, 1896. p. 568.

[3] University Medical Magazine, July, 1895.

[4] New York Medical Record, September, 1895.

[5] Journal of the American Medical Association, October, 1895.

[6] Read also the article on "Intestinal Bacteria," page 691, of this volume.

We may have, however, more favorable cases than those described above. The patient, by the vomiting and purging, has eliminated a good deal of the poison; it brightens up, there is amelioration in all the symptoms, and convalescence is established. The usual duration of these grave cases is about forty-eight hours, when the patient either dies or recovery takes place.

Diagnosis.—There is no difficulty in recognizing the affection, unless Asiatic cholera be prevalent, when bacteriological investigation of the stools will be necessary. The suddenness of the onset, the copious stools and vomiting, and the rapid emaciation point to the nature of the trouble. The prostration of sunstroke is more sudden in its onset.

Prognosis.—The prognosis is grave. The more persistent the vomiting and purging, the more marked the nervous symptoms, the graver is the attack. Amelioration of all these symptoms gives rise to hope, but the physician should not be too sanguine, for relapse may occur.

Treatment.—The affection is of so serious a character that the promptest treatment is necessary. Every particle of milk should be withdrawn from the diet. The stomach and bowels should be washed out to remove every particle of undigested food, warm water being used. This plan of treatment needs no defence at the present day; its worth is fully appreciated. If the fæces contain much mucus, borax is to be added to the water, and if astringents are indicated, the best is a one to two per cent. solution of tannic acid. If the toxæmic state develops, large quantities of saturated boric acid solutions are to be used (from two to four litres) once or twice a day. Blech[1] advises the addition of hydrozone, in the proportion of a tablespoonful to the pint of water (sixteen to five hundred cubic centimetres), for a stomach wash; for the intestinal irrigation the proportion is two ounces to the quart of water (sixty-five cubic centimetres to one litre). Ssokolow[2] has recently made some most interesting experiments, showing what the probabilities are of water injected into the colon passing the ileocæcal valve. Vaughan recommends that a solution of tannin in cool water should follow this.

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[1] See article by T. M. Rotch, M.D., in this volume.

The administration of calomel, in doses of from one-quarter to one-half grain every two hours, will be of service, by its antifermentative and antiseptic action. If necessary, stimulants, in the form of whiskey or brandy, may be given in julep iced. Thirst may be alleviated by a one-tenth per cent. of dilute hydrochloric acid in sterilized water. The nervous symptoms are to be controlled by a hypodermic of morphine, not too often repeated, one-hundredth of a grain to a child one year old. In conditions of extreme collapse and prostration, nitrate of strychnine and nitroglycerin may be resorted to, adapting the dose to the age of the child. The temperature should be controlled by an ice-cap to the head and ice-water sponging, but the tar derivatives should not be used. No food should be given, except Valentine's meat juice in ice-water, a few drops to the teaspoonful, or some other similar meat juice. Almost all the drugs having antiseptic properties have been recommended and used. Fomentations of spice to the abdomen tend to allay vomiting and soothe intestinal irritation.

The plan of treatment suggested by Luton, of Reims, and Remey, of Nancy, has been very favorably received. The plan is, briefly, to withhold all food for several hours in the beginning of an attack; the duration of this abstinence is to be determined by the strength of the child and the intensity of the disease. It is necessary to restore blood-pressure, to supply to the blood the liquid that it has lost, to allay thirst, and to cleanse the gastrointestinal tract of retained poisonous matters. This is accomplished by the exhibition of a feebly alkalized and sparkling water. Para suggests Vals or Soultzmatt. These waters are to be given in small doses, at first frequently and persistently, until thirst is no longer complained of. During a few hours the child should have received from one-half to one litre. At first some of it will be rejected, but if we persist in our efforts large quantities will be retained. The subsequent treatment of the case consists in the cautious return to milk-feeding.

SUBACUTE MILK INFECTION.

Synonyms.—Summer diarrhœa, Infectious diarrhœa, Gastro-intestinal catarrh, Entero-colitis.

Etiology.—"Due to the action of poisons generated by the growth and multiplication of bacteria." (Vaughan.) Prevailing in summer, when the temperature is above 60° F., and causing a large mortality among infants in cities.

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[1] New York Medical Journal, March, 1895.

[2] Jahrbuch für Kinderheilkunde, Bd. xxxviii. S. 16; Year-Book, 1896.

Symptoms.—The movements of the bowels increase in frequency and become more watery in consistence. In color they may be brown, yellow, or green, containing some lumps and small masses of undigested casein. Odor disagreeable. Continued fermentation of the intestinal contents sets up inflammation of the mucous lining of the bowel, with consequent glandular and follicular enlargement and resulting secretion of a pathologic mucus. Subsequently ulceration takes place, located in the lower small intestine and the colon. There is now general impairment of nutrition, emaciation occurs, the appetite fails, the tongue is covered with a white or grayish fur. Fever is present at least some time in the day, and its extent is a good indication of the progress of the intestinal lesion. In mild cases the presence of fever may not be appreciable, and, inflammatory changes not having progressed very far, proper regulation of diet, with the exhibition of some simple remedy, may result in recovery. But in the graver cases the progress of the disease is more certain. It drags on for several weeks, the child losing strength and flesh, and the case probably ends fatally. In these obstinate cases there may be more or less vomiting, due to stomachic indigestion and consequent fermentation, or, later, to a failure in nutrition. There may be acute exacerbations from fresh infection. The child becomes restless, cries out with pain, the abdomen is distended with gas preceding the movement of bowels, the limbs are drawn up, and the abdomen is more or less rigid. The relief comes by the passing off of large quantities of gas with the stool, and these symptoms recur with each action. If nature succeeds in removing the offending material, recovery takes place. Anatomical changes come on as the result of continued bacterial fermentation, and the disease termed entero-colitis is developed. The irritant alvine discharges take the skin off the buttocks. The lesions are more marked in the colon than in the ileum, and are due to the inflammatory action of the chemical products. Just above the sigmoid flexure the depth and extent of the tissue-damage are proportionate to the length of time the irritant fermentative matters have been acting upon it. The potency of these ferments may be judged of by an examination of the stools. There may be lumps of mucus in the stools, or mucus stained with blood,—dysenteric diarrhœa. Shreds of tissue may also be found; sometimes considerable pus is present, denoting ulceration. Too great pains cannot be taken in the examination of the stools. Erythema of the buttocks may be a distressing complication, to be treated by soothing applications, emollient and protective. The inguinal glands or those of the neck may become inflamed, or the child may be troubled with numerous boils breaking out on the face and head. In many cases the stomach remains in a good condition, but it is not uncommon to find the patient suffering with an aphthous sore mouth.

There may be lung-involvement, subacute broncho-pneumonia developing and subsequently proving the cause of death.

Diagnosis.—The symptoms are not of so high a grade as in acute milk infection: the fever is less, the prostration is not so great, the stools are less copious. To distinguish it from chronic intestinal indigestion, the season, the character of the food, and the general hygienic surroundings must be considered. From intussusception it is to be distinguished by the suddenness and violence of the attack of the latter, pain, tenesmus, absence of fever, and stercoraceous vomiting.

Prognosis is influenced by the duration of the attack, the extent of anatomical lesion, and the continued action of the poison. Heredity may be of importance if there is any vice of constitution in the parents. The long continuance of hot, depressing weather diminishes the chances of recovery. The environment of the patient, unhygienic surroundings, and, lastly, when conditions remain unfavorable, the liability to relapse, make the prognosis more grave.

Treatment.—First in importance is prevention. The mother should let nothing prevent her nursing the child, and in connection with this should guard carefully her own health, exercise extraordinary care in her diet, and be circumspect as to the time of nursing, never doing so when overheated or out of temper. The child should be dressed coolly, with simply its napkin and a slip on during the heat of the day, with free bathing in tepid salt water, and guarded from the heat of the sun. Cool changes in the evening should be met by putting on a light flannel skirt and sacque. The child should also be allowed an abundance of crushed ice with which to slake its thirst, and in the cool of the morning and evening should be taken on the trolley-cars into the suburbs for the invigorating influence of fresh country air. If the bowels show a tendency to relaxation, this should not be neglected, but all irritant matters should be at once removed by the administration of a dose of castor oil, followed by controlling doses of bismuth subnitrate, if necessary. Great care should be exercised in the change of the napkins of those children who are well, as well as those sick, the nurse thoroughly disinfecting her hands after changing the napkin, and the napkins being placed in water immediately, and carefully washed in scalding water before being hung up in such place that, in drying, the infants would breathe the same atmosphere. When the mother cannot nurse the child, the best food is cow's milk, uncooked, uncontaminated, if it can be obtained; if not, pasteurized or sterilized milk is best. Should milk disagree, as in some cases it does, meat broths of some kind should be substituted, or barley water, or solution of albumin (white of egg), or even all diet should be prohibited for a day or two, if the case prove very obstinate. Escherich has shown "that the bacterial flora of the infant's bowels changes quickly when milk is excluded from the diet."[1] If the child is seen early, a full dose of castor oil should be given, as above indicated. This should be followed by such agents, singly or combined, as bismuth subnitrate, salol, Dover's powder in small doses. If the action is very sour, from a quarter to half a grain of calomel, with two or three grains of precipitated chalk, should be given until there is a decided change in the character of the stools,—less frequent, less sour, more consistent, and looking like boiled spinach. Resort may then be had to suitable astringents—catechu, kino, or logwood—combined with chalk mixture. Fenwick considers resorcin the most efficient antiseptic, giving three grains every four hours to an infant a few hours old, and claiming decided benefit after the fourth dose. Arsenite of copper in one-hundredth-grain doses has been highly recommended. Solis-Cohen recommends equal parts of benzo-naphthol and salicylate of bismuth, with or without Dover's powder. Free lavage of the bowel daily with a weak salt solution may be resorted to with benefit. Stimulants may be given as indicated by the increasing debility of the child. I would especially commend iced mint-julep. Hot spice-fomentations to the abdomen should be used when there is much soreness or tenderness, and rectal enemata of thin warm starch and laudanum will be of great comfort. Finally, when the case proves obstinate, the child should be removed from the lower country to the more bracing and salubrious climate of the mountains.

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[1] American Text-Book of Diseases of Children.

INFANTILE ATROPHY.

By JOHN LOVETT MORSE, M.D.

The greatest confusion has existed, and unfortunately still exists, in the use of the term atrophy and its synonyms, athrepsia and marasmus. While some apply it to all conditions of wasting, and include half the pathology of infancy under this head, others, who consider that wasting is always a secondary process, do not use the term at all. Others limit it to tuberculosis of the mesenteric glands. Still others restrict it to those conditions of wasting which, in the light of our present knowledge, are primary and not secondary to other morbid processes. These cases of non-secondary wasting form a perfectly definite clinical group, and it is to them only that the term infantile atrophy should be applied.

Definition.—Infantile atrophy is a morbid condition of infancy, in which extreme wasting of the soft tissues of the body occurs without demonstrable organic lesions. It is the expression of continuous insufficient nutrition.

Etiology.—It occurs most frequently during the first six months of life, less frequently during the second six, and is rare after the first year. It is the result of the long-continued lack of a sufficient supply of nutriment to the tissues. This insufficiency of supply is probably the result of some impairment of the functions of digestion or of absorption, more probably of the latter. The primary cause of this impairment is at present unknown. When the failure of nutrition is due to congenital deformities, such as hare-lip or cleft palate, which prevent the ingestion of a sufficient amount of food, or when this food is suitable in character but insufficient in quantity, the condition can be regarded only as one of starvation. This is also the condition in the majority of the cases of emaciation resulting from food unsuitable in character. It is very probable, however, that unsuitable food favors the development of the disease atrophy. Congenital weakness of constitution and lack of warmth, light, pure air, and cleanli-ess predispose to the disease. In a certain number of cases, moreover, the velopment of the disease seems to follow severe intestinal disturbances, her functional or organic.

Pathological Anatomy.—All the tissues show marked anæmia. The is almost entirely wanting in the skin and about the internal organs.

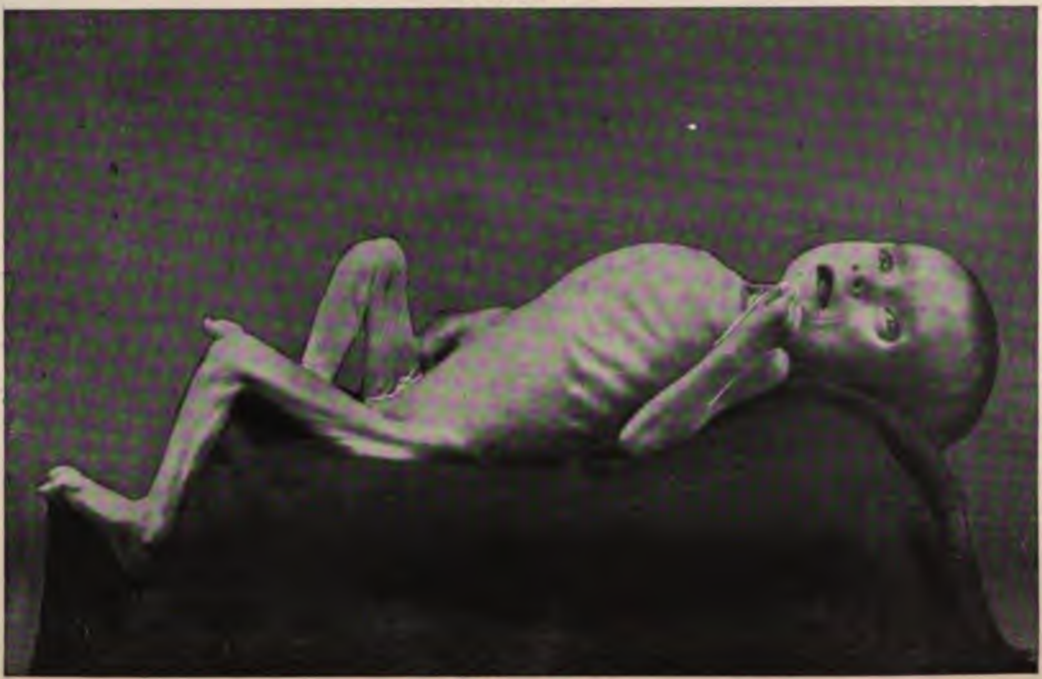
All the muscles show extreme atrophy, and are pale and dry. The brain is normal, but the cerebral sinuses at times contain thrombi as the result of feeble circulation. The heart is, as a rule, normal, but may be fatty. The lungs are normal, except for areas of atelectasis in the lower portions. They may show the lesions of a complicating bronchitis or broncho-pneumonia. The liver and spleen are, as a rule, normal, but the former may be fatty or hyperæmic. The kidneys are usually normal, sometimes fatty, and often contain uric acid infarctions. There is marked atrophy of the mucous and submucous coats of the intestine. Microscopic examination, however, fails to reveal any characteristic lesions. The lesions of complicating catarrhal or follicular inflammations may be present. The superficial lymph-glands are, as a rule, enlarged. The retro-peritoneal and mesenteric glands are not enlarged, although they may appear so from atrophy of the surrounding parts, and are normal on section. The blood is pale and usually fluid. The pathological conditions, therefore, afford little or no information as to the nature of the disease.

Symptoms and Course.—The earliest symptom of infantile atrophy is failure to gain in weight or loss of it. This loss of weight is progressive, and is the most prominent feature of the disease. For some time it may be the only symptom, the function of digestion apparently remaining intact, while that of absorption is impaired. The appetite is, as a rule, diminished, but may be voracious. Thirst is increased. Vomiting is unusual, and the stools are normal or constipated. The total amount in twenty-four hours is said to be increased, but this lacks verification. The child is usually quiet and apathetic, but may be peevish and irritable. The temperature is normal or subnormal. Sooner or later complicating functional disturbances of digestion or inflammatory conditions of the gastro-enteric tract arise, and add vomiting, diarrhœa, colic, and fever to the list of symptoms. These may be acute or chronic, temporary or lasting.

The picture of an advanced case of atrophy is very characteristic. The fontanelle is small and sunken. The face is pinched, the chin pointed, the cheek-bones prominent, and the skin wrinkled, especially about the mouth and forehead. Every change of expression becomes a frightful grimace. The eyes are wide open and staring, or half closed and dull. The cry is feeble and the movements slow and infrequent. The tongue is thickly coated or dry and red, while the mucous membrane of the mouth may show herpetic lesions or those of thrush. The skin, everywhere wrinkled, yellowish, dry, and desquamating, hangs in folds from the bony prominences, as if draped over the skeleton. Pustules on the scalp and body and intertrigo about the genitalia and buttocks are common, and there is often an excessive growth of lanugo on the neck and back. Ecchymoses occur not infrequently. The abdomen is sunken and lax. The extremities are cold, the hands bird-like, and the muscles—especially the adductors and gastrocnemii—look like strings. (Plate I.)

Emaciation continues, the strength progressively diminishes, and the

PLATE I.



Advanced case of atrophy.

child finally becomes too feeble to suck. The action of the heart becomes weak and the circulation poor. The pulse is feeble and rapid, although sometimes slow. The extremities become cold and cyanotic, and pulmonary atelectasis develops. The respiration is weak and superficial, and the temperature subnormal. Death finally occurs after gradual, progressive failure. In some instances sinus thrombosis results from the feeble circulation, and death may be preceded by spastic phenomena or convulsions. Functional and inflammatory disorders of the gastro-enteric tract are frequent and serious complications. Bronchitis and broncho-pneumonia are not unusual complications, while an acute, general, bacterial infection often brings the disease to a sudden close.

The urine is usually diminished in amount, and contains an excess of both urea and uric acid. It not infrequently contains albumin, and sometimes sugar. The blood shows the conditions ordinarily found in secondary anæmia. In some cases there is also a moderate increase in the number of white corpuscles.

Diagnosis.—The diagnosis is to be made from wasting secondary to functional or organic diseases of the stomach or intestine, starvation, congenital syphilis, and general tuberculosis. That from gastro-intestinal diseases associated with wasting must often be a difficult one, as infantile atrophy is frequently complicated by them. In them, however, the wasting is not the one prominent symptom, as it is in infantile atrophy. It is, moreover, not the earliest, but a late symptom, always following other symptoms of gastric or intestinal disturbance. Vomiting and diarrhœa are more common. The stools show evidences of inflammation and indigestion not present in those of infantile atrophy. The abdomen is, as a rule, distended. The temperature is usually elevated. There are fretfulness and sleeplessness instead of apathy. The course is not so progressively downward. Infantile atrophy in the vast majority of cases occurs in the first six months, and rarely after the first year; these others occur at any age. The diagnosis from starvation due to congenital deformities is self-evident. That from starvation due to insufficient but suitable food is also plain. That from starvation due to food of improper character can usually be readily made by a careful consideration of the food in question and by the rapid improvement when a diet suitable to the age is given. The history of disease in the parents or of miscarriages, the presence of eruptions, snuffles, mucous patches about the anus or on the lips, fissures at the corners of the mouth, or of enlargement of the spleen, will usually render the diagnosis of syphilis with emaciation easy. That from general tuberculosis, however, is frequently difficult, and at times almost impossible; for in the tuberculosis of infants the local symptoms are, as a rule, subordinate to the general. A family history of tuberculosis or of continued exposure is of certain but not of great importance. The temperature is likely to be elevated in tuberculosis, but is not always. On the other hand, some cases of infantile atrophy, especially if complicated, have fever. Diarrhœa cannot be considered as pointing especially to tuberculosis of the intestine, as it may as well be due to some complicating condition. Râles are almost always present in the lungs of cases of atrophy as the result of congestion and œdema or of a complicating bronchitis. Solidification of the lung is the most important point in the differential diagnosis, except in the rare instances in which tubercle bacilli can be demonstrated in the sputum or fæces. Even solidification, however, may be due to a complicating broncho-pneumonia.

Prognosis.—The prognosis is always grave. A large proportion of the cases end in death, and when recovery takes place it is always slow, and is usually interrupted by frequent relapses. Complicating functional or inflammatory disturbances of the stomach or intestines render the prognosis still more grave, as do diseases of the respiratory tract. Secondary general bacterial infections always result fatally.

Treatment.—There is no known drug which has any specific action in infantile atrophy, hence treatment must be largely by diet. This must be so constituted as to provide a food which can be not only digested but also absorbed by the individual infant. The natural food—human breast-milk—should always be given a fair trial, if possible. When this is for any reason impossible, substitutes must be used. The best substitute, and the only one which can be modified to the needs of the individual case, is one composed of cow's milk, water, lime water, and milk-sugar in various proportions. Food of this composition can be most accurately prepared in the laboratories established for the purpose. When these are not available it can be prepared very satisfactorily at home. Clinical experience with modified milks of known composition has shown that, as a rule, cases of infantile atrophy do best on milks which contain a low percentage of fat, a high percentage of sugar, and a moderate or somewhat high percentage of proteids. For some unknown reason they seem unable to utilize any but small amounts of fat, and continue to lose if larger quantities are given. Hence the treatment by large doses of cod-liver oil is not only useless but injurious. On the other hand, however, they seem able to digest and absorb considerable amounts of proteids. The proportions which are best suited for the early treatment of the majority of these cases lie within the following limits:

	PER CENT.		PER CENT.
Fat	0.50	to	1.00
Sugar	6 00	to	7.00
Proteids	1.00	to	1.75
Alkalinity	5.00	to	10.00

It is advisable, as a rule, to give small amounts at frequent intervals. While these proportions are the ones best suited to the average case, there are many cases which do not do well on them. In such cases various modifications must be tried until one is found which suits the individual infant. Unfortunately, it is not infrequently impossible to find such a modification, and the child continues to lose and finally dies. In those cases which improve it is usually possible gradually to increase the amount of fat in the food, and finally to work up to the proportions which are suitable for a normal child of the same age. At the same time the number of feedings should be diminished and the amount of each feeding increased. It is often advisable to give the proteids in the form of egg albumin or beef juice. These may be substituted for a portion of the feedings of modified milk, and in some cases may with advantage form the whole diet for a time.

Due attention must be paid to cleanliness and to the maintenance of an unusual supply of fresh air and sunlight. It is especially important to keep up the body temperature, as the vitality of these infants is very low. They must not be handled more than is absolutely necessary, and all excitement must be avoided. No point in the hygiene and nursing of these cases is so small as not to be of importance.

Stimulation, preferably in the form of brandy or whiskey, is often necessary.

Complications must be treated as they arise.

MUCOUS DISEASE.

(CHRONIC GASTRO-INTESTINAL CATARRH OF OLDER CHILDREN.)

By LOUIS STARR, M.D.

Etiology.—The group of symptoms to which the term "mucous disease" was first applied by Eustace Smith occurs most frequently in children who are passing through the earlier stages of the second dentition, a process which, while physiological, frequently seems to be a predisposing cause of the condition. The disease may, however, appear as late as the tenth or twelfth or as early as the third or fourth year of life. It is also occasionally seen in infants, its comparative rarity in them being explained by the fact that, as a class, they have not sufficient reserve power to enable them to survive the initial stages of such a severe chronic impairment of the digestive functions, and, furthermore, they are not subject to the many causes which are active in older children. These causes are to a large extent similar to those which in adults produce chronic gastric catarrh. They are: the winter season, with its consequent greater amount of in-door life; food improper in quantity, quality, preparation, or mastication; and irregular hours for eating, especially if food be taken too frequently. In the wealthier classes the dietetic indiscretions are more commonly in the direction of irregularity in feeding and over-indulgence in pastries and sweets. Among the poorer classes it is not unusual for children to be allowed the food prepared for their elders, including almost constantly ham and cabbage, often tea and coffee, and not rarely some alcoholic drink, as beer or porter. Insufficient clothing and exposure to cold frequently induce catarrh, which may affect the alimentary rather than the respiratory mucous membrane, especially if the patient have inherited or acquired a deficient functional activity of the stomach and intestines. The period of the second dentition, as mentioned above, is an important etiological factor, the attendant irritation in the mouth causing a reflex stimulation of glandular activity in the whole alimentary tract, thus favoring the susceptibility to other irritants. As can easily be inferred, dentition has a greater power in this direction among neurotic children, whose nervous systems are, so to speak, high-strung. Mucous disease frequently occurs as a sequel to whooping-cough, the catarrhal flux in this disease affecting the gastro-intestinal as well as the respiratory mucous membrane. The mucus expelled after a paroxysm of coughing often comes from the stomach, and in cases where the secretion is excessive it may persist and develop into a chronic gastro-intestinal catarrh,—mucous disease. In some cases, however, it is difficult to trace the affection to any prominent cause, and then a careful search must be made for some hygienic fault which, though seemingly trivial, may be the factor upon which the disease depends. Thus, one improper article of food, if used frequently, may cause slight alterations in the normal process of digestion, and these in turn may react as additional irritants, and the vicious circle continue until profound disturbances ensue.

Pathology.—There is little to be said in regard to the pathology of mucous disease, because the affection is not in itself fatal. Extensive organic changes probably do not occur; beyond the usual alterations attending chronic catarrh of all mucous membranes, the condition is mainly an outpouring of mucus by the epithelial cells, governed by a decided neurotic influence.

Symptoms.—The pathological changes being those of a chronic gastro-enteric catarrh, the onset of mucous disease is always slow. There are usually recurring attacks of indigestion, during which the child eats little, complains of pain in the stomach, and has a slightly elevated temperature. These attacks come on at progressively shorter intervals until the little patient is habitually unable to digest a meal. There are loss of flesh and strength and evidences of disturbed circulation, which are manifested in the more severe cases by actual fainting, in the less severe by paleness, varied at times by flushes which may be circumscribed over one or other cheek. There are dark circles around the eyes, the conjunctivæ are muddy, and there may be a transitory strabismus. The skin is dry, rough, and, when brushed gently, scaly, and the hair is dry and faded. The general complexion is sallow or muddy. The tongue, which is tooth-indented, presents a still further striking appearance and one pathognomonic of the disease. In the centre of the dorsum is an oval spot about one-half an inch long, which is bare, deep red, and shiny, the edges of the tongue being covered with a whitish coat, through which the fungiform papillæ are visible. This glossy appearance of the centre may in severe cases extend over the whole tongue. It is due to an excessive secretion from the mucous glands of the mouth. There is still another characteristic appearance of the tongue attending the periodic attacks of vomiting and purging, which usually occur every two or three weeks and during which there is slight fever with the expulsion of great quantities of mucus. At these times the tongue becomes less flabby and has a coating of thick white fur, of irregular outline, being indented with smooth, bright red, glazed patches along its edges. There is usually chronic hypertrophy of the tonsils, and the follicles are plugged by retained secretion, which vitiates the breath to a considerable degree. Although there is frequently a dry, hacking cough, physical examination of the chest may reveal nothing abnormal beyond a chronic bronchitis and acceleration of the heart's action. The appetite is variable at first and may become almost insatiable. There are two factors which cause this increase: one, the irritation of the stomach and intestines by their fermenting contents; the other, the natural craving of the system which is underfed by the imperfectly digested and imperfectly assimilated food. The child's abdomen is often distended, and palpation causes uneasiness if not actual pain. There is usually no one spot of greatest tenderness, but pain is most frequently referred to the left hypochondrium, and is due to flatus distending the splenic flexure of the colon. Sometimes the patient experiences attacks of enteralgia or paroxysms of pain centring around the umbilicus and accompanied by extreme pallor. These attacks are not ordinary colic, as nausea, purging, or passing of flatus are not present. They are probably toxæmic in origin and analogous to the painful paroxysms in chronic lead-poisoning. Movements of the bowels occur irregularly; constipation is the rule, giving way at times to a short diarrhœa, which empties the intestines and is followed by another period of constipation. The evacuations are usually scanty, consisting of small, hard lumps covered with mucus, which are voided after much straining. Prolapse of the rectum not uncommonly occurs. The urine is usually diminished in amount, with an excess of urates; but sometimes there may be a copious flow at the end of the attacks of pain. The patient is drowsy after eating, and suffers from frequent headaches. He is languid, peevish, and cannot enjoy playing, much less studying. The nervous phenomena during sleep consist of restlessness and grinding of the teeth, or, in a severe case, night-terrors, in which the child starts up suddenly with screams, as though in great terror and apparently unconscious of his surroundings. Somnambulism and enuresis may also be present. An obstinate paroxysmal cough is also often observed. In addition to the strabismus mentioned above, stammering is occasionally added to the group of symptoms of nervous origin.

Diagnosis.—The history and many of the symptoms of mucous disease strongly suggest tuberculosis. But the immediate pre-existence of whooping-cough, the time of the second dentition, the appearance of the tongue, the character of the stools, the condition of the skin, the absence of fever, unless accidental or during the periodic attacks of vomiting and purging, the drowsiness and disturbed sleep, the irregular course, and, finally, the result of careful treatment, all tend to exclude the more serious disease.

Prognosis.—In uncomplicated mucous disease the prognosis is good, but the associated impaired nutrition predisposes the system to the development of intercurrent diseases which may prove fatal.

Treatment.—With the adoption and carrying out of certain principles the cure of mucous disease may be confidently expected. Easily fermented food must not be allowed, while the food that is taken must be in small amount, so as not to over-distend the stomach, four meals daily being given at regular intervals in order to furnish sufficient nourishment. Stale or toasted bread may be allowed in small amounts. With this exception, all farinaceous foods must be excluded from the diet-list. Potatoes, peas, beans, parsnips, fruit-cake, pastry, sweetmeats, and butter cannot be taken without harm, nor can tea, coffee, beer, wine, nor any condiment except salt. If there be great debility, small doses of well-diluted whiskey or sherry may be allowed. The diet-list may well be arranged as follows:

Breakfast, 7.30 A.M. One or two tumblerfuls (eight ounces) of milk, guarded by lime water (two ounces to a tumblerful), the yelk of a soft-boiled egg, and a thin slice of stale unbuttered bread.

Luncheon, 11 A.M. A cup (four ounces) of beef, chicken, or mutton broth entirely free from fat, and a thin slice of dry toast.

Dinner, 2.30 P.M. Broiled mutton-chops entirely free from fat (one or two, according to size), a large spoonful of well-boiled spinach, and a slice of stale dry bread.

Supper, 7 P.M. One or two tumblerfuls of milk with lime water and a slice of dry toast.

For drink, pure water or Vichy.

Monotony in the above scheme may be avoided by a judicious selection from the following list: beef, poultry, game, fresh fish, raw oysters, cauliflower-tops, asparagus, lettuce, celery, turnips, onions, and carrots. In addition to the above rules, the hygienic management of the case is of great importance. The aim should be to restore and maintain the activity of the skin by baths, inunctions, and proper clothing. A sponge-bath with the water at 80° F. should be given every morning, the patient being in a warm room, and he should then be briskly rubbed with a coarse towel, after which warm olive oil should be gently rubbed into the body over its entire surface. At bedtime, twice a week, a tub-bath with the water at 100° F. should be given for five minutes, after which the rubbing and inunction are to be repeated. If the skin be very dry and rough, it may be softened by the use of soda in the bath. All the clothing worn next the skin must be of wool, of weight regulated by the season. The pernicious custom of letting the child, for æsthetic reasons, go with bare legs and knees must never be indulged. It is not necessary to enlarge on the importance of this.

The indications for medicinal treatment are, briefly, to check the secretion of mucus, to neutralize the acids arising from fermentation, to restore the mucous membranes to a normal condition so that secretion, digestion, and appetite may return, and to secure regular bowel-movements. All of these indications are met in the following prescription:

℞ Sod. bicarb., gr. v;
Ext. sennæ fld., ♏xv;
Inf. gent. co., q. s. ad f℥i. M.

Sig.—To be taken three times a day, before meals. (For a child seven years of age.)

To increase the flow of saliva there may be added to the above half a grain of potassium iodide, which will also have an alterative effect. After meals fifteen or twenty drops of the tincture of myrrh are very useful for a tonic effect on the intestinal mucous membrane. In place of senna, aloes is a valuable bitter laxative, which may be given in pill form if the child be able to swallow pills. Iron may be combined with the aloes if there be much debility, even though the tongue be coated. Under the above treatment the child will usually improve very rapidly and go on to complete recovery. In some cases, however, the improvement after a time becomes slower or even ceases, in which case it is well to change to an acid treatment, as in the following prescription :

℞ Quin. sulphat., gr. ss;
Acid. hydrochlor. dil., ♏v,
Aq. cinnamomi, q. s. ad f℥i. M.
Sig.—After meals, three times a day.

When periodic attacks of vomiting and purging occur, the child must be put to bed on a liquid diet and a powder of,—

℞ Pancreatin,
Sod. bicarb., āā, gr. v;
Pulv. aromatic., gr. i.
M. et ft. chart. no 1.
Sig.—To be given four times a day.

If the diarrhœa do not subside of itself, or if it tend to become excessive, it may be checked by five grains of bismuth subnitrate every fourth hour. As the tongue becomes normal and the active symptoms disappear, tonics are indicated, the best of which are the tincture of nux vomica, ferrated elixir of cinchona, and bitter wine of iron. The restricted diet must be maintained for at least twelve months after convalescence is complete before the danger of a relapse is passed, while to render the restoration to health well established a change of air to the sea-shore or mountains is of great advantage.

It remains to speak of the dry, paroxysmal cough, which may be so troublesome as to demand special treatment. The usual cough-mixtures are to be avoided, as they tend to derange the stomach, thus aggravating the condition. A small belladonna plaster may be worn over the larynx, and, if internal medication be needed, small doses of belladonna or bromide of potassium may be given every two or three hours, beginning at four P.M.

INTESTINAL BACTERIA OF CHILDREN.

BY JOHN SLADE ELY, M.D.

THE studies of the bacteria of healthy milk-fed infants made during the past ten years have furnished abundant corroboration of the results of the earlier investigations by Escherich and by Booker. The bacillus coli communis and the bacillus lactis aërogenes are still regarded as the predominating obligatory milk-fæces bacteria, and the surprising constancy of their presence, almost to the exclusion of all other varieties, to which Escherich first drew attention, has now come to be one of the most generally accepted features of their occurrence.

Also, as regards the distribution of the bacteria in the alimentary tract, the later researches have been confirmatory of the results of the earlier studies. It will be remembered that bacillus lactis aërogenes was found by Escherich to preponderate greatly over bacillus coli in the upper part of the small intestine, but that on passing downward a gradual increase in the number of the latter germ and a coincident decrease in the number of the former brought about a preponderance of bacillus coli in the lower part of the small intestine and in the colon. In the further study of this question a very exceptional opportunity to study the bacteria of the upper part of the descending colon has been afforded Schlichter[1] by the occurrence of an artificial anus in one of his cases. Cultures from the contents of the colon in this region showed a preponderance still of bacillus lactis aërogenes over the colon bacillus; and it would consequently appear that at times the preponderance of the colon bacillus may obtain only in the very lowest part of the colon.

The study of the biological characters of the obligatory milk-fæces bacteria has disclosed nothing new as regards their morphology, identity, and cultural characteristics which it is important to mention here. But the investigations of Baginsky[2] and Oppenheimer[3] have thrown light upon some of the products of their fermentative activity. It has been shown that when grown in lactose-containing media a very considerable amount of acid is produced by both species of the obligatory milk-fæces bacteria, provided only that the culture-medium also contain nitrogen. With nitrogen present, the fermentation occurred both in aërobic and in anaërobic cultures. Peptone was found to furnish the required nitrogen. Starch was

[illegible] The acids formed in this process differed somew
[illegible] though all appeared to belong to the fatty ac
[illegible] would seem to be the chief product of t
[illegible] associated, as a rule, with lactic acid, and at tin
[illegible] butyric acids (Baginsky), more particularly
[illegible] Oppenheimer, while in general confirming Bagi
[illegible] products of aërobic cultures, finds lactic acid
[illegible] growths. He accordingly expresses the belie
[illegible] lactic acid is first formed, and in the presence c
[illegible] further oxidation to acetic acid. This may accour
[illegible] of lactic acid in the stools, while in artificial aërob
[illegible] bacillus acetic acid is unquestionably in excess. Oppe
[illegible] determined that much more acid was produced in aërob
[illegible] cultures.

[illegible] the part played by the bacteria in the normal digestion o
[illegible] milk-fed infants, there is but little to record. The suggestio
[illegible] relatively small number of bacteria in the stomach of t
[illegible] and from the fact that it has been impossible to detect in t
[illegible] obligatory milk-fæces bacteria any proteolytic action on t
[illegible] is that the milk-fæces bacteria can have but an insignifica
[illegible] assisting the process of gastric digestion, and it is probable al
[illegible] little to do with the normal intestinal digestion. That
[illegible] amount of lactose is oxidized to acid under their influence is ma
[illegible] by the fact that von Jaksch has detected fatty acids (acetic an
[illegible] with much constancy in the urine of sucklings, and that expe
ments of Schotten show that these acids when introduced into the al
mentary canal are excreted in the urine. It is not unlikely, therefore, th
the acids detected by von Jaksch find their entrance into the body fro
the alimentary canal and as the result of the fermentative activity of t
milk-fæces bacteria.

It is apparent, then, that the investigations of the past ten years ha
added little to our knowledge of the intestinal bacteria of the healthy mil
fed infant, except so far as the studies of Baginsky and Oppenheimer gi
us more definite data as to the products of their fermentative activity.

Not a little, however, has been contributed to our understanding of th
rôle of the bacteria in the production of the summer diarrhœas of infant
The studies which have particularly borne upon this subject have bee
directed for the most part to the determination of the number, variety, an
distribution of the bacteria in the intestines of children suffering from dia
rhœa, though studies have also been made of their products, of their effec
on the intestinal mucous membrane, and of their passage through the inte
tinal wall and into distant parts of the body.

That a great increase in the number of the intestinal bacteria occurs
every case of severe intestinal disorder is now generally admitted, as is al
the fact that they are more widely and more equally distributed througho

the alimentary tract. Thus, in a careful study of the bacterial contents of the stomach in twenty-two cases of cholera infantum, Seiffert[4] has shown a very great increase in the number of the bacteria present, and the same has been demonstrated by Booker as regards the intestinal contents. Booker also particularly insists that the peculiarity of the distribution of the bacteria in the intestine of the healthy child is lost in cases of summer diarrhœa. In Seiffert's cases it is also claimed that a direct relationship existed between the number of bacteria present and the severity of the disease.

But the most interesting observations are those relative to the varieties of bacteria present in cases of intestinal disorder in children. Upon this point the conclusion reached by Booker in the article on the same subject in the body of this work—that in the induction of the gastro-intestinal disorders of children "not one specific kind, but many different kinds of bacteria are concerned"—has received abundant corroboration. This conclusion was based upon the results of examinations by Hayem and Lesage, by Baginsky and by Booker himself, all of whom found other species than the obligatory milk-fæces bacteria in cases of serious diarrhœa, but with no definite uniformity. Thus, it will be remembered that Booker isolated no less than forty different species from the stools of various children affected with diarrhœa. The majority of these germs were, nevertheless, shown to possess no definite pathogenic quality as regards the usual experiment animals, though this was found to be otherwise as regards four of the bacterial species. That described by Hayem and Lesage, one of those described by Baginsky, and two of Booker's were found to possess decided pathogenic qualities. Subsequent observers, so far as I am aware, have failed to isolate the bacillus of Hayem and Lesage from similar cases, and we must discard the theory of a specific relationship between the germ and the green diarrhœa of children.

It is of interest, however, to recall these results of Hayem and Lesage in connection with the more recent observations of Demme[5] as showing that very different germs may at times be responsible for similar diarrhœal affections. In Demme's experience seven children, whose only food was the milk of the same cow, suffered from unaccountable and obstinate diarrhœa. The cow was healthy, but examination of the bucket in which her milk was collected showed its crevices to be filled with a red yeast,—saccharomyces ruber,—which thus found its way into the milk in large numbers. Removal of all source of infection by this germ was quickly followed by the recovery of the children. As no other cause of the intestinal disturbance was discovered, and as puppies fed upon milk contaminated by the same yeast developed diarrhœa, Demme unhesitatingly ascribes the disease to changes in the milk induced by the germ. In the cases examined by Hayem and Lesage, Baginsky and Booker, the disturbance was attributed to bacteria, while here it would seem to have been due to the action of a yeast.

More recent investigations of Baginsky[6] lead him to the same conclusion, that no single germ is responsible for the diarrhœal affections of children, and the same opinion has also been strongly expressed by Flügge[7] in his exhaustive article on the sterilization of milk.

The most convincing evidence on this point is, however, advanced by Booker,[8] whose conclusions are based upon the results of careful studies of ninety-two cases of infantile summer diarrhœa of different grades of intensity. As this study represents the most extensive investigation of this subject which has as yet been made, it seems proper to present the results attained somewhat in detail. It will be seen that Booker's cases naturally divide themselves into four groups according to their etiological factor.

In a considerable number of the cases the obligatory milk-fæces bacteria were found to be the chief bacterial ingredient of the stools. These were, for the most part, mild cases, of short duration, and usually without apparent toxic symptoms. The stools were sometimes frequent, were usually acid in reaction, and lacked uniformity of consistence, being often lumpy. They contained no leucocytes. Twenty-four cases of this type were studied by Booker. Bacillus coli and bacillus lactis aërogenes preponderated; other bacteria when present appeared in very small numbers, and were apparently insignificant. Bacillus coli preponderated over bacillus lactis in the stools in all the cases.

In a second set of cases, represented by six only of the ninety-two, while the obligatory milk-fæces bacteria were greatly increased in number, the inconstant forms of the normal intestine preponderated. Thus, in three of the cases "Bacillus a" was the notable feature, and in one case each "Bacillus X," "Bacillus y," and "Bacillus d." These cases were all severe and presented evident toxic symptoms. The stools were frequent in some, infrequent in others, and varied much in consistence, having often a putrid odor. One of these cases, that in which "Bacillus X" preponderated, was fatal.

A third set of cases, comprising thirty-five, are grouped by Booker under the name "Bacillary Gastro-Enteritis," because of the enormous number of bacilli in the stools. Cultures showed the bacilli to be in considerable part the ordinary obligatory milk-fæces bacteria, but proteus vulgaris was also present in very large numbers in all the cases. In many of them there were also a few streptococci and some other inconstant forms. As might be expected from the large number of bacteria present, these were serious cases, usually chronic when not fatal, and characterized by emaciation, toxic symptoms, and stools which were liquid, yellow, or green in color, putrid, and neutral or alkaline in reaction. They seldom contained mucus, leucocytes, or epithelium.

In the fourth and last set of cases, twenty-seven in number, micrococci preponderated in the stools, though bacillus coli was also present in increased number in all, bacillus lactis in fourteen, and proteus vulgaris in four. The micrococci were, for the most part, streptococci. These cases were uniformly severe, and gave evidence of marked toxic disturbance. The stools were often very frequent, varying from three to more than twenty in the twenty-four hours. They were soft or liquid, often greenish, and usually contained mucus and leucocytes in abundance. At times they were very offensive. These cases not infrequently terminated in a general pyæmic condition.

While it is beyond the scope of this article to discuss the pathology of these conditions further than as it can throw light upon the part played by the intestinal bacteria in their production, it may be proper to say that in many of Booker's cases autopsies, performed within an hour after death, showed changes in the intestinal wall in accord with the known effects of the particular species of bacteria preponderating in the stools. In the majority of the fatal cases of the first three groups little or no change was noted in the intestinal mucous membrane, and the change in the internal organs was limited to parenchymatous degeneration. In a few of the third set of cases, and in most of those grouped in the fourth set, extensive inflammatory and ulcerative changes were usually present in the intestine, and in all these cases similar organisms to those in the stools were found in large numbers in the intestinal wall in and about the ulcers, and were obtained by culture from some of the internal organs, notably from pneumonic areas in the lungs.[1]

From these results the conclusion is irresistible that the intestinal disorders of children are to be attributed to no one specific form of bacteria. That in many cases the actual damage is done rather by the products of the bacterial growth than by the germs themselves seems clear, since in many even fatal cases no lesion of the intestinal wall is discoverable, and no penetration of the body tissues by the bacteria can be demonstrated. In the milder forms of these disorders it is not unlikely that the acids generated by the obligatory milk-fæces bacteria in moderate quantity, even under normal circumstances, may, when produced in larger quantity, so irritate the intestinal mucous membrane as to impair its secretory function or to bring about congestion or an actual inflammatory condition. This conception is strongly advocated by Baginsky, and is supported by the decided acidity of the stools in these cases. It has furthermore been shown by Vaughan[2] that the products of the growth of proteus vulgaris and of bacilli "X" and "a" of Booker in broth are distinctly toxic, producing vomiting, purging, and death when injected subcutaneously into young animals.

In the severer cases, and particularly when pyogenic or necrotizing bacteria are present from the start or gain entrance to the alimentary tract during the course of a milder digestive disorder, distinct inflammatory changes in the intestinal mucous membrane seem usually to permit the entrance of the bacteria into the underlying tissues, whence they may be disseminated throughout the body, thus inducing a condition of pyæmia.

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[1] Cf. in this connection an article by Fischl on Septic Infection of the Suckling with Gastro-Intestinal and Pulmonary Symptoms in the Zeitschrift für Heilkunde, xv., 1894, i.

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INTESTINAL PARASITES.[1]

BY LOUIS J. MITCHELL, M.D.

INTESTINAL parasites are a frequent source of distress in the young. Heisig's statistics show us that the proportion of children affected constantly increases from the first to the fifteenth year. Langer estimates that 57.31 per cent. of country and 16.66 per cent. of village children are affected with entozoa. The A. lumbricoides is most frequent in the former, the O. vermicularis in the latter. Miller reports a case of ascaris in a child of only three weeks, and Hoffman has reported a case of T. cucumevina in a child of four months. Cases of polyparasitism may occur, as in a boy of eleven who harbored a tænia, numerous specimens of ascaris, and trichocephalus at the same time. The ascaris is frequently found associated with oxyuris or with trichocephalus.

In addition to the two common tape-worms (T. solium and T. saginata) several other species have been found in children.

Tænia cucumevina is a very common parasite in the intestinal canal of cats and dogs. It is a small worm from ten to forty centimetres long and from two to three millimetres broad. The head is armed with four rows of hooks: the reddish segments are shaped like melon-seeds, whence the specific name. The dog louse, and especially the dog flea, serve as intermediate hosts, specimens of the latter sometimes containing as many as fifty larvæ. The close association of small children with dogs and cats during play accounts for the fact that nearly all the cases of this parasite in human beings have been found in children. Thomson records a case in an anæmic girl of fourteen months, which was traced to a white poodle in the family. The child's mother had noticed similar worms in the animal's passages.

T. flavo-punctata is from thirty to forty centimetres long. The small head has no hooks, and the anterior segments have a large yellow spot (the receptaculum seminis) shining through the skin. So far this form has been observed only in children under two years of age.

T. nana is the smallest tape-worm found in human beings. It is from twelve to twenty millimetres in length and from one-half to seven-tenths of a millimetre in breadth. The joints are much wider than long; the head is armed with a row of from twenty to twenty-eight hooks. Practically all the cases have been found in children. It is extraordinarily abundant in Italy, some subjects being estimated to harbor no less than five thousand specimens. Lutz, of Brazil, has reported several cases in children which he believes to have been introduced by immigrants from Italy. It is interesting to note that this species and the next seem to be slowly spreading in Europe, and with our large immigration and cosmopolitan population they may in time be frequently found here.

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[1] Professor Councilman has covered the ground of this article so thoroughly in his contribution to vol. iii. p. 217, that it is only necessary to note here the advances since made to our knowledge.—L. J. M.

Bothriocephalus latus, though the largest tape-worm attacking man (reaching a length of from six to nine yards), has been noticed in children as young as two years. The head has two suctorial grooves instead of suckers.

Askanazy has recently made some interesting observations on trichinosis. From his experimental researches this author is of the opinion that instead of the young being born in the intestine and piercing the intestinal wall afterwards, the embryos are deposited in or near the lymph-channels and thus carried away. He looks upon the presence of the worms in distant organs as instances of embolism instead of migration, and endorses the statement that purgatives are useless in the latter periods of the invasion.

In addition to the foregoing parasites belonging to the class of vermes, an infusorian, the Megastoma entericum, has also been observed in children. This organism is from ten to sixteen mikrons long by five to ten wide; the transparent body is pyriform in shape, with one side of the larger end cut off obliquely and scooped out. It is found principally in the duodenum and jejunum, where it exists in incredible numbers. Epstein, of Prague, noticed it in twenty-six cases of from less than one year up to six years, nearly half of the cases (eleven) being in children of two and three years. Moritz and Hölzl also found it in eighteen cases; ten of these were in children. These authors claim that tuberculous patients are especially subject to it. Its frequency is estimated at twenty per cent. in children, forty per cent. in adults, and fifty-four per cent. in tuberculous patients. The question of its pathological significance is still *sub judice*. Grassi at first thought it was capable of causing diarrhœa and anæmia, but he subsequently modified this opinion. Epstein is not convinced of its causal relation, although he states that six cases occurred in the same room within a fortnight, which could not be attributed to errors in diet, and in all of them this organism was found. Moritz and Hölzl found it more often in diarrhœal stools, but apparently this was due to the fact that they were more often investigated. Rats, mice, cats, dogs, rabbits, and sheep may all serve as hosts, and man is infected by drinking water or taking food contaminated by the fæces of these animals.

While intestinal obstruction due to worms is infrequent, yet cases are reported sufficiently often to make the condition worthy of remembrance. All the cases seem due to ascarides. Two such have been reported quite recently by Hillyer and by Wyeth, of New York. Laparotomy was successfully performed for the relief of the last case.

Prophylaxis.—Prophylaxis is equally important with the treatment, yet, owing to the inherent difficulties of the subject, it is almost impossible to carry it out thoroughly. The well-known habit of children placing everything in their mouths indiscriminately and their close contact with pet animals provide abundance of opportunities for infection. It is noteworthy that they are much more liable to infection before beginning to walk, when they creep around on the floor or in the dirt. Geophagy is also responsible in some instances: thus, Moosbrugger narrates the case of a boy of eighteen months, with one and two dozen passages a day; upon examination of the stools there were found several thousand eggs of trichocephalus in a cubic centimetre. Upon investigating it was found that the child was in the habit of eating the earth from the garden, and examination of this showed the eggs present in it. Garré has also reported a case of ascariasis due to the same cause.

Something may be done, however, by killing diseased animals, especially the mangy cats and dogs that are frequently taken in out of sympathy. Pet dogs and cats should be banished from the living-rooms. In cases where infection has already occurred, much may be done by strict cleanliness; the finger-nails should be closely trimmed, and the fingers frequently dipped in a solution of aloes or quassia. It may be advisable not to have the patient sleep with other children, but complete isolation, or not to allow the child to play with others, would seem impracticable and uncalled for. Some families are accustomed to giving young children raw meat or smoked sausage to suck; this practice should be strictly forbidden.

Treatment.—Pomegranate bark, male fern, and pumpkin-seed still remain popular as tæniacides. As the decoction of pomegranate is too nauseous to be generally used in young patients, its alkaloid pelletierine is usually substituted. The tannate manufactured by Tanret is preferable, as it is tasteless and very convenient. The writer has never known it to fail. Its efficiency is said to be greatly increased by previously administering a grain or two of tannic acid, which seems to enhance its toxic action.

Cocoanut has been introduced by Parisi, of Athens. It is very success-
ul as a tæniafuge in its native habitat. The pulp and milk of one nut are ken early in the morning, fasting. Of course this is too bulky and indistible to be used in young children.

Hager extols black oxide of copper very highly, and claims that in lition to expelling the worm it improves digestion and appetite. His erience has been corroborated by Schmidt. Hager gives two formulæ:

PILLS.

Cupri oxidati nigri, ʒ 1½;
Calcariæ carbonicæ, gr. 31;
Boli albæ lævigatæ, ʒ 3½;
Glycerinæ, fʒ 2½.
To make 120 pills.

r a child the dose is two pills twice daily, the number given not to exceed fifty pills.

TABLETS.

Cupri oxidati nigri, ʒ 1¼;
Calcariæ carbonicæ,
Magnesiæ carbonicæ, āā gr. 15½;
Tragacanthæ, ʒ 2½;
Glycerinæ, fʒ 1¼;
Sacch. alb., ℥ 1¼;
Aquæ, q. s.
To make 50 tablets.

For children of from eight to twelve years give one tablet three times a day; for younger children one-half tablet four times a day. With either preparation acid food or drink is to be avoided, and the course of treatment ended by a dose of castor oil.

For the ascaris santonin is still the favorite. As deaths are reported from its use from time to time, caution should be used in its administration. Demme holds that the dose for small children should not exceed one-sixth or one-half grain in each dose, or from one to one and a half grains daily. Combemale also states that many cases of vague nervous phenomena in children supposed to be due to worms are really due to santonin. He asserts that its presence in the urine shows the approach of danger. If a dose of the drug is followed by a purgative, such as jalap, to remove the intoxicated ascarides, the medicament will give better results, and the danger will be greatly lessened.

For oxyurids rectal injections of various astringents and bitters are recommended,—olive and cod-liver oils, carbolic acid and creolin, quassia, tannin, aloes, and vinegar. The diversity of the remedies shows how persistent the disease sometimes is. Probably the character of the parasiticide is of little importance, provided it be faithfully used: persistence is the watchword. The bowel must be largely distended and the fluid retained as long as possible; it will usually be advisable to give internal treatment simultaneously to destroy the worms high up in the bowel. The irritation about the anus caused by the sharp tail of the female may be relieved by carbolized vaseline, carbolic solution, or boric acid ointment. In cases of long standing the resulting anæmia is to be treated by the usual measures.

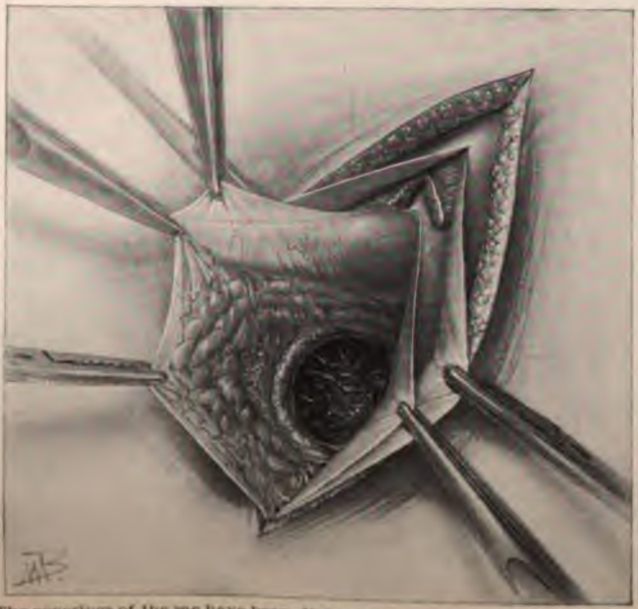
Naphthalin is spoken of both for oxyurids and tænia, in doses of two grains for a child of eighteen months, up to six grains for one of twelve or thirteen years. It is to be given powdered with sugar or in a capsule, preceded by a laxative.

For the Megastoma entericum male fern alone seems useful. Quinine, calomel, and thymol have all proved inefficacious.

In cases exhibiting marked symptoms, where some form of parasiticide has been administered and the worms expelled while the symptoms still continue, the possibility of polyparasitism should be borne in mind, the fæces examined, and appropriate treatment for other forms begun.

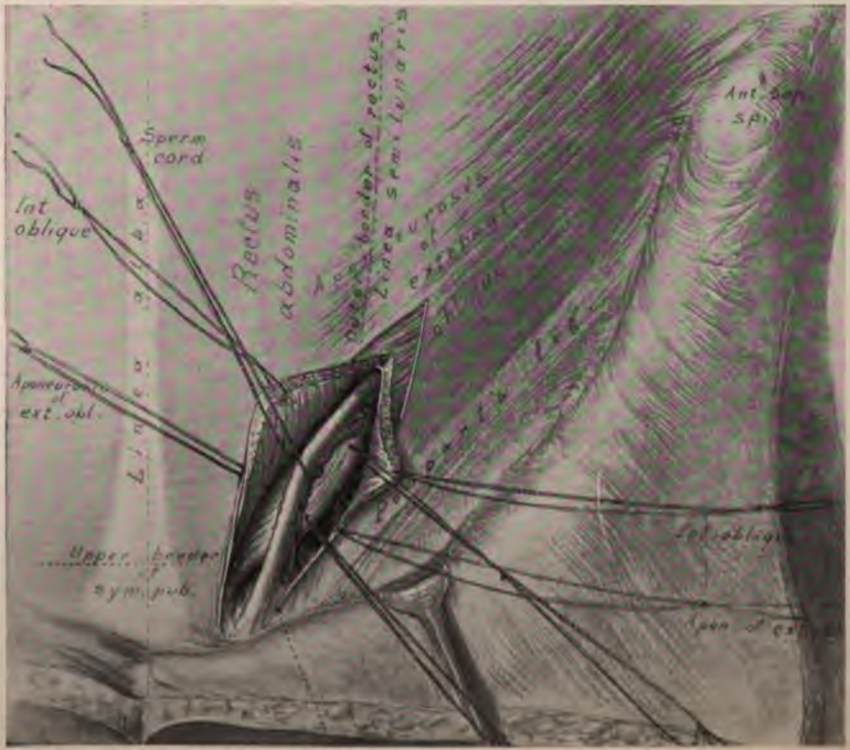
PLATE I.

Fig. 1.



The coverings of the sac have been divided; the sac is held upward and outward, showing the vas deferens and its accompanying vessels. (Halsted.)

Fig. 2.



The sac has been excised, and the peritoneal cavity closed. The internal oblique muscle has been divided and the rectus exposed and the cord transplanted; at this stage the wound is ready for the insertion of the deep sutures. It also shows Dr. J. C. Bloodgood's meth[illegible] tion when the conjoined tendon is obliterated or very attenuated.

HERNIA IN CHILDREN.

BY WILLIAM J. TAYLOR, M.D.

THE general consideration of hernia has been fully expounded in my original article (vol. iii. p. 231 of this Cyclopædia). Since its publication our main advances in knowledge have been almost entirely confined to improved methods of treatment, and it is to these that special attention will be called in the present communication, with the understanding that it is to be read in conjunction with the original article. Very little has been done in the past ten years towards improving the methods of mechanical treatment of hernia in children, and the principles remain the same, and those changes which have occurred have been in slight modifications in the shape and size of the pads used to cover the abdominal rings and of the material from which the trusses are made.

The mechanical treatment of umbilical and inguinal hernia in children by means of the various forms of trusses still retains its position as that form of treatment which is applicable to the largest number of cases, and when judiciously employed will result usually in an ultimate cure.

Marked advances have, however, been made in the methods of operation for the radical cure of inguinal and femoral hernia. It is a subject which has received the careful study of surgeons for a great many years, but it is only of comparatively recent date that these methods have been so perfected as to give successful and permanent results. The essential feature in the radical cure by operation is to obliterate the normal canal and to secure a strong barrier extending along the whole inguinal region, which will prevent any further protrusion of the sac and its contents. To secure this a firm cicatrix is essential, and it is obtained only by perfect asepsis and an accurate coaptation of the tissues by rows of buried sutures involving both aponeurosis and muscles.

The modern operations for the radical cure of hernia as devised by Bassini and Halsted—and by this is meant the radical cure of inguinal and femoral hernia, for umbilical hernia in children almost never calls for operative interference—have completely revolutionized the views formerly held by the majority of surgeons. These methods of permanent relief are now very generally employed in cases where a properly fitting truss has been worn for a year or two without effecting a cure, when the ring is large and shows no disposition to close, and when the hernia is complicated by an undescended testicle. The operation is of comparatively little danger to life when modern methods of wound-treatment are rigidly carried out, and, contrary to the general belief, children run much less risk than adults.

Dr. W. B. Coley[1] gives the mortality in eight hundred and thirty-two cases operated upon by Broca, Felizet, and himself as only four out of the grand total, or less than one-half of one per cent. Sufficient time has now elapsed since these operations were undertaken to prove the permanency of the cures, and to establish the fact that in children, in whom the inguinal canal is very short and the tissues elastic, the results are brilliant. In a personal communication received from Dr. Coley (October 9, 1897) he gives the results of one hundred and seventy-one cases in his individual experience in two hundred and ninety cases of operations for the radical cure of hernia in children under fourteen years of age. Bassini's method was employed in two hundred and fifty-seven; all but eight of these have been traced, with the following results: two were sound upward of five years; fifteen from four to five years; seventy-six from one to two years; thirty-five from six months to one year; forty-one had not yet passed six months, and two cases had relapsed.

The only death in the entire series was from double bronchial pneumonia caused by ether. Death occurred on the sixth day. Four cases died of various diseases after leaving the hospital, and were perfectly sound as regards the rupture at the time of death.

Of the thirty-three cases not operated upon by the Bassini method, thirteen were inguinal, and the technique of the operation was practically the same as Bassini's without the transplantation of the cord. Of these thirteen, eleven were traced; two were well over five years, three over four years, two over two years, and four had relapsed.

Dr. J. C. Bloodgood[2] states that during the past eight years in Dr. Halsted's service at the Johns Hopkins Hospital fifty-eight operations without a death were performed for inguinal hernia in children between the ages of one and fifteen. Fifty-five of these were males and three females. In four of the cases castration was performed on account of undescended and undeveloped testicles.

The condition, with but few exceptions, was known at the time of writing, and in every case the wound was solid and the result perfect. Twenty were between the ages of one and fifteen, nineteen between five and ten, and nineteen between ten and fifteen.

Coley's two hundred and ninety cases and Halsted's fifty-eight cases, making three hundred and forty-eight operations in children under fifteen years of age, with but one death, and that from bronchial pneumonia, is truly a most remarkable showing for any surgical operation.

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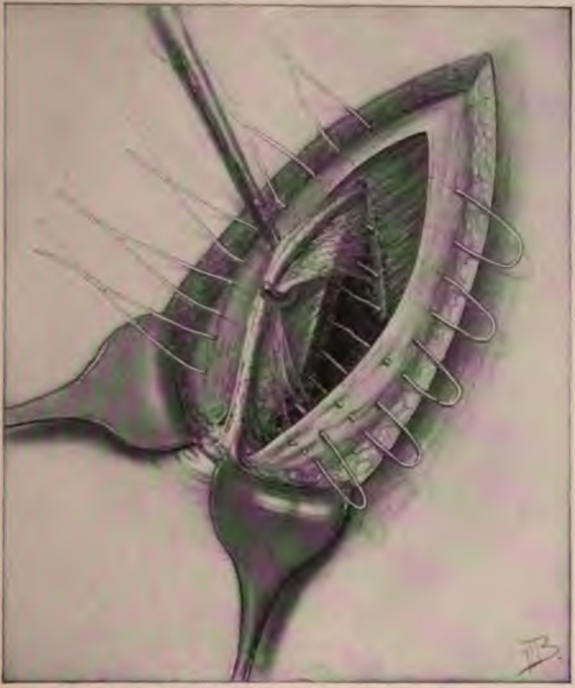
[1] Annals of Surgery, March, 1897.

[2] Personal communication, November 24, 1897.

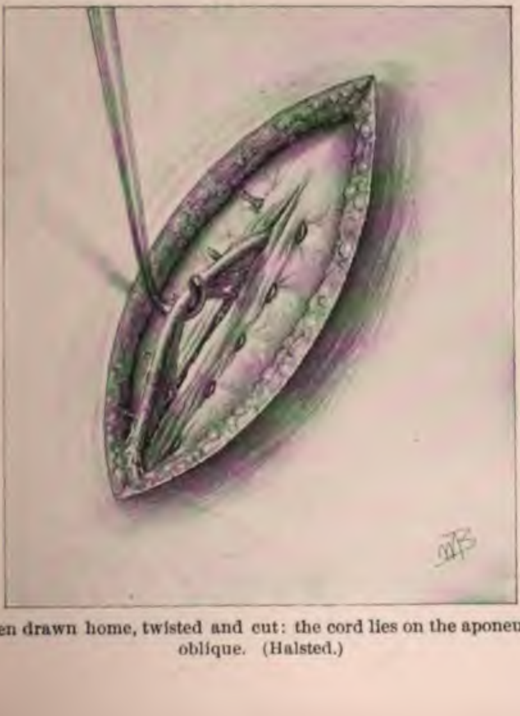
PLATE II.

FIG. 3.



The sac has been excised and the opening into the peritoneal cavity closed. The cord has been transplanted up into the angle of the divided internal oblique muscle, and the mattress sutures of silver wire inserted (four below and one above the cord). This also demonstrates how the divided internal oblique muscle is dislocated downward towards the conjoined tendon, so that it is included in the majority of the lower sutures. (Halsted.)

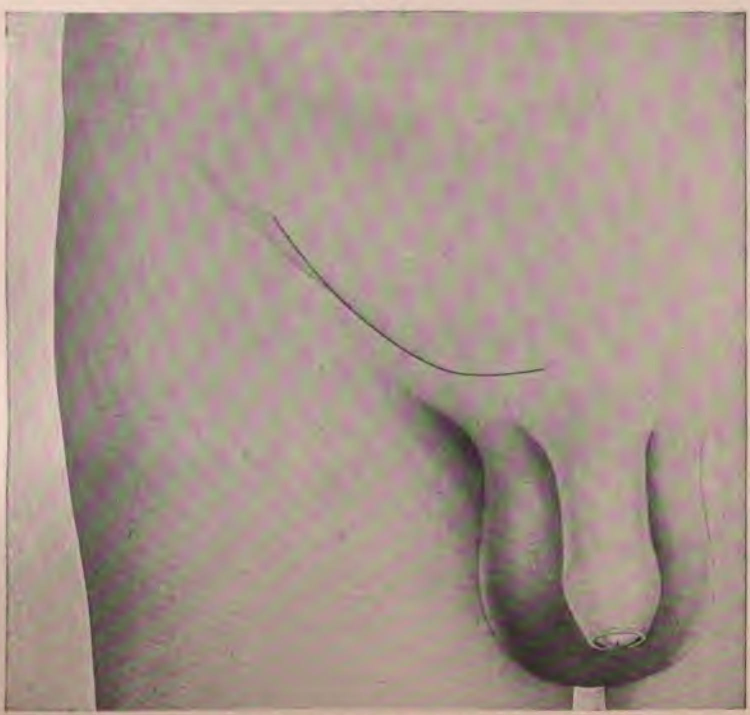
FIG. 4.



The sutures have been drawn home, twisted and cut: the cord lies on the aponeurosis of the external oblique. (Halsted.)

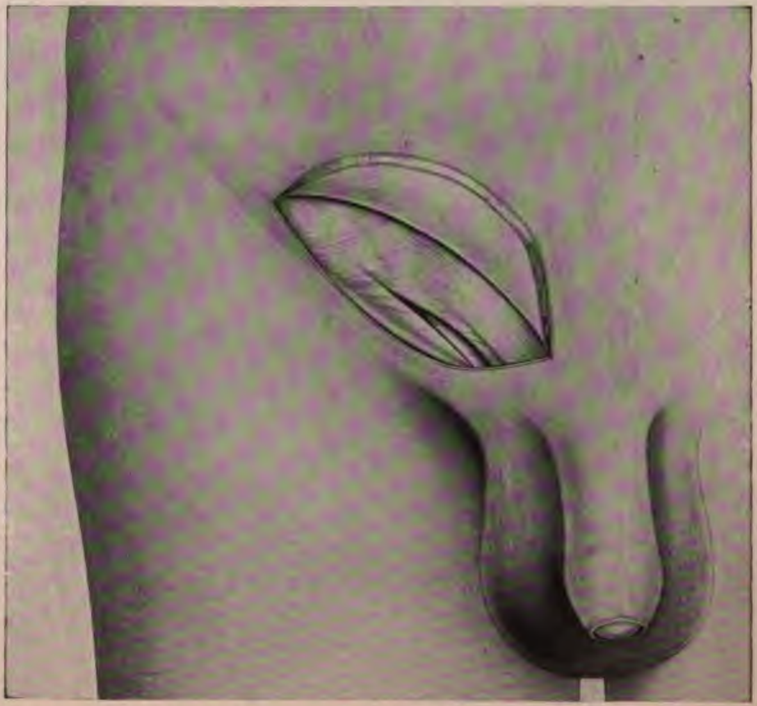
PLATE III.

Fig. 5.



Incision from spine of pubis and parallel with Poupart's ligament to level of internal ring. (Fowler.)

Fig. 6.



Flap turned back, showing aponeurosis of external oblique, external ring, and cord as it passes over the pubic bone. (Fowler.)

Preparation for Operation.—Before an operation is attempted the digestion must be very carefully regulated, the diet for a number of days should be free from all indigestible substances, and for at least twenty-four hours it should be liquid in character, either peptonized milk or meat soups, but never whole milk. For at least two days the child should be kept in bed to quiet the action of the heart and to get him accustomed to the confinement and the restraint of being an invalid. Each night a warm bath must be given, and twenty-four hours before the time appointed for the operation the abdomen, thighs, and pubes prepared, and an antiseptic dressing applied. During the operation, to guard against shock, the extremities should be covered with woollen clothing and dry external heat applied.

Operation for the Radical Cure of Hernia.—The operations of Bassini, of Padua, and of Halsted, of Baltimore, are the two operations for the radical cure of inguinal hernia now generally employed. They are both similar in design, and there is very little choice between them, although a large number of surgeons prefer the method of Bassini. They both consist in an incision parallel to Poupart's ligament over the inguinal canal and the exposure of the aponeurosis of the external oblique and pillars of the ring. The canal is then slit up to a point just beyond the internal ring, and the aponeurosis of the external oblique is dissected from the tissues towards the middle line and outward until the shelving portion of Poupart's ligament is exposed.

The cord is now drawn up and the sac separated and opened. Bassini ligates the sac at a point as high as possible, and any excess is cut away below the ligature. The cord is held up at the upper angle of the wound by a blunt hook, and the rectus, the edges of the internal oblique, the transversalis, and the transversalis fascia are brought together by buried sutures underneath the cord, thus completely closing the old and forming a posterior wall of a new canal. The cord must fit snugly in its new position as it emerges from the abdomen, and a suture passed through the tissues both above and below it to securely enclose it in the new internal ring, but care must be exercised not to constrict the cord too much.

The divided aponeurosis of the external oblique is sutured over the cord, thus making the anterior wall of a new canal, and the wound in the skin is closed without drainage. Halsted ligates and cuts away certain of the veins accompanying the cord, and sutures the aponeurosis of the external oblique beneath the cord, which is thus covered only by the skin and superficial fascia. He excises the sac and sutures it as in an ordinary abdominal wound. Bassini, on the other hand, brings the cord down and closes the fascia over it, thus making a new canal, but does not ligate the veins. The wound is closed by a series of sutures,—catgut for the peritoneum, kangaroo tendon for the fascia and muscles, and silkworm gut for the superficial stitches. Halsted uses buried mattress sutures of silver wire to unite the deeper tissues, and closes the external wound with his subcuticular suture, also of silver wire. The writer has hesitated to leave silver wire buried in the tissues of children, and personally prefers to use kangaroo tendon, which, if properly prepared in alcohol, and not kept in oil, is perfectly aseptic and satisfactory. Kangaroo tendon that has been prepared and kept in carbolized oil is objectionable; it loses its strength, and, as a rule, is not thoroughly aseptic. These operations can be done quickly, there is very little shock accompanying them, and they are most satisfactory in every way. No drainage is necessary; the wound should be covered first with iodoform gauze, then bichloride or sterile gauze.

Dr. George R. Fowler, of Brooklyn,[1] describes a new method for the radical cure of inguinal hernia by intra-peritoneal transplacement of the spermatic cord and typical obliteration of the internal ring and inguinal canal which is so ingenious and theoretically correct that it should be given a very thorough trial. Certainly in adults, and when the ring is very large and the pillars badly developed, it offers many advantages which neither of the other operations possess.

The presence of the spermatic cord in the inguinal canal is the chief cause for recurrence after all operations for the radical cure of hernia, and both Bassini and Halsted, by removing the cord from its canal and by making a new outlet at the upper angle of the wound, endeavor to overcome this and provide a new route for the spermatic cord. Fowler places the patient in Trendelenburg's position, and then makes a somewhat curved incision from the spine of the pubes and parallel with Poupart's ligament to the level of the internal ring. The skin, fat, and fascia to the aponeurosis of the external oblique are included in the incision, and the flap reflected, which thus exposes the whole region involved in inguinal hernia. The anterior wall of the canal is now slit up to the site of the internal ring, the cord and sac isolated together, then separated and thoroughly freed from all the tissues.

The sac is now opened, its contents reduced, and it is then cut away to the level of the muscular layer; the deep epigastric artery and vein are ligated and divided. A complete division with the scissors is then made from without inward, including the transversalis fascia, the subperitoneal connective tissue, and the peritoneum.

The spermatic cord is now pushed into the peritoneal cavity, and the edges of the opening drawn forward so that a broad approximation of their serous surfaces is obtained. Through-and-through sutures are passed from side to side which correct any relaxed condition of the transversalis fascia.

One suture is first passed above and obliterates the site of the internal rings, and the suturing continues until the lower angle of the gap in the posterior wall of the original inguinal canal is almost reached. The suture should be low enough to compel the cord to curve slightly upward and forward as it leaves the peritoneal cavity at its newly formed external ring, but should not be tight enough to constrict it.

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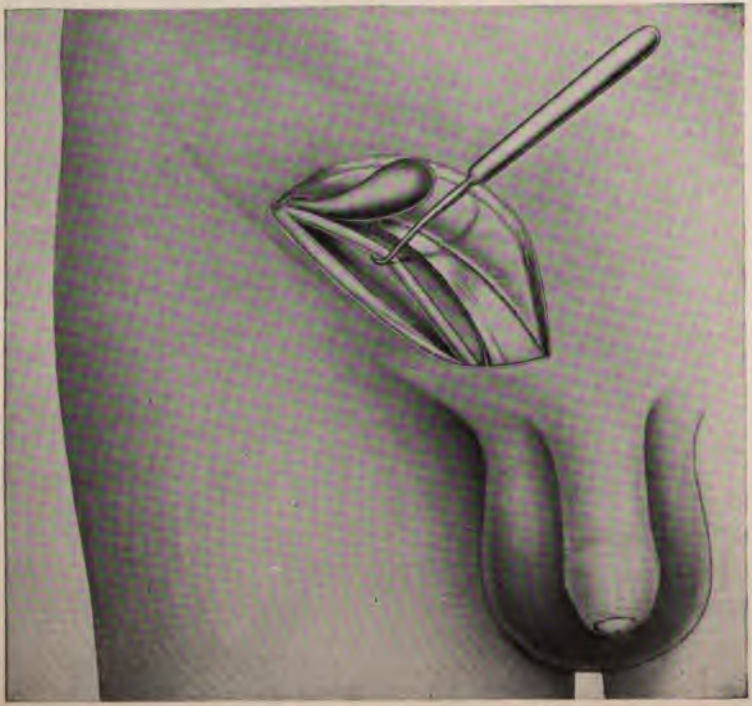
[1] Annals of Surgery, November, 1897, p. 603.

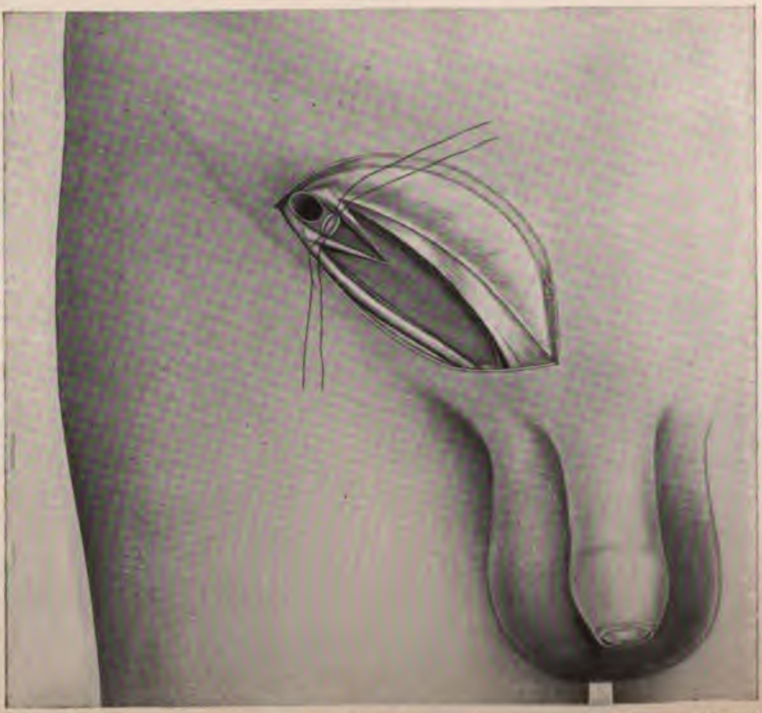
PLATE IV.

Fig. 7.



Inguinal canal opened up from external to internal ring, showing hernial sac and cord isolated. (Fowler.)

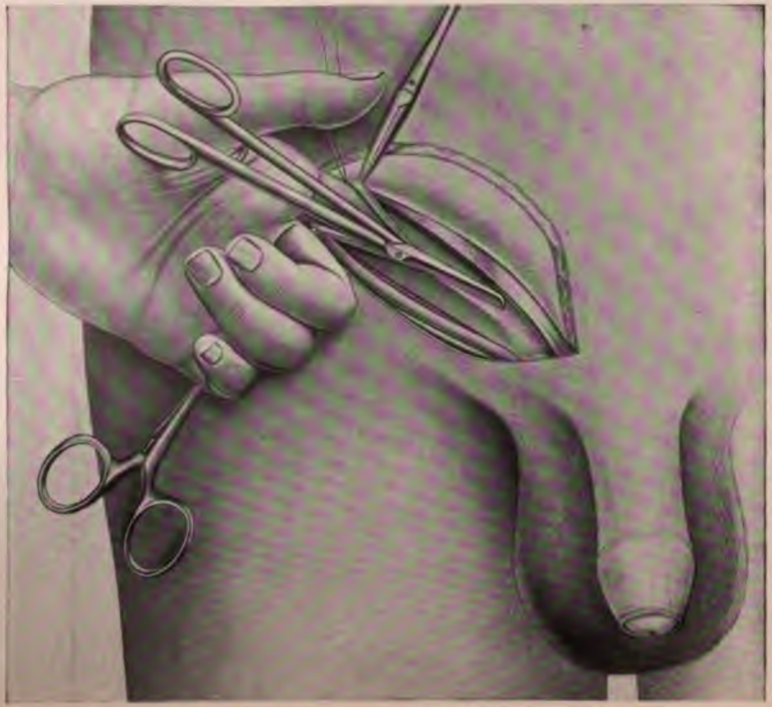
Fig. 8.



Hernial sac cut away, transversalis fascia opened, exposing deep epigastric vessels ligated in two places. (Fowler.)

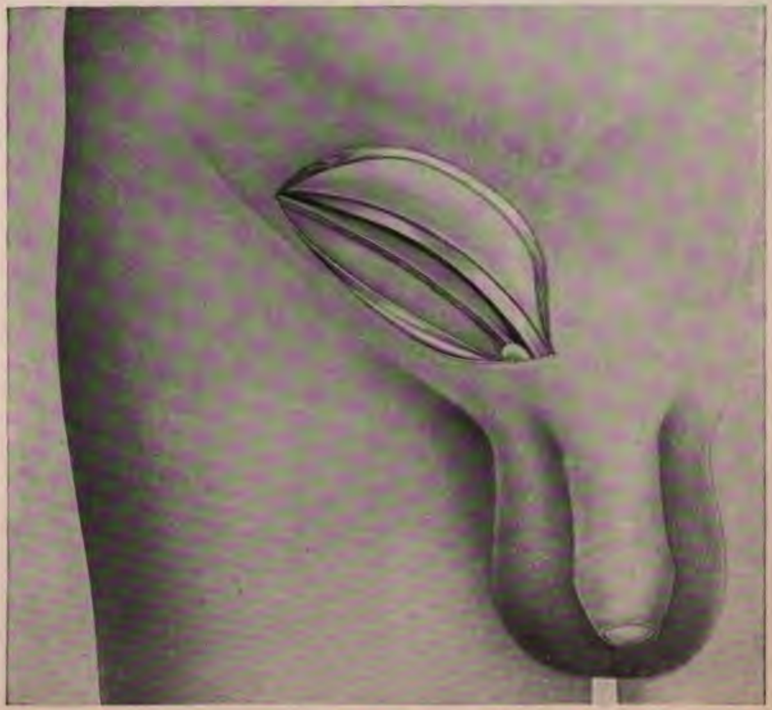
PLATE V.

Fig. 9



Incision of posterior wall of inguinal canal. (Fowler.)

Fig. 10.



Posterior wall of inguinal canal restored. (Fowler.)

The inguinal canal, the gap in the aponeurosis of the external oblique, and the skin are now closed. Kangaroo tendons should be used for the deep sutures.

One of the disadvantages of the operations of Bassini and Halsted is that the cord is brought out at a new opening at the upper angle of the wound, and that subsequently the dragging of the cord by the weight of the testicle may tend to enlarge the opening and lead to recurrence of the hernia; whereas in the operation of Fowler the cord emerges at the most dependent portion of the wound and rests practically upon the pubes.

In children this is a more theoretical than practical objection, as the cord is very small, closure more perfect, and there is less likelihood of the protrusion of a new hernial sac. Almost all operations performed in young children are successful when the sac is ligated and careful approximation of the canal and pillars of the ring is made.

In order to get equal pressure and to insure prompt healing of the wound, it is best to immobilize the parts. For this purpose a plaster of Paris spica bandage is applied over the dressing, encircling the body as far up as the axillæ and extending as far downward as the knee. The limb should be kept absolutely fixed until the wound is healed. By using a plaster of Paris bandage of this character, and covering it with shellac, all danger of infecting the dressing from soaking it with urine is avoided. The child must be kept in bed for three weeks on an easily digested diet, care being exercised that he is not overfed and that the bowels are moved regularly.

Frequently a retained testicle is a complication of the hernia, and an effort should be made in the course of the operation to pull the testicle down and secure it in its place in the scrotum with a chromicized catgut suture, which is used to give greater security. This can frequently be done with comparatively little tension, and then the wound closed as in an ordinary operation. If, however, it is impossible to accomplish this, castration should be unhesitatingly done. The most important points in the successful operations for the radical cure of hernia are absolute asepsis, perfect hæmostasis, and immobilization of the parts. Silk may be used for the sutures, and frequently answers very well. Kangaroo tendon, on the other hand, is stronger than catgut; it lasts about three months, yet it becomes absorbed in time; hence it is not so much a foreign body as silver wire. Chromicized catgut may be used also with very satisfactory results.

If during the operation there be much shock, a rectal injection of from four to six ounces of normal salt solution, to which whiskey and carbonate of ammonium have been added, may be given. No food whatever should be allowed for at least twelve, and preferably eighteen, hours, and no drink of any sort, if it be possible to get along without it. At the end of eighteen hours, and preferably twenty-four, peptonized milk may be given, an ounce every two hours, the quantity to be increased if it is properly digested. Then soups, and plain milk diluted with lime water, may be permitted in the course of forty-eight hours from the time of the operation. If the operation has been performed with care and the fascia united, no truss need be worn. In fact, the pressure of a truss will in part defeat our object, as the pressure upon a recent scar will cause rapid absorption of the tissues and predispose to hernia rather than aid in its prevention.

As this is an operation of expediency, except in the few cases of strangulation where operative interference is imperative, it should never be undertaken unless the child is in good health. A feeble state from constitutional causes or wasting diseases, or during a time of marked intestinal disturbance, contra-indicates all operative procedures. Children of the well-to-do class who can be under constant medical supervision may wear a truss for a much longer time, in the hope of obtaining a cure of the hernia without operation, but under no circumstances should we wait longer than two years in the belief that a cure will result. In the children of the poor, however, a radical operation should be undertaken much earlier, for it is seldom or never possible to obtain the attention necessary to the proper adjustment of a truss, while personal cleanliness is difficult to obtain,—a very important factor at all times, but especially when a truss is worn. Then, too, the child will probably be compelled to earn his living at hard physical labor, thereby running greater risk of strangulation.

Under all circumstances an operation should be performed, first, whenever a thorough trial of a properly fitting truss has been unsuccessful in effecting a cure of the rupture, which trial should not extend over a period longer than two years; second, whenever the inguinal ring remains persistently large; third, whenever the pillars of the ring are badly developed; and, fourth, if there be an undescended testicle or an adherent omentum or intestine.

UMBILICAL HERNIA.

Unless in very exceptional and in very large umbilical herniæ, no operative measures should be undertaken, as the results, at best, in children are only fair, and the large majority will recover by the use of a truss, if properly applied and persistently worn.

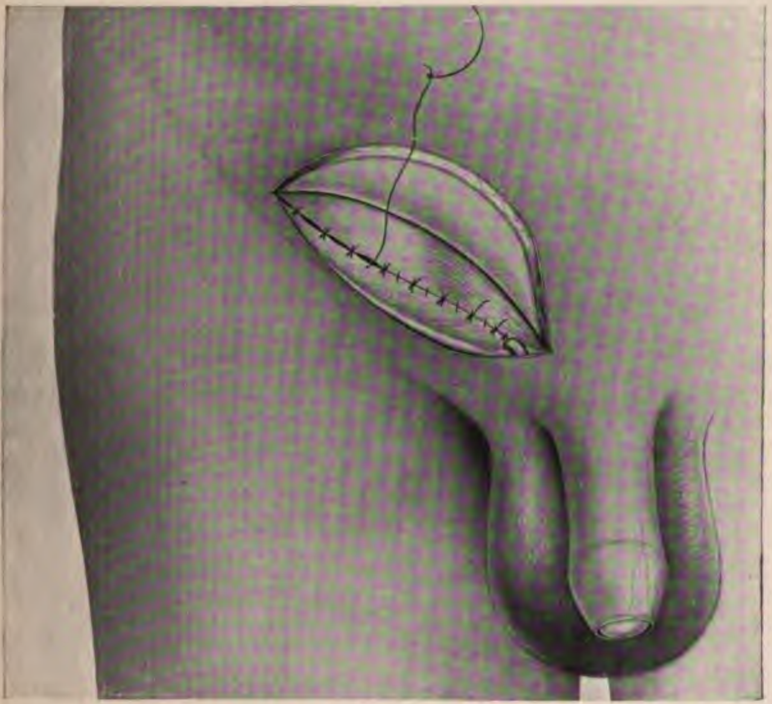
If, however, for any reason an operation is deemed advisable, it should be carried out with the same care and attention to detail as in an ordinary abdominal section. The edges of the umbilical opening should be freshened, the peritoneum closed by a continuous catgut suture, a row of kangaroo tendon sutures involving the fascia and muscles, and the skin closed by a subcuticular suture or by simple interrupted sutures.

FEMORAL HERNIA.

From the anatomical relations of the parts involved in its production, femoral hernia in children is extremely rare and is always acquired. While a truss if properly fitted will retain the hernia in place, no expectation need be entertained of curing this condition by palliative means, as it is almost never accomplished. The pad of a truss cannot be placed in such a posi-

PLATE VI.

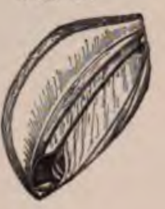
FIG. 11.



Obliteration of the inguinal canal. (Fowler.)

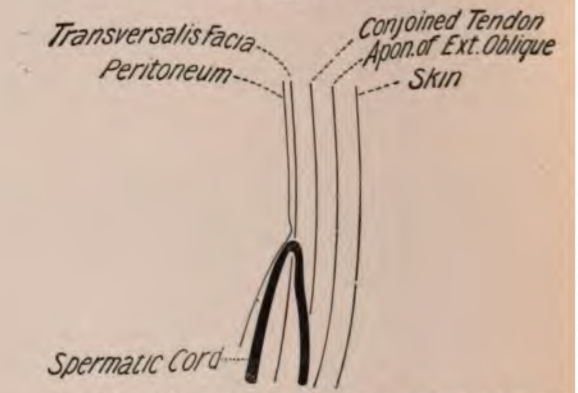
PLATE VII.

FIG. 12.



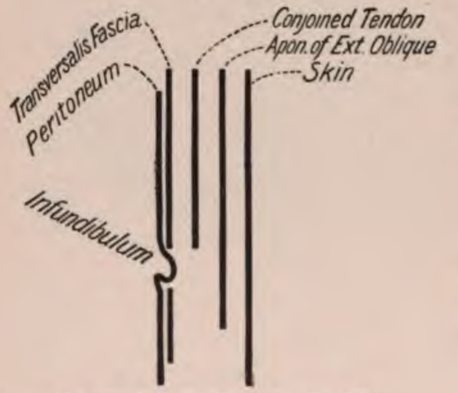
Illustrating the placing of the cord in the peritoneal cavity slit in sac. (Fowler.)

FIG. 13.



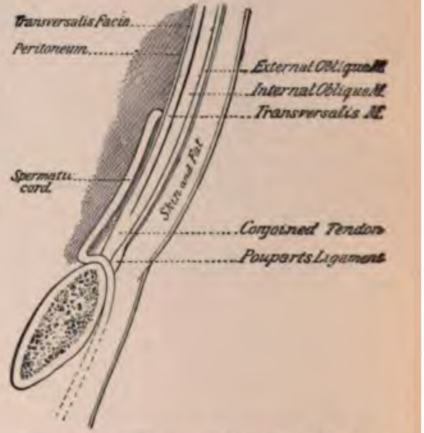
Showing the depression which occurs at the site of the internal ring, following the completion of the descent of the testicle. (Fowler.)

FIG. 14.



Showing the infundibulum. (Fowler.)

FIG. 15.



Showing new position of the cord. (Fowler.)

tion as to produce irritation sufficient to secure adhesions and obliterate the canal. An operation should therefore be undertaken for its radical cure, and should be done early, before the tissues have become stretched and lose their elasticity. The few cases that are seen occur, as a rule, in children much debilitated or wasted by illness, as is instanced by a child two years of age under my care, who had double femoral hernia, which occurred during the course of a severe attack of whooping-cough. The results of operative measures are highly satisfactory.

Dr. Coley[1] has operated upon fifteen cases in children under fifteen years of age without a death. Fourteen of these cases were traced, and remained well from two to five and a half years after the operation. Many other surgeons report equally satisfactory results, and as we are sure that a truss will not cure the condition, it is wise to operate in all cases just as soon as the general health of the child will permit. The operation is easily and quickly performed, and the method of Bassini is probably the best. He makes an incision parallel to Poupart's ligament and over the centre of the tumor, then ligates the sac as high up as possible, and cuts away the excess, if there be any. He then closes the canal by means of two rows of interrupted sutures, the first consisting of three sutures of silk or kangaroo tendon which unite the pectineal fascia with Poupart's ligament, and the second of four sutures passed through the cribriform fascia and Poupart's ligament and fascia and ending just above the saphenous opening. The wound is then closed without drainage.

The operation may be done also by ligating the sac as high up as possible, and then closing the femoral canal by a purse-string suture of kangaroo tendon. Both of these methods give very satisfactory results.

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[1] Personal communication, October 9, 1897.

[The writer takes pleasure in acknowledging his great indebtedness to Dr. Halsted for permission to use these beautiful plates illustrating his operation for the radical cure of inguinal hernia, and also to Dr. Fowler for permission to use the illustrations of his new operation for the radical cure of inguinal hernia.]

INTESTINAL OBSTRUCTION IN CHILDREN.

BY JOHN ASHHURST, JR., M.D., LL.D.

OBSTRUCTION of the bowels in children may be due to *congenital malformations* affecting the rectum or, more rarely, other portions of the intestinal tract; to the introduction of *foreign bodies;* to the presence of *fecal accumulations* or *enterolites;* to *strangulation* by diverticula or by bands and adhesions, either congenital or the result of previous inflammation; very seldom to *volvulus,* or twisting of the bowel; not very rarely to *enteritis* or inflammation followed by paralysis of the intestinal wall; and most commonly to *intussusception,* or invagination of one portion of bowel within another. As in the adult, the course of the disease may be *acute, subacute,* or *chronic.*

CONGENITAL MALFORMATIONS.

The congenital defects of the anus and rectum which may cause intestinal obstruction may be conveniently classified as partial or complete occlusion of the anus, imperforate anus, occlusion of the rectum, imperforate rectum, and occlusion or imperforation with abnormal openings into other parts.

PARTIAL OCCLUSION OF THE ANUS.—A small opening exists, which, though insufficient to allow the proper evacuation of the bowel, yet permits the escape of a small portion of meconium, and thus deceives the practitioner, who may probably not recognize the condition until the marked discomfort of the infant and the gradual distention of the abdomen lead him to make an examination, when the opening will be found to be so small as barely to admit a probe. The *treatment* consists in introducing a blunt-pointed knife and cautiously making radiating incisions, the finger or a bougie being subsequently employed in order to prevent recontraction.

COMPLETE OCCLUSION OF THE ANUS.—Here the anus is closed by a more or less thick membrane which may be seen to become tense and bulging when the child cries or struggles, and through which the color of the meconium may be recognized. The symptoms are more urgent than in the previously described condition, and the *treatment* consists in making a crucial incision, cutting away the resulting flaps, and arresting hemorrhage by stitching together the skin and mucous membrane. If this cannot be done, the rectum should be packed, as otherwise concealed bleeding may lead to a fatal issue. The use of a bougie is required to maintain dilatation.

IMPERFORATE ANUS.—In this condition the anus is entirely wanting, a dense fibrous or fibro-cellular mass occupying its normal position, and varying in thickness from a quarter of an inch to an inch; behind this the rectum is found, terminating in a blind pouch. If the case is not very urgent, the surgeon may wait a day before operating, in hope that the accumulating meconium may cause slight bulging and thus facilitate his manipulations. In the *treatment* of imperforate anus an incision about an inch in length is made from the coccyx forward, along the raphe of the perineum, and cautiously deepened, keeping strictly to the median line and following the curve of the sacrum until the rectum is reached, when its contents should be freely evacuated and the rectal mucous membrane brought down and fastened to the skin with fine silk sutures. If this cannot be done, plugging must be resorted to as a means of preventing hemorrhage. The opening is maintained by the subsequent use of a bougie.

OCCLUSION OF THE RECTUM.—Here the anus presents a normal appearance, the occluding membrane occupying a position from half an inch to an inch above the external opening, and the condition is therefore commonly not recognized until the symptoms of intestinal obstruction lead the surgeon to make an examination, when a bulging diaphragm will be found in the position indicated. A free incision would not be safe in this situation, and the *treatment* consists, therefore, in making a small opening which may be afterwards stretched with dressing-forceps, or cautiously enlarged with a bistoury carrying a concealed blade which is projected after the introduction of the instrument. Hemorrhage is to be prevented by plugging the rectum, and the use of the bougie will probably be required for some months after the operation.

IMPERFORATE RECTUM.—This is a much graver condition than those already described, the whole rectum being absent and the anus ordinarily being imperforate also. A dilated pouch, representing the termination of the colon, may be found opposite the promontory of the sacrum, or may occupy the left (very rarely the right) iliac fossa, often with a long, floating mesocolon. The diagnosis of this condition from that of imperforate anus is extremely difficult, but, according to Holmes, may sometimes be made by introducing a sound into the bladder or vagina; if the instrument seems to strike directly upon the posterior pelvic wall, it may be inferred that the rectum is absent. In the treatment of imperforate rectum the surgeon must choose between (1) a perineal incision, as in the case of imperforate anus, (2) colotomy, lumbar or inguinal, and (3) an attempt to reach the bowel by means of an aspirating needle, over which a larger tube may afterwards be slipped, or with a trocar and canula. The last-mentioned method is at best a blind procedure, and seems to me unjustifiable except in the contingency of the failure of the perineal operation and the positive refusal of the child's parents to permit the formation of an artificial anus. It is more than probable that a needle or trocar thrust in this manner into the abdomen will perforate a fold of peritoneum, when fecal extravasation and fatal peritonitis will almost certainly follow. The proper course, in my judgment, is to make a perineal incision as in the case of imperforate anus, cautiously following the curve of the sacrum until either the bowel is reached or it becomes evident that further search for it in this direction will be unavailing. Great care must be taken not to wound the peritoneum, the bladder, the vagina, or the iliac vessels. Verneuil advises a preliminary excision of the coccyx as a means of obtaining more room for the subsequent dissection. The bowel being reached, every effort should be made to bring it down and stitch it to the external wound, as otherwise the artificial passage will be apt to contract and form a troublesome sinus.

If the bowel cannot be found by the perineal operation, left inguinal colotomy (by Littre's method) should be resorted to. A small incision is made above and parallel to Poupart's ligament, and the colon, which may be recognized by its longitudinal bands, is secured to the parietes by silk stitches passing only through its serous and muscular coats; a small transverse opening is then made in the bowel, and its cut edges are fastened to those of the external wound by a second set of sutures. In the rare cases in which the sigmoid flexure and descending colon are absent as well as the rectum, the cæcum should be opened in the right groin, as advised by Huguier. I have recommended inguinal rather than lumbar colotomy, because although statistics still show the posterior operation to be less fatal than the anterior in the adult, yet in these cases of congenital malformation the weight of evidence is in favor of the latter method.

Should the patient survive, an attempt might be made to restore the natural passage by bringing down the gut by Demarquay's and Byrd's plan of introducing through the artificial anus a leaden or elastic ball, armed with a thread which is brought out at the perineum and gradually tightened.

CONGENITAL MALFORMATION WITH ABNORMAL OPENINGS.—In any of these varieties of malformation there may be an abnormal communication between the bowel and the vagina in the case of a female infant, the urethra or bladder in the case of a male, and the external surface of the body in either sex. If there be a *vaginal* opening, the best mode of *treatment* is to introduce a blunt hook or bent director through the abnormal aperture and make its end project in the position of the anus, when it may be cut down upon and the skin and mucous membrane then stitched together, leaving the vaginal orifice to be dealt with, if necessary, at some future time by a plastic operation. A *vesical* or *urethral* opening is best dealt with by introducing a small staff or curved director into the bladder and laying the parts freely open, as in recto-vesical lithotomy. If there be an opening on the *surface*, provided that it be in the vicinity of the normal anus, an incision may be made so as to restore the natural passage, the case being then treated as one of fistula in ano; but if far distant, as in the groin or umbilical region, showing that a large portion of the bowel is absent, it will be more prudent to decline interference, and simply dilate the abnormal opening so as to prevent fecal accumulation.

CONGENITAL OCCLUSION OF THE SMALL INTESTINE is occasionally met with, and if complete may be looked upon as necessarily a fatal condition; partial occlusion is compatible with some prolongation of life, and may not excite attention until the contracted portion of the bowel is suddenly closed by a plug of mucus or a mass of undigested food, when symptoms of obstruction follow. The only hope from *treatment* would be in division or resection of the constricted gut, and this, of course, would usually be out of the question in the case of an infant.

FOREIGN BODIES IN THE INTESTINES.

FOREIGN BODIES, while frequently swallowed by children, very seldom cause intestinal obstruction. The most important point in the matter of treatment is to avoid the use of purgatives, which can do but harm in these cases, and to give the child abundance of soft food—rice, milk-toast, mush, etc.—or mashed potatoes, as in Billroth's so-called "potato cure." If the foreign body be too large to be disposed of in this way,—as a spoon, pencil-case, button-hook, etc.,—and if symptoms of acute obstruction actually occur, *laparo-enterotomy* will be required. The abdominal cavity having been opened, the foreign body is sought for, and, having been found, a small longitudinal incision is made in the gut, and after removal of the offending substance is again closed with a Lembert suture, the external wound being treated as after abdominal section generally. It is possible that aid in locating the foreign body might be obtained by using the Roentgen ray. Foreign bodies are occasionally introduced into the rectum, but may generally be safely extracted thence, *per vias naturales*, by the cautious use of forceps, etc.

ENTEROLITES.

ENTEROLITES, or *intestinal concretions*, are sometimes met with in children, and may consist of phosphatic salts mingled with animal matter and cholesterin; of vegetable substances derived from the food, as in the *avenolites*, or oat-stones, said by Treves to be not rare in Scotland; or of various substances swallowed as medicines, such as the carbonate of magnesium, iron, chalk, or benzoin. *Gall-stones*, which are an occasional cause of obstruction in adults, are almost never found in childhood, and enterolites are rare at any period of life. The *treatment* of obstruction from an intestinal concretion should consist in the administration of opium, preferably combined with belladonna, the use of cataplasms externally, and the administration of enemata, so as gradually to solicit the bowels, as it were, from below. Purgatives by the mouth should be forbidden. Laparo-enterotomy should be considered a last resource.

FECAL ACCUMULATIONS.

Fecal accumulations are not uncommon in adults, but are rare in children, though occasionally observed. The symptoms are, at first at least, those of the chronic form of obstruction, the most striking local change being a tumefaction, sometimes distinctly doughy to the touch and pitting on pressure, and usually occupying the *right* side, in this respect differing from the tumor of intussusception, which is on the *left* side. The *treatment* consists in the use of repeated enemata, with gentle friction and kneading externally, and the administration of opium, if necessary, to relieve pain. When the mass has begun to move and to undergo disintegration, salines may be cautiously given by the mouth. The enormous distention sometimes caused by fecal accumulations may lead to the formation of ulcers (*stercoral ulcers*), and the cicatrization of these in turn may cause constriction, and thus prove a source of organic obstruction in later life. More rarely the distention may induce perforation, followed by extravasation and fatal peritonitis.

VOLVULUS.

Volvulus is a term used to designate three distinct conditions: in the most common the bowel is folded or twisted about an axis composed of the mesentery or mesocolon; more rarely it is twisted about its own axis; and occasionally two separate portions of bowel are twined or knotted together. The occurrence of volvulus requires a relaxed and elongated state of the mesentery, which may be congenital, but is oftener acquired, so that volvulus is seldom met with in childhood. The tumefaction in volvulus is irregular, and at first often limited to the left side, but ultimately the whole abdomen becomes distended. The pain is severe, at first paroxysmal, but afterwards continuous, and chiefly referred to the umbilicus and sigmoid flexure, and accompanied by much tenderness. There is complete constipation, but vomiting is not a marked symptom, and when it occurs it sometimes gives temporary relief. The *treatment* consists in untwisting or disentangling the gut, after laparotomy; and an attempt may properly be made to prevent the recurrence of the displacement by shortening the mesentery, folding it upon itself and fixing it with sutures, thus taking a reef, as it were, in the relaxed and elongated membrane.

There still remain to be considered the most important conditions which lead to intestinal obstruction in childhood,—viz., enteritis, internal strangulation, and intussusception.

ENTERITIS.

By this term is meant, of course, parenchymatous inflammation of the bowel,—the *enteritis phlegmonodes* of Cullen,—which Sir Thomas Watson has happily described as being "in most cases peritonitis and something more." It may be caused by exposure to cold and wet, and may follow the use of indigestible food or the abuse of cathartics. I have seen a case in a college student in which the attack was precipitated by over-indulgence in green apples, and another in a young child in which the origin was traced to the ingestion of a large quantity of tomatoes with their skins. The first *symptom* is usually pain, colic-like in character, followed by marked tenderness over the affected part of the gut. Vomiting is not very urgent, and seldom of a stercoraceous character. The bowels do not move spontaneously, but if enemata be administered they will often return discolored, or may even bring away small particles of fecal matter, showing that the obstruction is not absolute. There is dorsal decubitus, and if general peritonitis follows the limbs will be drawn up as in that disease. There may be a certain fulness and induration over the inflamed segment of intestine, but no well-defined tumor as in intussusception; and, on the other hand, there is no collapse, as in internal strangulation (unless gangrene has occurred), but a moderate elevation of temperature, usually increased if the inflammation becomes diffused.

Prognosis.—The prognosis of enteritis is favorable if the patient is judiciously treated, but by ill-advised medication many cases are allowed to end in general peritonitis and death. It seems to be almost impossible to make patients and their friends, and even many practitioners, understand that the constipation of enteritis is not the cause, but only a consequence, of the disease. The only thought is to get a movement of the bowels, and the patient is plied with cathartics in large and oft-repeated doses, sometimes, happily, with no effect, but too often with the most disastrous results. As a matter of fact, the constipation is not caused by an accumulation of fecal matter, but by the paralyzed state of the bowel due to its inflamed condition. Subdue the inflammation, and the bowels will move of themselves.

Treatment.—In the treatment of enteritis, if the patient's general condition permit, great benefit may be derived from the application of leeches over the seat of greatest pain and tenderness, but in a very young or in a feeble child this remedy will not be admissible, as such patients do not well support the loss of blood, and under such circumstances the use of a blister may be substituted. After the leeching, the whole abdomen should be covered with mercurial ointment, or with mercurial and belladonna ointments in equal parts, thickly spread upon lint, and this again may be covered with a warm poultice of flaxseed meal, renewed as often as it becomes cool. Opium should be given, preferably by suppository, not only to relieve pain, but also for its effect in modifying the processes of inflammation, and it may properly be combined with belladonna if this is not already being employed locally. I cannot subscribe to the doctrine, so fashionable at the present day, which declares that opium should be avoided in abdominal inflammations because it "masks the symptoms." The only symptoms masked are pain and tenderness, and not only is their abolition desirable in itself, but the facility afforded by their absence for physical examination by palpation, [illegible] aid to diagnosis. The dose of opium [illegible] age of the patient; for a child six years old, [illegible] of a grain of the extract of opium with [illegible] extract of belladonna may be given every hour [illegible] the child is awake, watching the breathing and [illegible] if the respirations are reduced to twelve in the [illegible] using opium will be recognized as that recommended [illegible] in the treatment of peritonitis. If properly carried [illegible] successful now as it was in Dr. Clark's day, and it [illegible] of treatment which in my judgment is worthy of confi-[illegible]

[illegible] tendency to vomiting it is well to give little if any food [illegible] and to rely mainly upon nutritive enemata. Peptonized or [illegible] partially digested foods may be used for this purpose, but upon [illegible] I know of nothing better than the yolk of an egg with beef tea. For a child of six years half a yolk with a fluidounce and a half of beef tea may be given every three hours, and will ordinarily be retained and absorbed without difficulty. During the whole course of the disease cathartics by the [illegible] should be positively forbidden, but after the acute symptoms have subsided, an opening enema, as of warm soapsuds and olive oil, may be administered once or twice a day, and, besides keeping the rectum in a healthy condition suited for the absorption of nutriment, will be found rather soothing than otherwise to the patient.

INTERNAL STRANGULATION.

This is rare in children, but may occasionally be met with, the strangulation being caused by Meckel's diverticulum (a remnant of the vitelline duct passing from the lower end of the ileum to the umbilicus); by an adherent appendix vermiformis; by slits or holes in the mesentery or omentum; or by bands, sometimes originating in peritoneal adhesions, the result of previous inflammation, but more often probably, as taught by Fitz, in obliterated or patent omphalo-mesenteric vessels. The most striking *symptom* of internal strangulation is pain,—sudden, severe, and continuous,—but without tenderness until after the development of peritonitis; the pain is colicky in character and usually referred to the umbilicus. There is early and distressing vomiting, accompanied by great thirst and becoming stercoraceous about the fifth day. Constipation is complete, and the flow of urine is often diminished. There is marked collapse, with subnormal temperature, which may not rise even after the occurrence of peritonitis. The only *treatment* which offers any hope of benefit is prompt laparotomy with disentanglement of the strangulated gut, any constricting bands being divided between two ligatures as a precaution against hemorrhage. After the operation opium should be administered in moderate doses until the risk of peritonitis has passed, and no food should ordinarily be given by the mouth for the first twenty-four hours.

INTUSSUSCEPTION.

INVAGINATION OR INTUSSUSCEPTION of the bowel, which is much the most common form of mechanical obstruction occurring in the intestines of children, consists in the introduction of one portion of bowel into an adjoining portion, as into a sheath, the latter being almost always below,—that is, nearer the anus than the part which enters it. Thus, the jejunum becomes invaginated into the ileum, that into the cæcum, the colon into the rectum, etc. This is sometimes spoken of as *direct* intussusception, to distinguish it from the *retrograde* form, in which a distal enters a proximal portion of gut, and which, though not rarely seen among the multiple invaginations which occur in the act of dying, is very seldom met with during life, so as to demand treatment. A complete intussusception contains three layers of intestine, each embracing all the coats of the bowel; the innermost is called the *entering layer*, and together with that which comes next, the middle or *returning layer*, constitutes the invaginated part, or the *intussusceptum;* while the outermost layer, or that into which the others enter, is called the *sheath*, or *receiving layer*, the *intussuscipiens.* The junction of the entering and returning layers, the lowest point of the intussusceptum, is the *apex*, while the junction of the returning and receiving layers, the highest point of the intussuscipiens, is the *neck* of the intussusception. *Double intussusceptions*, involving *five* layers of gut, are occasionally seen, either a second intussusceptum having been driven into the first, which then forms its sheath, or the first intussuscipiens with its contained intussusceptum being itself invaginated into a fresh portion of bowel, which then constitutes a second sheath. Very rarely *triple intussusceptions* containing *seven* layers of bowel are met with.

Locality of Intussusception.—In more than half of all cases intussusception occurs in the neighborhood of the ileo-cæcal valve, the cæcum itself usually becoming inverted and following the small into the large intestine, the invagination thus increasing at the expense of the latter, and the neck of the intussusception continually shifting its position, while its apex remains constant, being formed by the ileo-cæcal valve, which may ultimately protrude through the anus. This, which constitutes the *ileo-cæcal* form of intussusception, is much commoner than the *ileo-colic* variety, in which the small intestine slips through the ileo-cæcal valve, which then remains fixed as the neck of the intussusception, this increasing, with a constantly changing apex, at the expense of the lesser bowel. *Ileal* and *jejunal* intussusceptions, in which the small intestine alone is involved, form less than one-third of the whole number of cases, while the remainder, about one-sixth, are *colic* intussusceptions, and are limited to the large bowel.

In all invaginations except those of the ileo-colic variety the increase is at the expense of the intussuscipiens, the resulting tumor thus changing its position, and in the most common or ileo-cæcal variety, though ori [illegible] the midline or even the left side of the [illegible] to be recognized by external palpation. [illegible] the intussusceptum may, as already mentioned, [illegible] through the anus, and can almost always be felt, in [illegible] digital exploration of the rectum. As the entering [illegible] carries the mesentery with it, traction is exerted [illegible] the intussusceptum, and the apex is thus displaced, the [illegible] curved or sometimes sharply flexed upon itself, in this way [illegible] the degree of obstruction, and perhaps leading to a sudden aggra[illegible] the symptoms from the occurrence of partial strangulation of the [illegible]. The amount of bowel included in intussusceptions varies from a [illegible] to several feet.

Mechanism of Intussusception.—The causes of invagination have been [illegible] studied by Nothnagel, who describes two varieties, a *spasmodic* and a paralytic, the former being the more common. It is ordinarily taught that the intussusceptum is pushed into the sheath by peristaltic [illegible], but this author maintains that the normal gut is rather drawn over the spasmodically contracted portion, and Treves also lays stress upon the influence of the longitudinal muscular fibres of the intestine, acting from the contracted segment as from a fixed point, and thus pulling the uncontracted bowel over the other. Age and sex are commonly regarded as *predisposing causes* of intussusception, the affection being mainly one of childhood and more frequent in boys than in girls; the greater relative length of the infantile colon and the width of the mesocolon no doubt favor the displacement of the bowel, and furnish one reason for the greater liability to invagination in children as compared with adults. General relaxation of the tissues from previous ill health, diarrhœa, the irritation caused by undigested or unwholesome food, intestinal polypi, strictures and tumors of the bowel, and old adhesions, may all predispose to intussusception. The *exciting cause* is always to be found in irregular and augmented peristaltic movements, whatever their origin.

Morbid Anatomy.—Within a short time, usually, after the occurrence of invagination, the adjacent serous surfaces of the entering and returning layers become adherent, the adhesions being most dense in the region of the neck, though sometimes extending over a considerable space; they are rarely limited to the apex. In chronic cases, however, according to Baur and Carter, as quoted by Mayland, adhesions may be absent after a duration of even several months. Pressure may cause ulceration and sometimes perforation of the intussuscipiens, but ordinarily the sheath, though congested and inflamed, is not otherwise changed. The intussusceptum, however, in acute cases becomes strangulated, and is eventually separated as a gangrenous mass, when, if the adhesions are firm, it may pass downward and be evacuated through the anus, either in one piece or in fragments, and the patient may thus recover, at least temporarily. If the adhesions yield, however, fecal extravasation may occur, with consequent fatal peritonitis,

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IMPERFORATE ANUS.—In this condition the anus is entirely wanting, a dense fibrous or fibro-cellular mass occupying its normal position, and varying in thickness from a quarter of an inch to an inch; behind this the rectum is found, terminating in a blind pouch. If the case is not very urgent, the surgeon may wait a day before operating, in hope that the accumulating meconium may cause slight bulging and thus facilitate his manipulations. In the *treatment* of imperforate anus an incision about an inch in length is made from the coccyx forward, along the raphe of the perineum, and cautiously deepened, keeping strictly to the median line and following the curve of the sacrum until the rectum is reached, when its contents should be freely evacuated and the rectal mucous membrane brought down and fastened to the skin with fine silk sutures. If this cannot be done, plugging must be resorted to as a means of preventing hemorrhage. The opening is maintained by the subsequent use of a bougie.

OCCLUSION OF THE RECTUM.—Here the anus presents a normal appearance, the occluding membrane occupying a position from half an inch to an inch above the external opening, and the condition is therefore commonly not recognized until the symptoms of intestinal obstruction lead the surgeon to make an examination, when a bulging diaphragm will be found in the position indicated. A free incision would not be safe in this situation, and the *treatment* consists, therefore, in making a small opening which may be afterwards stretched with dressing-forceps, or cautiously enlarged with a bistoury carrying a concealed blade which is projected after the introduction of the instrument. Hemorrhage is to be prevented by plugging the rectum, and the use of the bougie will probably be required for some months after the operation.

IMPERFORATE RECTUM.—This is a much graver condition than those already described, the whole rectum being absent and the anus ordinarily being imperforate also. A dilated pouch, representing the termination of the colon, may be found opposite the promontory of the sacrum, or may occupy the left (very rarely the right) iliac fossa, often with a long, floating mesocolon. The diagnosis of this condition from that of imperforate anus is extremely difficult, but, according to Holmes, may sometimes be made by introducing a sound into the bladder or vagina; if the instrument seems to strike directly upon the posterior pelvic wall, it may be inferred that the rectum is absent. In the treatment of imperforate rectum the surgeon must choose between (1) a perineal incision, as in the case of imperforate anus, (2) colotomy, lumbar or inguinal, and (3) an attempt to reach the bowel by means of an aspirating needle, over which a larger tube may afterwards be slipped, or with a trocar and canula. The last-mentioned method is at best a blind procedure, and seems to me unjustifiable except in the contingency of the failure of the perineal operation and the positive refusal of the child's parents to permit the formation of an artificial anus. It is more *of urine*, a symptom which depends rather upon the acuteness than upon the locality of the disease.

Discharges of blood and mucus accompanied by *rectal tenesmus* are characteristic of acute intussusception, and are regarded by Pollock as almost pathognomonic; in these cases fecal matter is usually absent from the stools on account of the lateral displacement of the gut by mesenteric traction and of the secondary enteritis; but in chronic cases, on the other hand, fecal passages may continue while the bloody and mucous discharges are absent.

The *tumor* of intussusception is a very characteristic symptom, occurring usually on the left side, and being elongated and sausage-like in character. It is painful and tender to the touch, and in children may often be felt by a finger in the rectum; it sometimes causes patulousness of the anus, and may even protrude through that orifice. It may commonly be distinguished from the tumor of fecal impaction by the fact that the latter occurs ordinarily on the right side and may pit on deep pressure, while in intussusception the right side is often flattened and depressed (Dance's sign) from the displacement of the invaginated intestine.

Chronic intussusceptions have been particularly studied by Rafinesque, who says that sixty per cent. are ileo-cæcal and ten per cent. ileo-colic, the remaining thirty per cent. being about equally divided between the large and the small intestine. Their symptoms are much less definite than those of the acute variety, the tumor changing its shape and place from time to time, constipation being absent or sometimes alternating with diarrhœa, and the pain and vomiting perhaps occurring only at long intervals. The characteristic bloody and mucous discharges, tenesmus, and fever may be entirely wanting.

Diagnosis.—Invagination may be mistaken for colic, appendicitis, enteritis, dysentery, impacted fæces, and other varieties of mechanical obstruction. The paroxysmal pain, bloody and mucous discharges, tenesmus, vomiting, and presence of the tumor should suffice to distinguish intussusception from simple *colic*. From *appendicitis* and *appendiceal abscess* it may be differentiated by noting the same symptoms, and by observing that in those affections there are tympany, with fulness and tenderness in the right iliac fossa, and early development of fever, contrasting with the right-sided depression and flattening and the late occurrence of fever in invagination. In *enteritis* there are also early fever and constipation, without discharge of blood and mucus, and without any well-defined tumor. I have, indeed, known the convexity of the lumbar vertebræ, in a case of enteritis, to be mistaken for the tumor of intussusception; but this error could hardly be made by a careful observer. *Dysentery* presents the tenesmus and bloody and mucous discharges, but the pain is different, and the other symptoms of the two affections are quite diverse in character. The tumor of *fecal impaction* is on the right side and pits on pressure, and the peculiar discharges of intussusception are absent. The only other form of *mechanical* *obstruction* likely to occur in children is internal strangulation, which may be distinguished by the early occurrence of collapse, the subnormal temperature, and the stercoraceous vomiting.

Prognosis.—Intussusception is always a very grave affection, Fitz's statistics showing a mortality of sixty-nine per cent., and Leichtenstern's larger figures a mortality of seventy-three per cent. Spontaneous reduction of the invagination, which is of course the most favorable termination, can, as a rule, be hoped for only during the early days of the attack, as adhesions ordinarily form promptly. When reduction is not obtained there is still a chance for recovery after sloughing and separation of the intussusceptum, the death-rate after this event being only forty-one per cent. as compared with eighty-five per cent. when sloughing does not occur. There are secondary risks, however, after this mode of cure, which must not be ignored, the contraction which follows cicatrization sometimes leading to the formation of a stricture of the bowel, while internal strangulation at a later period may sometimes find its origin in the preservative adhesions by which the sloughing process was prevented from proving immediately fatal.

Chronic intussusception, while not as dangerous at its onset as the acute variety, has its special risks; for here the possibility of cure by sloughing is commonly absent, and unless relief be afforded spontaneously or by operative interference, the patient, sooner or later, will surely perish from exhaustion.

Treatment of Intussusception.—In the treatment of *acute* intussusception the surgeon should in all cases adopt means to husband the patient's strength, relieve pain, and diminish peristalsis which tends to cause further invagination; and must further choose between attempts to effect reduction without operation, waiting for the chance of recovery after sloughing and separation of the intussusceptum, and some form of abdominal section, followed by reduction, the establishment of an artificial anus, or intestinal resection, according to the special conditions encountered. The first indications are to be met by keeping the patient quiet in bed, by administering opium and belladonna, preferably by suppository, and by giving concentrated food and nutritive enemata. For a child of two years a twelfth of a grain of the extract of opium with a fiftieth of the extract of belladonna may be given every hour or every two hours, according to the urgency of the symptoms, and the abdomen may be covered with mercurial ointment and a warm poultice. As a nutrient enema, half the yolk of an egg beaten up with a fluidounce of beef tea may be given every three hours, the rectum being cleansed once a day with a little soap and water. Only small quantities of food should be given by the mouth, and if vomiting be troublesome, relief may be afforded by washing out the stomach through an œsophageal tube.

Attempts to effect *reduction* of the invaginated bowel without operation may properly be made during the first few days of an *acute* intussusception, but at a later stage would be dangerous, as possibly leading to rupture and fatal extravasation. From four to six days should probably be the limit, and the longer period should be allowed only when the absence or small amount of bloody and mucous discharge shows that the constriction is not absolute. In *chronic* cases it is different, and here these attempts may be made more persistently. Reduction may be aided by the use of large injections, by inflation, or by abdominal taxis.

Injections of warm water, or, which I think better, of warm olive oil, may be administered twice daily in quantities varying from one to six quarts according to the patient's age. They should be given through a long tube, so that they may if possible impinge directly upon the apex of the intussusception and not waste their force upon the walls of the rectum. They are best given with a fountain-syringe, utilizing the force of gravity, the reservoir being raised not more than eight feet in the case of an infant, and not more than twenty in that of an adult, and the patient being etherized and in a partially inverted position, the hips higher than the shoulders and the trunk elevated at an angle of about forty-five degrees. Escape of the injected fluid by the side of the tube may be prevented by using an india-rubber collar devised by Mr. Lund for the purpose, or by wrapping the tube with cotton or lint, which is introduced as a plug within the sphincter.

Inflation, with the same precautions against leakage, may be practised with long-nozzled bellows, or through the long tube, atmospheric air being commonly employed. Dr. Senn prefers the use of hydrogen gas, furnished by an india-rubber balloon holding four gallons, from which the gas is slowly expelled by steady compression. Libur, Jate, and Ziemssen employ carbonic acid gas, supplied in the nascent state by successive injections of solutions of sodium bicarbonate and of tartaric acid.

Abdominal taxis, or systematic kneading and manipulation of the invaginated bowel through the abdominal wall, is a mode of treatment introduced into modern practice by Mr. Jonathan Hutchinson, who advises that it should be employed as thoroughly and persistently as is the ordinary taxis in cases of hernia. I have resorted to this plan once, with entire success, in a patient under the care of Dr. Morris J. Lewis at the Pennsylvania Hospital, but it seems to me that its use should be limited to the early periods—say during the first few days—of an intussusception, or to those cases in which the absence of bloody and mucous discharge from the rectum shows on the one hand that the invagination is of a subacute character, and on the other that spontaneous recovery by separation of the affected portion of gut is hardly to be anticipated. In very acute cases, unless at their beginning, abdominal taxis would certainly be a dangerous remedy, from the risk of producing rupture, and under such circumstances I have no doubt that it would be safer to bring the patient under the influence of opium and await further developments.

The *operative treatment* of intussusception may consist of puncture of the bowel, of enterotomy, or of abdominal section (laparotomy).

Puncture of the intestine with the fine tube of an aspirator serves to relieve the gaseous distention of the bowel, and thus may aid in prolonging life until relief is afforded by sloughing of the invaginated gut. The operation is, of course, very easily performed, and is attended by little, if any, shock, but is not entirely free from risk, since it may lead to fecal extravasation, and under no circumstances could it be expected to prove curative; indeed, intestinal puncture, in spite of the praise which it has received, seems to me to merit little consideration except as a euthanasial measure.

Enterotomy, or, as it is often called, Nélaton's operation, consists in making an incision, ordinarily in the right iliac region, and opening the first distended coil of bowel which is encountered. It is a better operation than mere paracentesis, though attended with more immediate danger, and may properly be employed in any case in which it is thought that the patient's condition demands operation and yet will not permit laparotomy. On the other hand, it is not a curative procedure in cases of intussusception, but only a means of prolonging life till nature, or a subsequent laparotomy, can give permanent relief. Enterotomy may be practised in either of two ways: if it is not designed to form an artificial anus, a loop of intestine is gently drawn out through the wound and packed around with sterilized gauze, and is then opened by a transverse incision and permitted to discharge its contents outside of the abdominal cavity. A large drainage-tube may be passed into the upper end of the bowel, and when the flow of fecal matter has entirely ceased and the abdominal distention has subsided,—this process sometimes requiring several hours,—the intestinal wound is carefully closed with a Lembert suture and the external wound likewise closed and dressed in the customary manner. If, on the other hand, it is determined to form a temporary artificial anus, the bowel should be stitched to the abdominal parietes and cautiously opened, and additional sutures should then be employed to fasten the lips of the intestinal wound to the abdominal wall, so as to prevent any risk of peritoneal infection. When the natural passage has been restored by the sloughing of the intussusceptum or by a secondary laparotomy, the artificial anus may be allowed to close, or, if necessary, may be dealt with by a plastic operation.

Laparotomy,—and this term, for reasons which I have given elsewhere, seems to me a better one than *cœliotomy*, which has been suggested as a substitute, but which is really much more indefinite than the other,—while attended with more risk than enterotomy, has the advantage that it permits the adoption of radical measures for the relief of the intussusception, and seems to me, therefore, to be the preferable operation when the condition of the patient is such as to justify its adoption. It is particularly adapted to cases of chronic invagination, in which there is no prospect of a cure by sloughing, but has also been employed in a considerable number of acute cases, even in quite young children, and with an increasing measure of success, so that it can no longer be regarded as an unjustifiable procedure. My judgment is that in cases in which non-operative measures have failed, and in which the absence or small amount of bloody and mucous discharge shows that a cure by sloughing is not to be expected, laparotomy is proper, provided that the patient be in fairly good general condition, enterotomy being substituted when he is too feeble to endure the graver operation. In the rare cases in which the invaginated bowel protrudes through the anus, excision may be practised, retraction of the gut being prevented by transfixing it with strong pins and thus keeping it in place until healing has occurred.

Laparotomy for intussusception may be performed as follows. The patient is carefully etherized, and the abdominal wall thoroughly cleansed by washing it first with oil of turpentine, then with soapsuds, then with alcohol, and finally with a hot sublimate solution, 1 to 2000. If a tumor can be felt, the incision is made directly over it, but otherwise is made in the median line. The wound is cautiously deepened until the peritoneum is reached, when this is opened by picking it up with two pairs of forceps and making a small aperture, which is afterwards enlarged as much as necessary with blunt-pointed scissors guided by the left forefinger used as a director. The intussusception is drawn out of the wound, the neighboring bowel being restrained by pressure with sterilized gauze, and an attempt made to effect reduction by gently compressing and pushing up the intussusceptum from below, a manipulation which is both safer and more likely to succeed than traction efforts from above. If the intussusception is not readily found, the surgeon introduces his hand through the incision (which in such a case would be median), and, beginning in the right iliac fossa with the cæcum, searches upward or downward according as that part is or is not distended with fæces. The direction in which the search should proceed in examining the small intestine may be determined by remembering that, as pointed out by Mr. Head, the mesentery is attached to the posterior abdominal wall higher on the left side than on the right, beginning at the level of the second lumbar vertebra and crossing over to the right sacro-iliac symphysis. If reduction cannot be accomplished, an artificial anus may be made just above the seat of invagination, or, if the condition of the patient permit, the intussusception may be excised (*enterectomy*), and the cut ends of bowel brought together with a *Murphy's button*, the adjustment of which requires much less time than either a *lateral anastomosis* or a *circular enterorrhaphy*, and is, therefore, to be preferred under these circumstances, since prolonged manipulation of the intestines is very apt to cause an unfavorable termination. After carefully cleansing the parts with dry gauze, or, if blood or fecal matter have entered the peritoneal cavity, by douching with hot sterilized water, a glass drainage-tube is introduced, armed with a thin strip of gauze and a rubber dam, so as to prevent soiling the surrounding tissues, and the wound is then accurately closed with sutures of silkworm gut passed through the whole thickness of the abdominal parietes, and is dressed antiseptically with a strip of protective, a deep and a superficial gauze dressing, a sheet of Mackintosh cloth or water-proof paper, sublimated cotton, and a many-tailed abdominal binder. No food should be given by the mouth until the next day, and pain should be relieved by the use of suppositories of opium and belladonna in such quantities as may be needed.

The limits of this article will not permit a description of the various modes of excising and suturing the intestine, for an account of which the reader is referred to works on General Surgery.

ably merge to such an extent that there can be no dividing line. The term *appendicitis* should be used, therefore, to designate inflammation of the vermiform appendix, no matter how slight or how far advanced the disease may be.

History.—In the beginning of the present century the writers of medicine seldom, if ever, associated general peritonitis with any inflammation arising in the right iliac fossa. There was a great diversity of opinion as to the cause of the peritonitis, much of it being due, no doubt, to the incomplete knowledge of the anatomy of the abdominal cavity and especially of the peritoneum. Laennec gave the first complete description of this membrane in 1803, prior to which time it was supposed that the peritoneum invested none but the solid organs. From that time advances were made in the knowledge of the pathological conditions found in connection with the peritoneum, and it was soon determined that inflammation could, and did, extend by continuity and contiguity of structures from one part of the abdominal cavity to another. Dupuytren, working in this line, first called attention to the fact that abscesses found in the right iliac fossa were often due to disease of the cæcum. His observations were made about 1820, at which time he laid no stress upon the appendix and did not mention it as the original seat of the disease.

Prior to the investigation of Dupuytren, one case had been reported by Mestivier, in 1759, in which a distinct localization of a lesion in the appendix had been made.

J. Parkinson, of England, reported another case of perforation of the appendix in 1812. In 1813 he was followed by Wegeler, who had seen a similar case.

In 1824 Louyer-Villermay reported a case of fatal peritonitis which he attributed to perforation of the appendix.

In 1827 Husson and Dance gave the first detailed description of the diseases found in the right iliac fossa. Dupuytren gave the same theories as Husson and Dance in his "Lectures on Clinical Surgery" in 1833. He described irritation and inflammation of the mucous membrane of the cæcum and the extension of the same to the retro-cæcal tissue and, at times, to the general peritoneum. The appendix was not associated with the trouble in any way.

In 1827 Mélier reported four cases, three of which he described as cases of perforative appendicitis with fulminating peritonitis, the fourth as a case of relapsing appendicitis. He gave full descriptions of the appendiceal disease, and made special mention of two distinct symptoms in the perforative cases,—viz., more or less severe abdominal colic and fixed pain in the right iliac fossa. From his observations in regard to the causes, the character, and the consequences of appendicitis, Mélier first advanced the idea that appendicitis might be a surgical affection, and that operative interference might be of great avail. He said, "If it were possible to establish with certainty the diagnosis of this affection, we could see the possibility of curing the patient by operation. We shall, perhaps, some day arrive at this result."

In 1831 Goldbeck wrote a thesis "On a Peculiar Inflammatory Tumor in the Right Iliac Fossa." His views were similar to those of the French writers, the disease being called perityphlitis. He mentioned a case of perforative appendicitis with resulting peritonitis, and a case of perityphlitis in which the appendix had been found intact.

In 1831 Ferrall published a monograph on "Phlegmonous Tumors in the Right Iliac Fossa." His observations led him to the belief that the primary seat of the disease was in the cæcum, and that the appendix and the peritoneum did not enter into the production of the tumors found to as great an extent as did the retro-cæcal connective tissue.

In 1834 Copland, in his "Dictionary of Practical Medicine," made an advance in the pathology of the inflammatory conditions found in the right iliac fossa. He described what was known as perityphlitis as "inflammation of the cæcum." His descriptions of the diseases of the cæcum were very full and in great detail, but he acknowledged that the appendix might be the primary seat of disease, and that the appendix might necrose or become gangrenous as the result of the presence of a foreign body within the organ.

In 1837 John Burne made marked advances towards the correct diagnosis of inflammatory conditions found in the region of the appendix. His article on "Inflammation of the Cæcum" attempted to differentiate between the diseases of the cæcum and those of the appendix, and he mentioned ulceration of the appendix the result of the presence of a foreign body as a cause of general peritonitis with pus-formation.

In 1839 Burne published a second paper, in which he advanced still further and asserted that but few, if any, of the troubles assigned to the cæcum really started in that organ, the primary seat of the disease being in the appendix.

In 1838 Albers published an article on inflammation of the cæcum, in which he claimed that the primary seat of the trouble was in the cæcum, although he admitted that it might originate in the appendix. He described typhlitis and gave four varieties,—stercoral typhlitis, simple typhlitis, perityphlitis, and chronic typhlitis.

In 1839 Grisolle opposed the writings of Albers, and concluded that the cæcum played but a small *rôle* in these affections, although he held that the cæcum was of more importance in the causation than was the appendix.

In 1840 Louyer-Villermay reported cases of inflammation of the appendix which terminated rapidly in gangrene.

In 1843 A. Voltz published a paper on "Ulceration and Perforation of the Appendix occasioned by Foreign Bodies." He practically ignored the cæcum as the primary seat of the trouble, and stated that the many cases previously reported had been due to the disease of the appendix.

In 1843 Rokitansky, in his work on "Pathological Anatomy," gave the first description of catarrhal appendicitis due to fecal concretions or foreign bodies. He thought that this inflammatory condition might result in perforation with consequent general peritonitis, but that the general peritonitis was not always a necessary result of the perforation, as numerous adhesions might be formed and thus wall off the inflammatory area.

In 1848 Hancock operated on one case of appendicitis, with recovery. His advocacy of early operation met with little, if any, encouragement.

In 1856 G. Lewis published an article in which he held that the mild cases of trouble in the right iliac fossa were due to inflammation of the cæcum, and that the severe ones were caused by inflammation in the appendix, the latter being always induced by a foreign body or concretion. He placed the pus-forming cases under the cæcal variety.

In 1858 Oppolzer described iliac phlegmon, and suggested the term paratyphlitis for that form of phlegmon which was found between the iliac fascia and the pelvic bone.

In 1859 Leudet abandoned the cæcal theory, and stated that ulceration of the appendix was common in cases of phthisis with ulcerations in the cæcum, and that appendiceal ulceration was found in conjunction with, or rather caused by, enteric and typhoid ulcerations and foreign bodies. He asserted that perforation of the appendix was more common than all perforations in other portions of the intestine. He also thought that disease of the appendix might lead to peritonitis or abscess-formation which might open into the rectum, vagina, bladder, or cæcum. He advised large doses of opium and belladonna as the best form of treatment.

In 1867 Willard Parker, of New York, said that three out of every four cases of appendicitis could be saved by early operation. He was followed by others who advocated operative interference in inflammatory troubles in the right iliac fossa, and Noyes in 1883 published a scientific article on the subject, giving a report of one hundred cases of this disease. All these cases had been operated upon, ninety per cent. of the operations having been performed by American surgeons.

In 1875 Samuel Wilkes, in Wilkes and Moxon on "Pathological Anatomy," considered that the appendix seemed to be the seat of the inflammation more often than did the cæcum, although he contended that inflammation of the cæcum itself, independent of the appendix, did occur.

In 1880 C. With, of Copenhagen, stated that peritonitis never originated in disease of the cæcum.

In 1886 Reginald Fitz, of Boston, published an extensive article on the subject, in which he made a report of two hundred and nine cases of typhlitis and perityphlitis and two hundred and fifty-seven cases of perforative appendicitis. He claimed that the symptoms of the two classes of cases were practically the same. In 1888 he published another article, in which he came to the conclusion that the terms typhlitis, perityphlitis, paratyphlitis, and so on, were varieties of one affection, appendicitis.

In 1888 Ransohoff wrote an able paper in which he paid special attention to the anatomical details in connection with the organs occupying the right iliac fossa.

In 1888 Murphy and Sands published articles calling for operative interference.

In 1890 Fenger stated that "perityphlitis, although a Dupuytren wrote its history, has naturally, as an intra-abdominal disease, been almost exclusively in the domain of internal medicine. Surgical interference in abscesses of the iliac fossa was limited to those pointing under the skin, ready to open spontaneously. The writer on internal medicine has studied its symptoms and treated it with opium,—that is, left the pus to take its course; the pathologist has carefully pointed out all the details of the cause, course, and progress of the inflammation; but it has been left to the American surgeon to place suppurating perityphlitis where it properly belongs,—in the domain of surgery."

In 1892 Osler observed that the cases described as perityphlitis and paratyphlitis were, with rare exceptions, appendicitis, and that the former terms should, therefore, be dropped.

In 1893 Kelnyack, of Manchester, England, published a monograph which advanced the knowledge of the disease.

In 1894 Pepper, in his "Text-Book of Medicine," wrote of typhlitis as a disease much less common than formerly supposed, as the affections of an inflammatory character found in the right iliac fossa were due to disease of the appendix.

In 1894 Fowler, of Brooklyn, published a monograph in which he paid special attention to the various positions in which the appendix might be found. He also advocated early operation.

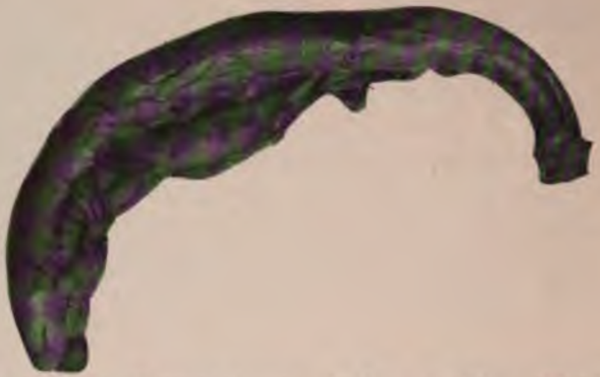
In 1895 Hawkins, of London, published a monograph in which he claimed that all cases of so-called perityphlitis, whether severe or mild, were due to disease of the appendix.

In 1896 the present writer published a monograph in which he asserted that all inflammatory diseases in the right iliac fossa were the result of appendicitis; that appendicitis was solely a surgical affection; and that the appendix should be removed in all cases, whether pus were present or not, where there had been an inflammatory condition of the organ.

In 1897 Morris, of New York, wrote a short treatise on appendicitis in which he took the stand that all appendicitis cases belonged to the domain of surgery, and that all appendices which had once been the seat of inflammation should be removed. He claimed that this procedure was in the line of preventive medicine, in that it protected adjacent structures from contamination.

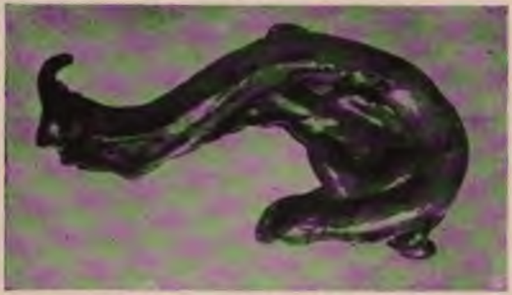
Other writers on the subject of appendicitis during 1896 and 1897 included Chenoweth, who published an article entitled "Etiology of Appendicitis. Why is it more common in the Anglo-Saxons?" Grohe, on "Pathologie und Therapie der Typhlitiden," and Siegel, on "Appendicitis and its Complications." These and numerous other authors have brought

Fig. 1.



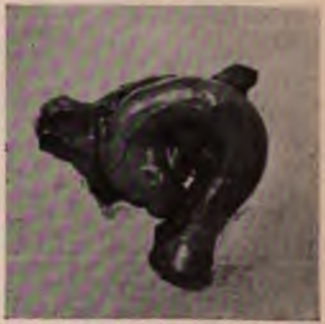
Large appendix. Microscopical examination demonstrated lumen dilated the entire length by fecal concretion.

Fig. 2.



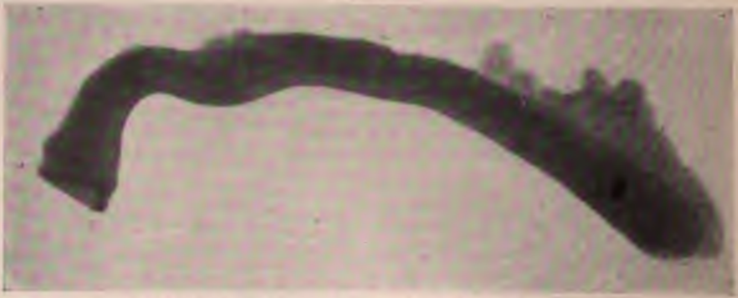
Twist and kink of appendix caused by short mesentery. Fecal matter in lumen.

Fig. 3.



Twist and kink of appendix greatly exaggerated.

Fig. 4.



Foreign body in lumen of appendix. X-ray photograph.

the subject well before the medical and surgical world, so that appendicitis is written about and discussed as much as any other topic in the medical literature of to-day.

Etiology.—The causes of appendicitis may be divided into predisposing and exciting. Among the former may be mentioned the anatomy of the organ, its position, length of mesentery, and so on; the general anatomy of the iliac fossa; age; and sex. The chief of these is the anatomy of the appendix and of the right iliac fossa, a thorough understanding of which is essential not only in explaining the cause of appendicitis, but also in treating the disease.

Anatomy.—Laennec was the first to give a full description of the peritoneum, in 1803, but his researches failed to connect the inflammatory processes around the cæcum with a primary seat of disease in the appendix. Luschka, Oppolzer, Hyrtl, Treves, Ransohoff, Lockwood and Rolleston, and many others have made special reference to the peritoneal distribution in the right iliac fossa, and their writings have added much to the knowledge of the disease of the appendix.

The cæcum and the appendix are, as a rule, entirely covered with serous membrane. The cæcum is devoid of a mesentery in ninety-seven per cent. of all cases (Ransohoff), and for this reason is free in the abdominal cavity. I have yet to find a cæcum with a mesentery, either on the operating-table or post mortem. In the few cases reported, the peritoneum was reflected from the cæcum to the posterior abdominal walls; in the majority of cases the peritoneum is not reflected before the first part of the ascending colon has been reached. This freedom from mesentery allows the cæcum and, consequently, the appendix to move about and to occupy different positions, this mobility depending upon the length of the ascending mesocolon and the point at which the mesocolon begins. In cases of non-descent of the cæcum, the latter may be found high up and to the left of the median line. Leander has reported one case in which the cæcum was found in the left hypochondriac region, near the spleen. It may be an occupant of the pelvis in about fifteen per cent. of all cases.

The peritoneal covering of the appendix is derived from the inferior layer of the mesentery of the ilium. The organ is entirely invested, as a rule, although the peritoneum does not extend to the tip of the organ in a small number of cases, the distal one-third or two-thirds being free. By the reflection of the peritoneum to the appendix a triangular mesentery (meso-appendix) is formed, the base of the triangle being formed by the free edge of the folds. The size and shape of the meso-appendix vary with the length of attachment to the appendix, a small attachment giving a narrower triangle, and *vice versa*.

The length and breadth of the meso-appendix play a most important part in appendicitis. Usually it seems to be too short, and thus draws the tip of the organ backward, causing twists and kinks in it. Especially is this the case where there has been an inflammatory condition. Again, gross structure is similar to that of the large intestine, but its component layers are not so highly organized.

The serous coat is derived from the inferior layer of the mesentery of the ileum, as previously stated. Beneath the serous covering is the muscular layer, divisible into two strata, a longitudinal and a circular. The circular fibres are rather strong, the longitudinal less well developed and not so compact. The muscular power of the organ is much less than is required at times to expel foreign matter from its lumen, and this one fact alone will account for most cases of inflammation in the appendix.

Beneath the muscular layer is the submucosa, formed of areolar tissue and containing many lymph-glands, a small quantity of fat, and a few small arteries and veins which supply the mucous membrane. Beneath the submucosa is the mucous membrane. This consists of a delicate retiform tissue with numerous lymphoid cells in its meshes. Dipping into it are many solitary glands and glands of Lieberkühn. The latter glands may be numerous and large, or entirely absent. The retiform tissue of the mucous membrane is generally lined with a basement membrane covered with clusters of various forms of micro-organisms. The latter are normally found in the intestinal tract, and are apparently harmless as long as the basement membrane remains intact. They become pathological, however, with the slightest abrasion of the membrane.

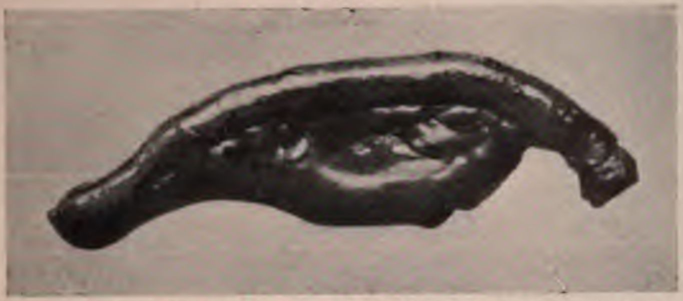
"The liability of the appendix to vary, both in position and size, is probably one of the most important predisposing causes of appendicitis. This organ, functionless and undeveloped, is a narrow, musculo-membranous tube, lined with mucous membrane, ending in a blind extremity, and having a common orifice of exit and entrance; its blood-supply is limited, consisting of one small artery, with no anastomoses to make up for the deficiency of supply; it is an organ of low vitality, on account of the retrograde metamorphosis it is undergoing in the process of evolution; it is rich in lymphoid tissue, a fact that vastly increases its absorptive powers, and when attacked by inflammation is, therefore, more liable to destructive processes."

Age plays a part in predisposing towards appendicitis, the majority of cases occurring in those under thirty years of age. About seventy per cent. of all cases occur in persons under this age. In the very young it is comparatively uncommon, but few cases having been seen in those under five years of age, although cases are on record where the patients were under one year. One case has been reported where the sufferer was seventy years of age.

Sex plays an important part in appendicitis, males being more frequently subject to attacks than females. This is possibly due to the greater exposure of boys and men, and to the fact that women have a larger blood-supply going to the organ, both in actual amount and in proportion to the size of the appendix.

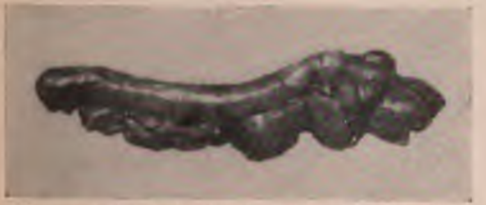
Fatigue, cold, traumatism, typhoid fever, influenza, and so on, have also been noted as predisposing causes.

FIG. 5.



Microscopical examination demonstrated partial occlusion of lumen, beginning at tip of organ.

FIG. 6.

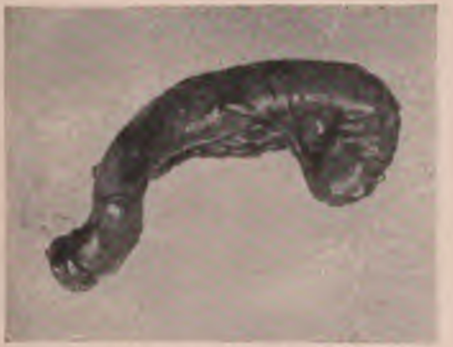


Microscopical examination demonstrated total occlusion of entire lumen.

FIG. 7.

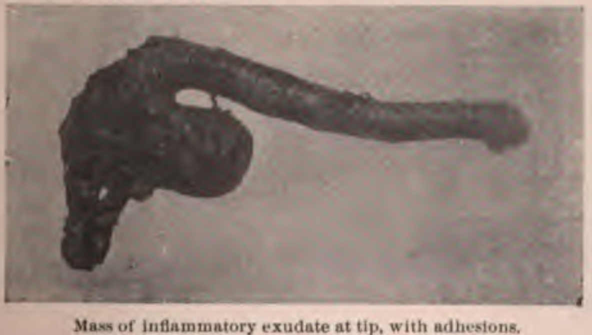
Microscopical examination demonstrated total occlusion of entire lumen, with atrophy.

FIG. 8.



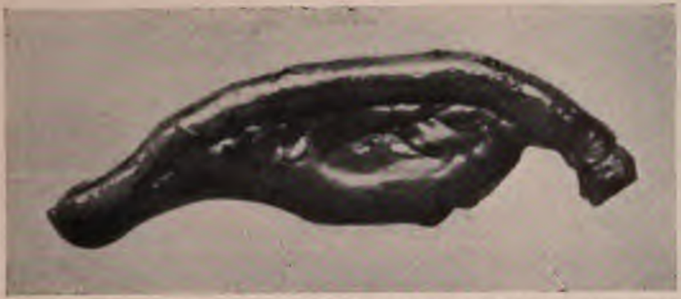
Kink of appendix caused by inflammatory band.

FIG. 9.



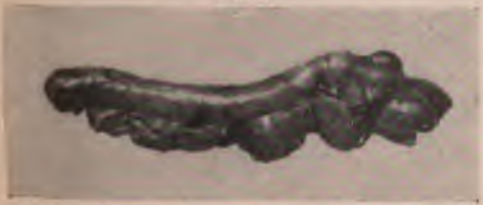
Mass of inflammatory exudate at tip, with adhesions.

Fig. 5.



Microscopical examination demonstrated partial occlusion of lumen, beginning at tip of organ.

Fig. 6.

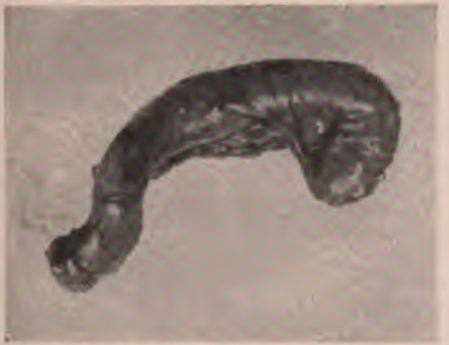


Microscopical examination demonstrated total occlusion of entire lumen.

Fig. 7.

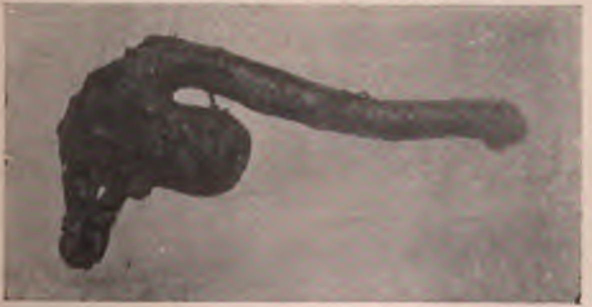
Microscopical examination demonstrated total occlusion of entire lumen, with atrophy.

Fig. 8.



Kink of appendix caused by inflammatory band.

Fig. 9.



Mass of inflammatory exudate at tip, with adhesions.

dicitis is due to the invasion of micro-organisms, brought about by masses of fecal matter, or, rarely, by foreign bodies, causing an erosion of the mucous membrane of the organ. Fecal matter is introduced into the lumen of the appendix by the contractions of the cæcum. The expulsive force of the appendix, which is in a retrograde condition, is not sufficient to expel it, and hence it remains to irritate the mucous membrane, at first merely by its presence. The muscular movements of the organ tend to mould the mass into a round or an oblong shape. This is augmented by the natural secretions of the mucous membrane of the appendix and by further accessions of fecal matter from the cæcum, until it becomes so large that it irritates by pressing against the walls of the organ. Nature attempts to expel the mass by muscular contractions, but, instead of doing so, causes erosion of the mucous membrane, which is followed by the invasion of micro-organisms that are invariably present. The repeated efforts of nature to expel the mass may prove successful, when there will be general subsidence of the more serious symptoms, only to be followed, in a short time, by another attack or by severe pain from pressure of the contracting tissues on the nerve-filaments. It is possible to find appendices in which there is no fecal mass. The exciting cause in these cases is the invasion of the micro-organisms through lymphatic channels, brought about by hyperæmia or deranged circulation of the organ. This condition may be caused by kinks or twists in the mesentery of the organ, by fatigue, cold, traumatism, typhoid fever, influenza, and so on.

Pathology.—But little can be stated positively as to the exact changes that are brought about by an inflammatory process in the vermiform appendix. Various writers have described conditions that were supposed to prevail in appendicitis, the main four stages being described as follows: 1, endo-appendicitis, in which there is more or less inflammation of the mucous membrane and submucosa; 2, parietal appendicitis, in which the inflammation attacks the interstitial or intermuscular tissues of the body of the appendix; 3, peri-appendicitis, in which the inflammation attacks the serous covering of the appendix, being limited by adhesions to that portion of the peritoneum between the appendix and the serous surfaces immediately adjoining; 4, para-appendicitis, in which the inflammation attacks the tissues in relation with the appendix. In the fourth stage we are supposed to find pus-formation, either localized by the limiting adhesions formed during the third stage, or general with involvement of the general peritoneum.

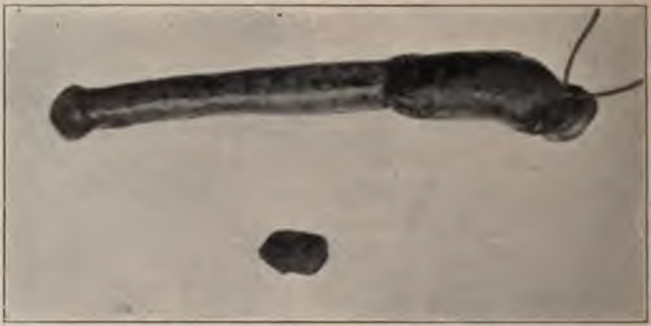
Theoretically, the above division into stages seems to be correct to a marked degree; but when we try to demonstrate these changes in the microscopic specimen, we fail. It is almost impossible to divide the entire process into more than two stages,—viz., one in which the inflammation is confined to the appendix, and another in which the inflammation has attacked the tissues surrounding the organ. A study of over a thousand slides made from diseased appendices at the German Hospital convinces me that the above statement is correct, and that an attempt to recognize the

dicitis is due to the invasion of micro-organisms, brought about by masses of fecal matter, or, rarely, by foreign bodies, causing an erosion of the mucous membrane of the organ. Fecal matter is introduced into the lumen of the appendix by the contractions of the cæcum. The expulsive force of the appendix, which is in a retrograde condition, is not sufficient to expel it, and hence it remains to irritate the mucous membrane, at first merely by its presence. The muscular movements of the organ tend to mould the mass into a round or an oblong shape. This is augmented by the natural secretions of the mucous membrane of the appendix and by further accessions of fecal matter from the cæcum, until it becomes so large that it irritates by pressing against the walls of the organ. Nature attempts to expel the mass by muscular contractions, but, instead of doing so, causes erosion of the mucous membrane, which is followed by the invasion of micro-organisms that are invariably present. The repeated efforts of nature to expel the mass may prove successful, when there will be general subsidence of the more serious symptoms, only to be followed, in a short time, by another attack or by severe pain from pressure of the contracting tissues on the nerve-filaments. It is possible to find appendices in which there is no fecal mass. The exciting cause in these cases is the invasion of the micro-organisms through lymphatic channels, brought about by hyperæmia or deranged circulation of the organ. This condition may be caused by kinks or twists in the mesentery of the organ, by fatigue, cold, traumatism, typhoid fever, influenza, and so on.

Pathology.—But little can be stated positively as to the exact changes that are brought about by an inflammatory process in the vermiform appendix. Various writers have described conditions that were supposed to prevail in appendicitis, the main four stages being described as follows: 1, endo-appendicitis, in which there is more or less inflammation of the mucous membrane and submucosa; 2, parietal appendicitis, in which the inflammation attacks the interstitial or intermuscular tissues of the body of the appendix; 3, peri-appendicitis, in which the inflammation attacks the serous covering of the appendix, being limited by adhesions to that portion of the peritoneum between the appendix and the serous surfaces immediately adjoining; 4, para-appendicitis, in which the inflammation attacks the tissues in relation with the appendix. In the fourth stage we are supposed to find pus-formation, either localized by the limiting adhesions formed during the third stage, or general with involvement of the general peritoneum.

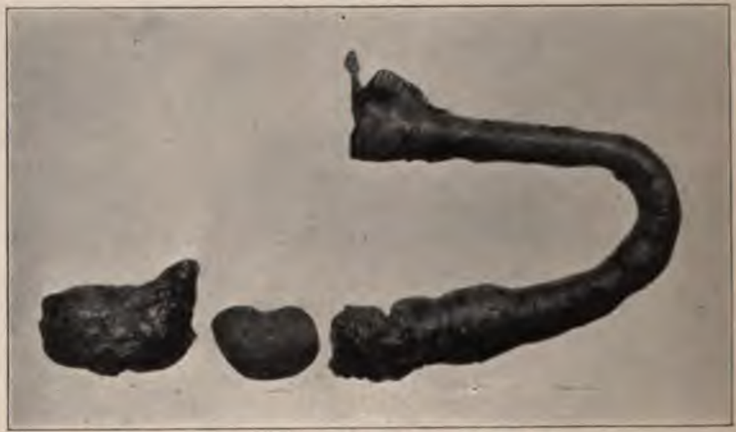
Theoretically, the above division into stages seems to be correct to a marked degree; but when we try to demonstrate these changes in the microscopic specimen, we fail. It is almost impossible to divide the entire process into more than two stages,—viz., one in which the inflammation is confined to the appendix, and another in which the inflammation has attacked the tissues surrounding the organ. A study of over a thousand slides made from diseased appendices at the German Hospital convinces me that the above statement is correct, and that an attempt to recognize the

Fig. 10.



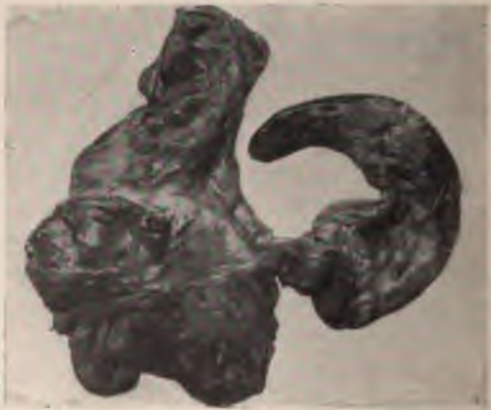
Fecal concretion, with perforation at extreme tip.

Fig. 11.



Fecal concretion, with perforation near tip.

Fig. 12.



Perforation at base of appendix. Post-mortem specimen.

various divisions previously described would be futile. It is next to impossible to formulate any definite changes that we may expect to find in a diseased appendix, for no two organs show exactly the same changes; in fact, we rarely, if ever, find the sections from different portions of the same appendix agreeing in the pathological changes.

Appendicitis is generally due to an invasion of micro-organisms, made possible by destruction of the mucous membrane, as has been described in the preceding section on Exciting Causes. This destruction of the mucous membrane is almost invariably caused by a fecal concretion or foreign body. The fecal matter is forced into the lumen of the appendix by the contractions of the cæcum, where it rapidly increases in size by additional matter from the cæcum and by the deposition of the normal secretion of the mucous layer, together with the débris that is being thrown off continually by the same. Nature endeavors to expel the mass from the organ, but fails on account of the contraction of the organ on the proximal side of the concretion, and because of the weak condition of the muscular force of the appendix. The consequence is that the mucous membrane becomes eroded, and there is immediate invasion of the micro-organisms that are always present in the organ. The increased flow of mucus caused by the irritation may liquefy the fecal matter sufficiently to allow of its expulsion, and we then find the attack terminating favorably, with immediate repair of the destruction following. At other times the expulsive force is not sufficient, and the process then progresses with various degrees of rapidity, either to a favorable termination of the attack, or to perforation, gangrene, and so on.

Since no two specimens give exactly the same changes in the pathological condition, nothing definite can be stated as to what we may expect to find in any case, and nothing but a general statement of the conditions often found can be made. We find the ordinary changes induced by inflammation in some part of the diseased organ, although the extent of these changes cannot be definitely stated. In one appendix we may find the entire mucous membrane eroded and the destructive process extending through the entire thickness of the walls of the organ. Again, the appendix may show normal, healthy mucous membrane with healthy walls in some portions and eroded mucous membrane in others. We may find a constriction of a portion of the lumen, brought about by the deposition of fibrous connective tissue, with ballooning of the distal portion, consequent thinning, and, at times, almost obliteration of the walls. We can generally demonstrate destruction of some portion of the mucous membrane, either at the point of contact with the irritating mass or throughout the entire organ; more or less round-celled infiltration, extending for various depths into the wall of the organ; enlargement of some of the lymph-glands and the crypts of Lieberkühn, with infiltration of their tissues; at times partial destruction of the muscular layers, at other times complete obliteration of all muscular fibres; and congestion of the serous coat with hyperplasia.

The position of fecal concretions in the lumen plays an important part in the outcome of appendicitis. Nature's endeavors to expel foreign or irritating material often result in the concretion being forced to the distal end of the canal, in the direction of least resistance. The stronger resistance to the proximal end of the organ is brought about by efforts at repair at what was probably the point of the first infection. This causes a barrier across the lumen through which the appendix cannot force the fecal matter. The consequence is that the concretion is forced to the distal end. We then have the condition presented of a mass of matter in an appendix which must be removed somehow, and, as it cannot be forced through the opening at the base, it is driven forward and causes perforation at the tip of the organ. Some specimens show this condition perfectly. In these appendices we find either a normal condition of the mucous membrane, with a constriction to the proximal half of the organ, or obliteration of that portion of the lumen, with a fecal concretion confined in the distal half. Unless removed by operative interference, we shall find the appendix perforating at the extreme tip.

The point at which perforation takes place is also of the greatest moment. Ordinarily, the fecal matter works its way through the walls of the appendix at some point along the extent of the organ. In these cases the perforation usually empties into a walled-off cavity, and but little additional damage is the result. In a few cases, however, the perforation takes place at the junction of the appendix with the cæcum, and in these instances the matter is almost invariably discharged into the general abdominal cavity. Why nature allows this to happen no one can say, but that it is so no one can question. The limiting membrane or wall of the pus-cavity seldom shuts off the entire organ, the consequence being that the base of the appendix is without the cavity, and a perforation occurring at that point will almost invariably cause a general peritonitis.

In a few cases we may find that the mucous membrane and even the basement membrane are intact, but that we have a gangrenous portion of the organ. This is brought about by a kink or twist of the appendix which interferes with the blood-supply, the portion of the organ deprived of its arterial supply undergoing a gangrenous process. In a few other cases we find that the invasion of the micro-organisms has taken place through one of the numerous lymph-channels, the inflammatory process beginning within the walls of the organ, while the mucous membrane and basement membrane remain intact. These cases are, however, very rare. Gangrene often supervenes upon an inflammatory process, the blood-supply being interfered with by the infiltrate that nature throws out.

When the inflammatory process extends beyond the serous coat of the organ, we find the ordinary process of inflammation persisting in the structures immediately surrounding the appendix. Nature attempts to defend itself from the invading forces, and throws up a wall or barrier of lymph around the diseased organ. This lymph gradually increases in strength and thickness until we find a firm limiting membrane, which protects the peritoneum completely. With the formation of pus, the abscess is limited in extent to this membrane, and no communication is allowed between the appendix and the general peritoneal cavity unless the appendix, unfortunately, forms part of the limiting wall, when we may find perforation into the general cavity. With the involvement of the structures surrounding the appendix, there is a deposition of lymph which completely covers all the structures affected. The peritoneum that has been exposed to the process becomes greatly congested and inflamed, with a "scalded" look. The pus gradually works its way in the direction of least resistance, and may "point," unless evacuated, in the right iliac fossa, through the abdominal walls, in the groin or in the thigh, or it may open into the cæcum, bladder, rectum, vagina, or pleural cavity. At times the limiting membrane will give way, and we shall find a general purulent peritonitis. The pus will burrow unless evacuated at once, and the larger the abscess and the greater length of time it is allowed to remain, so much the greater will be the consequent damage. The following case is of interest in this connection:

C. B., colored, aged forty-five, had been sick for about two weeks with crampy, colicky pain in abdomen. Appetite very poor, stomach irritable, nausea, but little vomiting. Had had numerous chills and sweats, with decided temperature of a hectic type. Admitted to German Hospital for treatment. Examination revealed a painful, fluctuating mass in the right thigh beneath the fascia lata, also a bulging of the abdominal walls over the right iliac fossa. Pressure and counter-pressure demonstrated a communication between the two masses. Diagnosis of appendiceal abscess made, with extension of the pus along the iliac vessels into the thigh. Abdominal walls incised and large amount of pus evacuated. Counter-opening made in the thigh and drainage-tube introduced. Patient died the next day. Post-mortem examination showed a large abscess-cavity in the right iliac fossa, with free communication along the iliac vessels into the thigh. The appendix was gangrenous and almost entirely disintegrated.

Other changes that may be found in an acute appendicitis include lymphangitis, which may extend to the lymph-channels of the colon and mesentery and thus set up a wide-spread inflammation. The veins are often the seat of thrombi as a result of infection, frequently followed by extensive thrombo-phlebitis, pylo-phlebitis, portal embolism, and abscess of the liver. The following case will be of interest in this connection:

J. K., male, aged thirty-three, railroad engineer, had had an attack of acute appendicitis about six months ago. Present attack began three days prior to admission to the German Hospital.

Examination on admission revealed marked tenderness and rigidity in the right iliac region. No tumescence.

Operation, usual incision. Appendix found pointing southwest and surrounded by omentum. Distal one inch of appendix gangrenous, also the omentum attached to that portion of the organ. Omentum tied off and removed. Appendix cut out of cæcum. Cæcal wound closed with black silk. Abdominal wound closed and dry dressing applied.

Immediately after operation temperature rose to 102° F., pulse 100. Had a chill while coming out of ether. No abdominal distention. Not much pain. Patient continued in this condition for two weeks, with sweats, chills, and sudden rises of temperature to 103° F. Wound examined carefully and found to have healed by first intention. Developed pain and tenderness over the hepatic region. Had loose, tarry stools. Jaundice appeared on the fifth day, and persisted until the patient died. Lost power of deglutition; had several attacks of hemorrhage from the nose. Died sixteen days after operation.

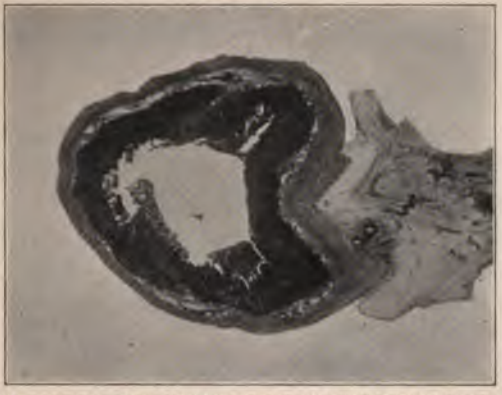
Post-mortem, thoracic organs normal. Large intestine normal. Omentum adherent to wound above and mesentery below, thus enveloping a small pocket of pus. Liver soft and enlarged. Multiple foci of pus on surface. Section showed pus throughout the organ. Portal vein full of thick pus.

Endarteritis of the appendicular artery is seen at times, with resulting patches of necrosis and gangrene, according to the extent of the inflammation in the vessel. The entire appendix has been known to slough on account of deficient blood-supply.

Nature endeavors to repair the injury done the appendix by round-celled infiltration, followed by contraction and deposition of fibrous connective tissue. Although nature is generally proficient in her curative endeavors, the appendix seems to suffer more from these attempts at a cure than it would were it left severely alone. The fibrous connective tissue takes the place of the destroyed tissue of the organ and contracts, causing constriction in the lumen at the points of contraction. At times this contraction begins at the distal extremity and gradually continues until the entire lumen has been eradicated. This is a positive cure as far as any inflammatory condition in the organ is concerned, but, unfortunately for the comfort of the patient, there are numerous small nerve-endings which have been impinged upon, with consequent suffering. The two cases quoted under Etiology will demonstrate this condition. The patient suffers excruciating pain and gives every symptom of a continuous attack of inflammation, with the exception of a rise of temperature. The entire appendix becomes nothing but a whip-cord in a few instances, but the symptoms presented are almost identical with those of an acute attack. Could we diagnose between these two conditions, we could ease the mind of the patient, but the treatment would be the same as far as the trouble is concerned,—removal of the organ.

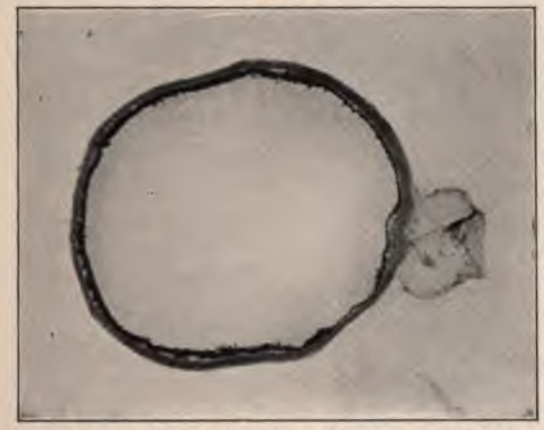
In the majority of cases the constriction does not begin at the distal extremity, but at any portion between the tip and the base of the organ. Sometimes we find two or more constrictions in the same organ, with dilatation of the intervening portions. When the constriction occurs along the course of the lumen, a portion of the organ to the distal side becomes di-

FIG. 13.



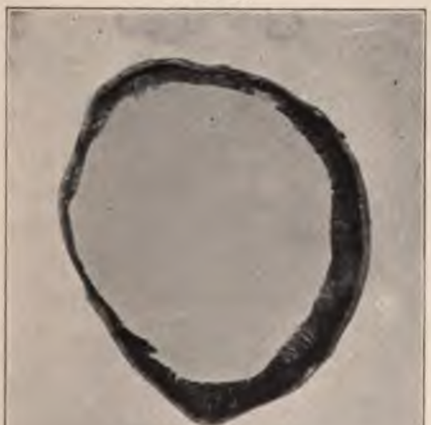
Transverse section showing comparatively healthy mucous membrane.

FIG. 14.



Transverse section showing destruction of mucous coat and marked dilatation. Same appendix as Fig. 13.

FIG. 15.



Transverse section showing dilatation and destruction of mucous coat by fecal concretion.

lated, on account of the débris and secretion thrown down by the mucous membrane. In some of these cases we find a perfectly normal area of mucous membrane, the secretion and débris forming a collection which soon dilates the lumen at that point, or burrows into the walls of the organ, and sets up another attack. The patient suffers attacks of severe colicky pain, which are due to nature's effort to expel the accumulated material through the constricted lumen. This pain gives the impression that the patient is suffering from another acute exacerbation, although there may be no inflammation there whatever. The outcome of these cases is almost always the same,—viz., suffering from pain until another attack sets in, which will require operative interference at once, or terminate in perforation and gangrene with purulent peritonitis, if allowed to take its own course.

The bacteriology of appendicitis has not been given as much attention in the past as the subject demands. In almost every case of appendicitis we find micro-organisms in some part of the lumen or the wall of the organ, and invariably when we have fecal matter in the lumen. A few cases will show a condition in which the mouth of the organ has been shut off by a previous inflammation, with consequent blocking up of secretion in the distal portion of the lumen, and in which we shall be unable to discover a single micro-organism. This is accounted for by the fact that the appendix recovered from the previous attack, with expulsion of the contents of the lumen. Constriction of the lumen in the proximal portion of the organ followed. The healthy mucous membrane to the distal side of the constriction continued secreting and throwing off débris which became dammed up back of the obstruction. This matter is generally absolutely sterile, and causes trouble only by its presence, and by the fact that it thins the walls in time, with consequent perforation.

In every case where we find fecal matter we find micro-organisms in varying numbers. The position in the organ in which these are found can never be stated positively, as they seem to have no special point of greatest intensity. We find them, probably, in greatest numbers in the fecal matter in the lumen; often they are scattered through every portion of the wall, being clustered in the lymph-follicles, in the crypts of Lieberkühn, through the muscular layer, or uniformly through the entire wall.

The most prominent of the micro-organisms are the bacillus coli communis, the staphylococcus pyogenes aureus, the streptococcus pyogenes, and the proteus vulgaris. These are normally found in the intestine and appendix, where they seem to be absolutely harmless as long as they do not invade the walls of the organ. As soon, however, as the mucous membrane is in the slightest degree destroyed, an invasion follows, during which the micro-organisms seem to take renewed life, and then become most virulent.

Symptomatology.—In discussing the symptoms of appendicitis, the disease must be divided into two classes, acute and chronic. Acute appendicitis includes all the steps through which the disease may run, lasting until the acute inflammatory process has abated.

In acute appendicitis in the adult the attention of the physician is almost always brought to bear on the condition of the right iliac fossa by the symptoms pointed out by the patient. In the young this is different, and in many cases the physician discovers the presence of the disease without his attention having been called to that region at all. For this reason the symptoms presented by the young patient are of very little avail to the physician, and nothing but a very careful examination will suggest the main trouble. The young child may be fretful and irritable, with irregular bowels, loss of appetite, some nausea, and slight rise of temperature, all of which may be accounted for by a mild disturbance of the digestive functions. The patient's attendants will generally overlook the real cause of the trouble and consider the disease of little moment. A careful examination of the abdomen, however, will reveal to the medical adviser the inflammatory condition of the appendix. The following history will well illustrate the ease with which a bad case of appendicitis may be overlooked by those in charge of the young.

C. H., aged nine, an inmate of the Southern Home for Destitute Children. For two or three days had had lack of appetite and was fretful. Slight nausea and a moderate rise of temperature were noticed, and the boy was put to bed to rest. The visiting physician saw the patient in bed, but was informed that it was nothing but a slight attack of indigestion. An examination was made, however, and acute appendicitis with pus was diagnosed. Patient was referred to me for operation. Operation revealed a large abscess, the pus being foul and discolored. The appendix was bent on itself and had perforated. Patient made a good recovery.

There have been many more such cases, no doubt, where, because of the apparent slightness of the trouble, or because the patient was unable to express suffering, the true state of affairs was entirely overlooked and the child treated for slight indigestion. No case of even the mildest attack of "colic" or "indigestion" should leave the physician's care until a thorough examination of the abdomen has been made. This will elicit the three main symptoms of appendicitis,—namely, pain, tenderness on pressure, and rigidity of the abdominal walls.

The *pain* of appendicitis in children may not be of as much importance as are the other two main symptoms, as the child may try to hide it, or it may be accounted for by the attendants as the pain of indigestion. When we can obtain a history of pain, we find that it has been sudden in its onset and colicky and paroxysmal in character. This is the *initial pain*, and is not located for some little time. It takes the form of intestinal colic, griping, paroxysmal, but never absent. The starting-point seems to be near the umbilicus, from which point it invades the general peritoneal cavity, then becomes localized over the site of the appendix. The intensity of this initial pain varies in different cases, being almost absent in some, excruciating in others.

After it has become localized, the character of the pain changes to a marked degree. Instead of being colicky and paroxysmal, it becomes much more constant and of a lancinating character, and is then known as the *secondary pain.* The position of the patient has a marked bearing on the intensity of the suffering, the dorsal decubitus with the right knee drawn up being the position in which the pain seems to be the least severe. Coughing, sneezing, deep inspiration, or an attempt to extend the leg increases the pain.

The location of the pain must be borne in mind. It is not essential that it be found in the right iliac fossa, and, in fact, it is seldom that the primary pain is referred to that region. It usually starts around the umbilicus, in the epigastric region, or it may be general. It is found least frequently in the region of the appendix.

The secondary pain, the constant, lancinating pain, may be referred to as many positions as the appendix may hold. Thus, with a long appendix overhanging the pelvis, the pain will be referred to the left side of the median line; if pointing towards the pelvis, the pain may be referred to the pelvis or along the spermatic cord to the testicle; if behind the cæcum, the pain may be referred to the right loin; if it occupy the left iliac fossa, the pain will be found in that region.

Tenderness on pressure is even more constant than is the pain. It is always present as long as any disease of the appendix remains, and is continued in the chronic variety and even in some cases where there has been apparent recovery from the disease, as will be shown later. The ease with which this tenderness can be elicited, and the point of greatest tenderness, depend upon the position of the appendix and, to a certain extent, upon the condition of the organ. With the appendix lying anterior to the cæcum, but little trouble is had in finding the tenderness, even with the slightest amount of pressure; if it lie back of the cæcum, however, it may be necessary to exert great pressure in order to elicit any tenderness at all. Ordinarily the point of greatest tenderness is midway between the umbilicus and the anterior superior spine of the ilium,—McBurney's point.

The severity of the tenderness varies to a certain extent with the condition of the appendix and the degree of inflammation present. We may be able to elicit marked tenderness in an appendix in which the disease has advanced but little, yet this is the exception. During the onset of the disease there may be no tenderness at all, but as soon as the pain becomes localized the tenderness is unmistakable. As the inflammation increases and extends to the peritoneal covering of the organ, the tenderness will increase, and will reach its maximum intensity coincidently with perforation or pus-formation. After an abscess has formed and has been walled off, the tenderness will decrease to a marked degree.

Rigidity of the abdominal walls on the right side goes hand in hand with the tenderness on pressure, as a rule, although we may find a few cases where there will be great tenderness with but little rigidity, and *vice versa.* This can be accounted for in some degree by the mental condition of the patient. A patient who has had severe tenderness on pressure, or who is suffering from severe pain, will naturally stiffen the abdominal muscles as much as possible, generally involuntarily, to protect the diseased part. The impression left by that pain will make the patient contract the muscles whenever there is any danger of approaching that part. Other patients may be able to stand the pain, and, with good control of the nervous system, will allow severe pressure without wincing. The rigidity is almost always, however, an involuntary reflex, and is more decided in those cases where we find marked tenderness on pressure. The point of greatest rigidity varies slightly, but is generally over the right iliac fossa. With the appendix behind the cæcum, the rigidity will be more pronounced in the right groin. When the organ occupies the pelvis, both sides of the abdominal walls may be rigid. A diagnosis placing the appendix in the pelvis can be made with certainty in a majority of the cases where we find bilateral rigidity.

Although pain, tenderness on pressure, and rigidity of the abdominal walls are the most frequent and the most constant symptoms of appendicitis, there are other signs which play a more or less important part.

Vomiting may or may not be present. When present, it generally begins with the onset of the initial pain. In some cases it will cease as soon as the pain becomes localized; in others it will continue and be almost uncontrollable. When the vomiting continues until it becomes stercoraceous, the ejecta are finally regurgitated, and this condition may be taken as an indication of a fatal termination.

Nausea is generally present at the onset of the disease, but may be absent throughout.

The bowels are usually constipated, although frequently we find a diarrhœic condition. One condition may follow the other, especially when the disease progresses unfavorably. After the peritoneum has become invaded, constipation, often very obstinate, prevails.

Temperature.—With the onset of the disease there is almost invariably a slight rise of temperature, to 100° or 101° F. This usually subsides with a free evacuation of the bowels, and then generally remains below 99° F. Coincident with perforation there will be a drop in the temperature, followed by a decided rise as the general peritoneum becomes involved. But little reliance can be put on the temperature as an indication of the presence or absence of pus, as the following case will illustrate:

Mr. D., student, aged twenty, presented himself in the dispensary of the German Hospital for treatment for what he considered an attack of indigestion. Had had two attacks of colic, but relief immediately followed free evacuation of the bowels. Examination revealed marked tenderness in the right iliac fossa, with rigidity of the abdominal walls. Temperature normal, pulse 78; was advised to have appendix removed. Three days later was admitted to the German Hospital. The same symptoms presented themselves. No rise of temperature was noted. The abdomen was opened and a gangrenous appendix with pus was found. Appendix removed and wound drained. Patient made a good recovery. At no time while he was under the care of the hospital did his temperature rise above 98.4° F.

Those who depend on the temperature as a sign of the progress of the case will be likely to err often. Many cases show a drop to normal in the temperature while the disease is increasing in severity. In fact, the physician should be more guarded when there is a fall in temperature, as it often precedes perforation or rupture of an abscess. In some of the most favorable cases where the acute inflammation soon subsides there may be a temperature as high as 104° F.

The face often wears an anxious expression that is almost typical of intra-abdominal affections. The tongue is furred, but moist. In advanced stages, where there has been absorption of pus or where peritonitis has set in, the tongue will become much dryer and at times fissured. Sordes may be present.

Restlessness is generally indicative of pus. As a rule, the patient will lie quietly on the back with the knees drawn up, as there is less pain experienced when quiet; but with the involvement of the peritoneum, or with the presence of pus, there will be decided restlessness.

The urine may show decided changes both in quantity and in character. It is usually high-colored and scanty in amount during the febrile stage. Blood, albumin, and casts may be present. These conditions generally disappear with the subsidence of the inflammation. Frequency of urination is often a prominent symptom in young children. The urine is more scalding and irritating than normally.

Palpation in acute cases is generally of little aid, on account of the rigidity of the abdominal walls. Rarely, if ever, can a tumor be demonstrated during the early stages of the disease. After the formation of pus a distinct swelling or tumescence will be found in the right iliac fossa, in cases where the pus is confined to the fossa. In patients where the appendix occupies the pelvis, a bulging may be found by rectal or vaginal examination, which will be accompanied by great tenderness.

Percussion in the later stages may elicit dulness, especially where there is an abscess. Prior to the pus-formation percussion will reveal but little.

Summary.—The three main symptoms are sudden onset of pain, tenderness on pressure over the site of the appendix, and rigidity of the abdominal walls. Nausea, vomiting, constipation, or diarrhœa may be present or absent. There is always a rise of temperature with the onset of the disease.

CHRONIC APPENDICITIS.

The symptoms of chronic appendicitis differ materially from those of the acute affection. The history is of the greatest importance, as a rule, although many cases are seen where no history of a previous attack can be obtained. Especially is this the case with young children, the former attacks being ascribed to slight disturbances of digestion. If the patient be seen in the acute exacerbation of a chronic inflammation the symptoms will generally coincide with those of acute appendicitis, although they may not be so marked. In these cases the history alone will tell whether we have to deal with a case of acute appendicitis or with an acute exacerbation of a chronic inflammation.

Between the attacks the patient at times feels perfectly well. Again, he will suffer from loss of appetite, disturbance of the digestive functions, nervousness, restlessness, fear of a second attack, pain and tenderness on pressure.

The three main symptoms are the most constant and the most characteristic of chronic inflammation in the appendix, or of the result of inflammation of that organ. A thorough study of them is necessary to diagnose the condition we expect to find, and especially in giving a prognosis as to the ultimate outcome of the trouble.

The pain is the most constant symptom found, and it can be said to exist in all appendices in which there has been inflammation. Naturally it is greater in some cases than in others, but it is always to be noticed from time to time. This pain is not always due to an inflammatory process present at the time the pain is experienced. As was shown under Etiology, there are cases where all the inflammatory conditions have subsided, but in which the pain remains as a result of nature's attempt to bring about a complete recovery. In these cases the pain is almost constant, its intensity depending upon the amount of tissue that has been destroyed and replaced. The granulation tissue and fibrous connective tissue, which are used to replace the eroded and necrosed tissues of the appendix, contract and impinge upon the nerve-endings of the organ, thus setting up the pain. Change of position, irregularity of the bowels, traumatism, or violent exercise will increase the pain, which is more constant in these cases than in those in which the inflammatory conditions persist in some portion of the organ. With an appendix in this condition there will be much less rigidity of the abdominal walls. The organ can be easily palpated, and it is only when it is pressed upon that we find marked tenderness.

In cases where a chronic inflammation persists excruciating pain is experienced at irregular intervals. This is caused by nature attempting to expel the secretions and débris accumulated in a portion of the lumen to the distal side of a constriction through the latter. The pain is intense at these times, but does not last long. The tenderness on pressure is more marked than in the former cases, but not so much so as in the acute exacerbations. The rigidity also is more pronounced. During an acute exacerbation the pain is almost as severe as during the acute attack, with corresponding tenderness and rigidity. The following case will illustrate the condition where we find constant pain with absence of inflammation:

W. T., aged nine years, had had one attack of acute appendicitis, from which he recovered with difficulty. All acute symptoms subsided, with the exception of great pain and tenderness on pressure. Pain was almost constant. Palpation revealed little, percussion nothing. Bowels fairly regular and appetite good. Felt well, with the exception of the pain in the right side. Abdomen opened and cæcum brought into the wound. Some trouble was experienced in locating the appendix, but it was soon found back of the cæcum in the form of a cord-like body. It was removed and abdominal wound closed. Pain ceased soon after operation, and did not return. Examination of the appendix showed that it had undergone what is known as obliterative inflammation. The lumen was entirely occluded, and in no portion of its entire length could any normal tissue be found.

The bowels in chronic appendicitis are generally irregular, mucous diarrhœa alternating with constipation.

Fever is of importance only when of the hectic type.

Prognosis.—The prognosis of acute appendicitis depends almost wholly upon the treatment instituted. With an early operation following an early diagnosis the prognosis is always favorable, the mortality under such circumstances being almost *nil*. Under treatment with non-operative interference the prognosis must always be guarded, even unfavorable. No one can tell what course the disease will pursue, and, while it may terminate favorably, there is just as much likelihood of the inflammatory process continuing until a gangrenous appendix, perforation, abscess-formation, or general purulent peritonitis results. Of one hundred and twelve cases (in children) reported by Holt "there were sixty-two recoveries and fifty deaths, —a mortality of forty-five per cent. General peritonitis was the cause of death in eighty per cent., pyæmia in eight per cent., all of them being protracted cases." Operative interference would have reduced this mortality considerably.

When the case is under the opium treatment the prognosis must be even more guarded, for the opium will mask all the symptoms, ease the pain, and check the restlessness, the patient feeling decidedly better and giving the physician the impression that the disease is under control. Many times the reverse is the case, the inflammatory process spreading with fatal rapidity and showing itself even to the experienced eye only when perforation has taken place or general peritonitis has set in.

Subsidence of pain and even of tenderness must not always be taken as favorable symptoms, nor must a favorable prognosis be given in all of these cases. When the principal three symptoms—pain, tenderness, and rigidity —have subsided to a degree, the prognosis can be much more hopeful, and yet even here we are liable to err, as the following case will illustrate:

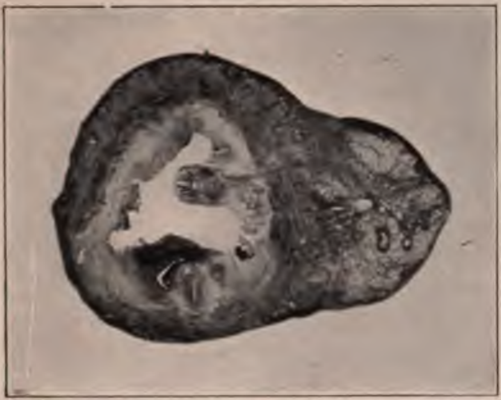
R. L. R., aged twenty-six, male, had been in perfect health for years; was strong and active and had wonderful recuperative powers. Was taken suddenly ill with crampy, colicky, paroxysmal pains, nausea, and some vomiting. Pain soon settled in the right iliac fossa, where there were marked tenderness on pressure and rigidity of the abdominal walls. Diagnosis of acute appendicitis was made. Temperature at the onset of the disease was slightly higher than normal. Under free purgation pain and tenderness subsided. No surgeon was called in consultation. Patient apparently grew steadily better, but still had some pain and tenderness, with rigidity. A most favorable prognosis had been given from the first subsidence of the pain. When apparently recovered, patient was ordered to the shore to recuperate, the physician thinking him well enough to travel. The next morning, prior to departure, patient was suddenly seized with excruciating pain in the region of the appendix, purulent peritonitis of a fulminating character set in, and he died the same day. No post-mortem examination was made, but it is probable that there was perforation of the appendix or rupture of an abscess. A guarded prognosis would have been of inestimable value to the physician in charge.

From our present knowledge of *chronic appendicitis*, the prognosis in that affection must always be guarded. There are a few cases where the appendix is a cause of great suffering to the patient, although not a source of immediate danger. They are the cases where there is total obliteration of the lumen of the organ, with cicatricial impingement on the terminal nerve-filaments. As we are unable to diagnose these cases absolutely, the prognosis in all cases where there has been an attack should be decidedly guarded. Probably eighty-five per cent. of all appendices that have been the seat of an inflammatory process are a source of great danger to the patient. An acute exacerbation is liable to supervene at any moment, thus placing the patient again in jeopardy of his life.

Frequent attacks make the prognosis more unfavorable, not only on account of the trouble in the appendix itself, but also because of the many adhesions that are liable to be formed at each attack. The mortality in cases operated on between attacks is almost *nil*.

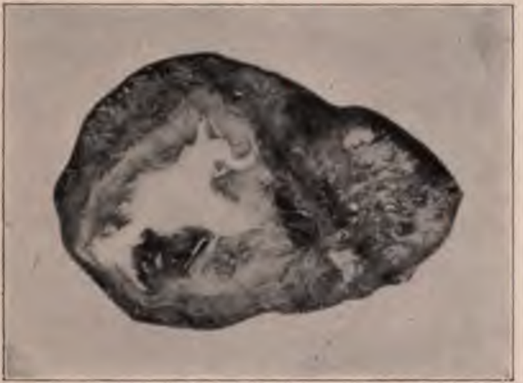
Diagnosis.—The diagnosis of acute appendicitis is rarely a difficult one to make, as we almost always find three symptoms present upon which a positive diagnosis can be made. These are the sudden onset of colicky, paroxysmal pain which becomes localized in the right iliac fossa, tenderness on pressure over the site of the appendix, and either unilateral or bilateral rigidity of the abdominal walls in one previously well. Unfortunately, these symptoms cannot always be recognized at once in children, for the simple reason that they are often masked by an opiate which has been given by the mother or nurse to allay the pain of a supposed mild attack of indigestion. Under these circumstances a positive diagnosis cannot be made until the effect of the opiate has worn off, or until there are unmistakable signs of abscess-formation or of purulent peritonitis. While the colicky pains at the onset of an attack of appendicitis are very similar to those of acute indigestion, the former very soon become localized in the right iliac fossa, and are then associated with tenderness on pressure and rigidity of the abdominal walls, while the latter remain more general and then disappear. Nausea, vomiting, constipation or diarrhœa, restlessness, furred tongue, slight fever, are common to a great many diseases of childhood, and therefore but little reliance can be placed on them in making a

Fig. 16.



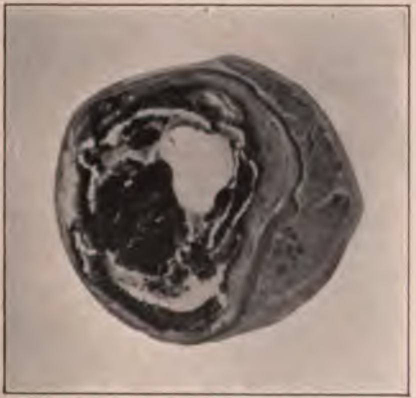
Transverse section showing almost total destruction of mucous coat, with round-celled infiltration.

Fig. 17.



Transverse section showing total destruction of mucous coat, with erosion of submucous and muscular coats.

Fig 18.



Transverse section showing erosion of mucous coat by fecal concretion.

diagnosis of appendicitis. Especially is this so on account of the fact that one or all of them may be present or absent in acute appendicitis.

Some difficulty may be experienced in diagnosing appendicitis from intussusception. In the latter affection we find intense pain, colic, vomiting, tenesmus, and bloody stools. There is but a slight rise in temperature, if any. A tumor can almost always be found to the left of the median line or through the rectum. The trouble is very rare, except in the very young.

In cases of internal strangulation there is no demonstrable tumor. Vomiting is constant, becoming fecal in a short time unless the strangulation be relieved. There is obstinate constipation, and no flatus can be expelled. There is no localized pain nor tenderness with rigidity. The temperature is normal or subnormal.

The diarrhœas of children may be differentiated from appendicitis by the absence of localized symptoms in the former. Appendicitis may be accompanied by bloody stools, tenesmus, and diarrhœa, all of which may tend to a mistaken diagnosis. This can readily be cleared up by a local examination.

In typhoid fever we often find pain and gurgling in the right iliac fossa, with general disturbance of the digestive function. The prodromal period of typhoid, the general lassitude, the epistaxis, the headache, the temperature, and, finally, the typhoid spots, will present a different picture from that of acute appendicitis. In the later stages of appendicitis, where there has been general septic absorption, the diagnosis may be more difficult to make. Spots may be present in both, both may run a similar course of fever, the spleen will be enlarged in both, and lassitude, weakness, and disturbance of the digestive system will be similar. In these cases a careful study of the history of the case, with a thorough local examination, will generally suffice to remove all doubts.

Inflammation in an appendix holding a post-cæcal position, when associated with pus-formation, may be considered as a perinephritic abscess. In the latter affection there will be less marked digestive symptoms, there will be some decided change in the urine, the pain and tenderness will be found deep in the posterior part of the loin, and the abdominal walls will be flaccid instead of rigid.

In abscess of the abdominal walls the collection will be circumscribed if in the superficial fascia, diffuse if between the muscular layers. The swelling will move with the abdominal walls, and there will be local and constitutional evidence of pus, with absence of intestinal disturbance. These symptoms, with the history of the case, will render a diagnosis between an abscess and appendicitis comparatively easy.

In hip-joint disease we find a characteristic deformity, impairment of the normal movements of the joint, arching of the lumbar spine when the affected limb is extended, and pain referred to the knee. There is absence of intestinal disturbance. The history of the case will point to a disease slower in its course than appendicitis. In cases where there [illegible] fficulty in diagnosing between it and appendicitis, anæsthetization has been recommended, under which the true condition of the joint can be determined.

A psoas abscess may be confounded with the later stages of appendicitis where there has been pus-formation in the latter affection. In psoitis there is much less pain, with but little, if any, rise in temperature; fluctuation is more pronounced and more general. There will be absence of sudden onset, of intestinal disturbance, and of peritoneal symptoms. These symptoms, with a careful examination of the spine and iliac bones, should always clear up any doubt in the diagnosis.

In lumbar abscess the slow onset, and the absence of acute pain, of rigidity, and of intestinal disturbance, will preclude a diagnosis of appendicitis. It is well to remember that acute or subacute suppuration in the right iliac fossa is almost always due to appendicitis.

Treatment.—Appendicitis, whether acute or chronic, *is essentially a surgical affection*, and should be placed at once under the care of a skilful surgeon. The truth of this statement is becoming recognized in direct proportion to the general knowledge of the course and uncertainties of the disease, and at the present time only those who have but a limited idea of the course of the affection and have seen but a few cases attempt to treat appendicitis without the advice of a surgeon.

Provided a diagnosis shall have been made before general purulent peritonitis and collapse have set in, the only thorough treatment of appendicitis is the removal of the organ. Operation is the only procedure by which we can be certain of curing our patient. It is true that some cases do recover from an *attack* of appendicitis without an operation, but the percentage of those that recover from the *disease* is almost *nil*. After an attack, the patient possesses, in almost all cases, an appendix that is the seat of a subacute or chronic inflammation, with constrictions in its lumen, thickened walls, necrosed patches of mucous membrane, adhesions binding it to adjacent structures, encapsulated pus; in short, it is an organ that is diseased, and in which a treacherous inflammation is likely to spring up at any moment that may not only cause great suffering and distress to the patient, but place his very life in jeopardy. The cases where this condition does not persist are so small numerically, when compared with those in which it does, that they are practically not to be considered.

The main reason, however, why the appendix should be removed as soon as possible is that no one can state positively what course the disease is taking. All symptoms may subside to a marked degree, and yet the disease may be progressing rapidly towards gangrene, perforation, general peritonitis, or what not. Could we point with certainty to this case or that, and say positively that it would recover from an attack, or that it would go on to fatal results without operative interference, there would be but little trouble in dealing with the disease. Such is not the case, however. While the appendix remains, there is a lurking disease present which may spring forth without warning and cost the patient his life. The possessor of a once inflamed appendix should be given the benefit of the doubt as to the course the disease may pursue, and be relieved of the seat of the affection,—the appendix.

But there are some cases where this is impracticable on account of an objection to operation, because of some underlying contra-indication to anæsthetization, such as Bright's disease, diabetes, advanced tuberculosis, and so on. Under such circumstances the only alternative is the expectant treatment. This consists of rest in bed, the proper administration of laxatives, the alleviation of the pain, and a regulated diet.

With the onset of the disease, the patient should be put to bed, and kept there until all inflammation has subsided. The dorsal decubitus, with the legs drawn up, will generally be chosen by the patient, as the pain is less severe in this position. It is always well to place a large, firm pillow under the knees, in order to assure as much ease to the patient as possible.

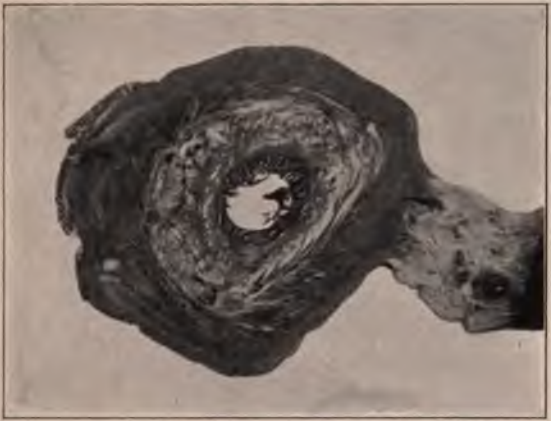
Laxatives should be administered at once. The most preferable is castor oil, because it will empty the bowel of all its contents and will not drain the intestinal circulation of its serum. Where there is pronounced nausea with vomiting, or where the stomach will not retain the castor oil, calomel should be administered, preferably in the powder form, with a little bicarbonate of sodium to hasten its action. It is often advisable to give the calomel from the beginning, on account of its soothing effect on the stomach. Salines should be given when depletion of the intestinal circulation is required. The main objection to salines is that they will cause a free watery evacuation, but will allow solid particles to remain in the bowel, a defect that is not noticed when castor oil is administered.

I believe that laxatives should be given in the beginning of every attack of appendicitis. Diarrhœa does not act as a contra-indication, as this condition generally calls for laxatives as urgently as does the opposite one of constipation. I give a laxative with a threefold purpose: to relieve pain by clearing the intestinal tract of all irritating materials; to diminish the virulence of the attack, as I believe the presence of foreign or irritating material in the intestinal tract, and especially in that portion adjacent to the cæcum and appendix, has a favorable influence upon the development of the invading micro-organisms; and to set up an active peristalsis in the intestine and appendix, and so aid the latter in ridding itself of any abnormal material. The administration of laxatives is contrary to the teachings of many eminent surgeons, but my experience justifies me in the statement that it is the only successful form of treatment when operation cannot be performed, and also the only one that will aid in checking the disease and at the same time prepare the intestinal tract for operative interference, if operation be decided upon. If given early, before nature has matted together coils of intestine with adhesions for the purpose of protecting the general peritoneal cavity, they cannot be other than decidedly helpful. Even after some adhesions have been formed, the waves of peristalsis cause much less danger to the patient than would a full bowel. The danger of breaking up forming adhesions is more than counterbalanced by the good derived from a free evacuation of the bowels. In the later stages of the disease, however, after the barrier between the pus-bearing appendix or an abscess and the general peritoneal cavity has been formed, active purgation is not advisable, as peristaltic contractions may destroy the only safeguard which prevents a purulent peritonitis. Mild purgation at this time is always advisable.

The alleviation of the pain has always been one of the greatest bugbears to the attending physician or surgeon. The patient in the throes of an acute attack of appendicitis does suffer, undoubtedly, and the natural instinct of the attendant is to make his patient as comfortable and as free from pain as possible. Hypodermics of morphine or large doses of opium are given, and the patient blesses the doctor, being free from his suffering at once. Opium does relieve the pain, but it also binds up all the secretions, checks the bowels, masks the symptoms, every guide by which the physician is led to a proper knowledge of the course the disease is pursuing. It also causes retention of gases in the intestinal tract, with consequent distention, and increases the nausea. But the worst objection to the use of any form of opium is the masking of the symptoms. Many a mistaken diagnosis has been made, untoward symptoms have developed without the knowledge of the attendant, and many times a surgeon has been called too late. Pain is nature's great and most reliable signal of distress; it is her means of telling us that some part of the human organism is in need of prompt attention. In no class of cases is this signal of as much importance to nature's assistants as in intra-abdominal affections, and especially when such a treacherous disease as appendicitis is under consideration. The patient is relieved from pain while under the influence of opium, and the impression is given that the disease is abating. Such may be the case, but more often the patient who is now lying quietly will in a short time be writhing in agonies produced by perforation or purulent peritonitis. The surgeon is then called, and he finds the abdomen distended, whether from the effects of the opium or from peritonitis he cannot state; the patient is still drowsy from the opium and cannot answer questions intelligently; the tenderness on pressure, and consequently much of the marked rigidity of the abdominal walls, have disappeared, whether on account of the abeyance of the disease or from the effect of the drug no one can tell. Thus all symptoms are masked, hidden from the consultant, and the best he can do is to guess at the condition within the abdominal cavity and act according to the surmise, or give a very guarded prognosis and await the subsidence of the narcosis. Had laxatives been given in place of opium, the pain would have been alleviated somewhat, nature would have been free to signal distress if need be, and the surgeon would have known at once the true state of affairs. If pain increases after purgation, operation is indicated, as perforation is about to take place or pus has formed.

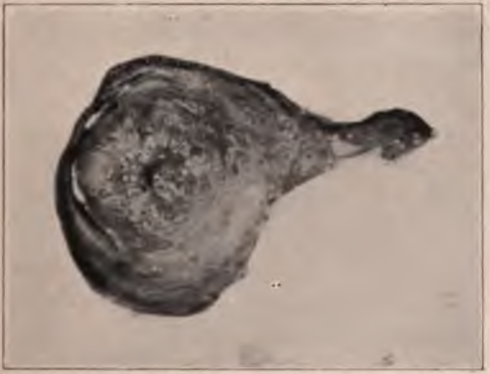
With the localization of the pain over the appendix an ice-bag should

Fig. 19.



Transverse section showing contraction of lumen due to pressure of fibrous connective tissue in submucous layer.

Fig. 20.



Transverse section showing almost total occlusion of lumen by fibrous connective tissue.

be applied to the abdominal walls over the seat of the greatest tenderness. This is often objectionable to the patient at first, but he will soon become accustomed to the cold, and the alleviation of the pain will induce him to keep it in place. Often the severest pain of an acute appendicitis will be entirely relieved in a short time by the application of an ice-bag. Again, heat may be more acceptable to the patient, and turpentine stupes may be applied in these cases. Dry cold is always preferable, however, as it acts as a local anæsthetic, modifies the degree of inflammation, and hastens resolution.

Blisters, leeches, the application of the tincture of iodine, are all contra-indicated, because they do no good, and also because they add to the discomfort of the patient.

Enemata should be given in conjunction with laxatives, but never to the exclusion of the latter. In cases of constipation consequent upon a paralytic condition of the intestine, forced enemata should never be given, as this condition often indicates perforation of the appendix and the enema might be forced into the general peritoneal cavity, as the following case will illustrate:

Mr. B., aged twenty-seven, was seized suddenly with cramp-like pain in the abdomen, accompanied by nausea and vomiting. Morphine, gr. ¼ (0.015), was administered by his physician. All symptoms immediately ceased. Twelve hours later pain reappeared, localized in the right iliac fossa, with tenderness on deep palpation. Temperature, 100°–101° F.; pulse, 95. No apparent change in his condition for about ten days, when obstinate constipation associated with tympanites set in. Purgatives were administered without effect. A forced enema was then given, but little of it returning from the bowel. Obstruction diagnosed.

Upon examination I found excessive distention, extreme tenderness of the abdominal walls, temperature 102° F., and a rapid, running pulse. Palpation impossible because of the distention; rectal examination *nil.* Diagnosis of appendicitis with peritonitis consequent upon perforation was made from the history of the case.

Operation by incision through the right semilunar line. Peritoneal cavity filled with pus, fæces, and fluid. Cæcum lifted up, when it was found that the appendix had either sloughed off or had been forced off by the high enema, leaving a hole in the posterior part of the cæcum. The opening in the cæcum was closed, the peritoneal cavity drained. Patient never reacted from the operation.

At times asafetida suppositories will relieve the pain by emptying the bowel of gas; belladonna suppositories, gr. ¼ (0.015), will relieve the paroxysms.

The diet should consist of those foods which leave as little residue in the intestinal tract as possible. Broths, beaten eggs, peptonized milk, whey, buttermilk, or strained rice and gruels may be given. In chronic appendicitis, where operation will not be allowed, or where it is contra-indicated forate at any time either into the general peritoneal cavity or into the pus-cavity, resulting in purulent peritonitis in the one case and in fecal fistula in the other. The cases are very few where the appendix cannot be removed, and I believe that it should be, whenever possible, even if we have to invade the limiting membrane of the pus-cavity to find it. The danger of leaving the appendix in the abdominal cavity is much greater, in my judgment, than the danger attendant upon its removal, in the majority of cases. The proper distribution of gauze to prevent infection of the peritoneum, with careful attention to every detail of technique, will lessen the danger of removing the organ to such an extent that I do not hesitate to remove it whenever possible. The only exceptions to be made to this rule are found in cases, especially children, where the constitutional condition of the patient would not warrant a protracted anæsthetization. In those cases I always advise liberation of the pus, to be followed by removal of the appendix as soon as the patient has sufficiently recovered to permit of a secondary operation.

Although a strong advocate of the removal of the appendix in almost every case of inflammation of that organ, yet there are a few conditions under which I prefer to delay operation. When we find a patient with persistent vomiting, a leaky skin, a rapid, running pulse, a diffuse peritonitis, and signs of collapse, I believe that operative interference is contra-indicated. Under these conditions an operation would invariably be followed by loss of life. Ice to the abdomen, calomel pushed to free purgation, a small fly-blister below the ensiform cartilage, nutritious enemata, with stimulants in the form of whiskey or champagne, and hypodermics of strychnine, give a more hopeful prospect than would operation. When the peritonitis has subsided and the constitutional condition warrants, operation may be performed with a much better prognosis.

Operation.—When possible to carry out all the precautions necessary to insure an aseptic field of operation, this should be done. In chronic cases, where the operation is performed between the attacks, this can be accomplished, but many cases have to be operated on at once, and thus no time is afforded for the preliminary treatment of the field of operation. The abdomen should always be washed with soap and water, ether, or alcohol, and an antiseptic solution. When time allows, the patient should be given a bath with soap and hot water, followed by rinsing with boracic acid solution. The abdomen should then be thoroughly washed with thymol or green soap, particular attention being paid to the umbilicus. The entire abdomen should be shaved, to remove any hairs or loose, dead epidermis. Soap and water should follow the shaving, to be followed in turn by washing with ether, turpentine, and alcohol, to remove any fatty material that may remain. The parts should then be washed with a corrosive sublimate solution, 1 to 1000. A sterile towel, or a large piece of gauze wet with sublimate solution, should be placed over the part and allowed to remain until the time of operation. After anæsthetization the e access to the abscess. Further division necessary to locate e. Large fecal concretion found, and large hole in cæcum. Latter nd abscess-cavity packed. Patient made a good recovery.

G., male, aged seven years, taken sick November 2. Complained vere abdominal pains, had fever and repeated attacks of vomiting. On ssion, two days later, abdomen was tense and very tender to the touch. tinct mass on right side. Temperature, 101° F. Appendix behind the cum, shaped like a letter S; much pus in pelvis, offensive in odor. Apndix removed; cavity cleaned and packed with gauze. Made slow covery.

In chronic appendicitis, where the inflammation has subsided into a subacute or chronic state, with exacerbations, the appendix should be removed between the attacks,—that is, during the subsidence of the inflammation. If we could tell positively those very few cases which possess an entirely occluded lumen after an attack of appendicitis, in which there is decided suffering but no inflammation, we might be able to overcome the pain without operative interference. But this cannot be done at present, and, rather than run the risk of leaving a thoroughly diseased organ within the abdominal cavity, all appendices that have once been the seat of inflammation should be removed. It must be borne in mind that every attack places the patient in jeopardy of his life, and that the adhesions formed at these attacks render the removal of the organ more difficult and add to the gravity of the operation. The mortality following operations in chronic appendicitis is practically *nil*. The result of leaving an appendix that has been the seat of inflammation in the abdominal cavity is such that the patient is generally a chronic invalid, with indigestion, constipation or diarrhœa, and mental anxiety on account of the fear of a subsequent attack; he has to take the greatest care of his diet, and is deprived of those things which may be of the greatest relish to him; he is liable to suffer from an acute exacerbation at any time, without a moment's warning, that will imperil his life.

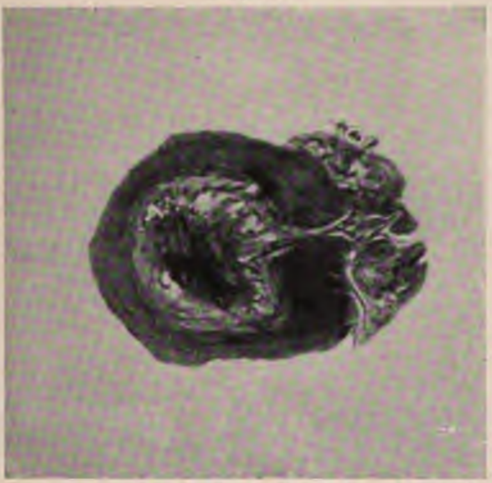
In cases where pus has formed it should be liberated without delay. A few days' delay might make the limiting membrane stronger, but of this we cannot be sure. There is always a liability, even a probability, that the limiting membrane will rupture spontaneously and allow the peritoneal cavity to be flooded with a purulent mass; the appendix may slough away from the cæcum, or be the seat of perforation; septic infection may take place with consequent metastatic abscesses, pyelo-phlebitis, and abscess of the liver. At times the appendix will constitute part of the limiting membrane, and will become harder to remove as the adhesions become firmer and more solid, if the process of protection is not interrupted by some untoward occurrence. The pus should be liberated at once, and, when possible, the appendix should be removed. If the appendix be left *in situ*, the seat of the trouble will still remain within the abdominal cavity, and the patient will be under the ban of a diseased organ. This is liable to per-

In cases that are free from pus the caput coli should be drawn into the wound and the appendix located. This can easily be done by following the anterior longitudinal band to the end of the cæcum, at which point the appendix always arises. Pieces of gauze should be placed in the wound, around the cæcum, in order to preclude the possibility of infecting the peritoneum. One of two procedures should be followed in removal of the appendix from the cæcum. In the first, a circular incision should be made around the base of the organ, through the serous covering, about one-eighth of an inch from its junction with the cæcum. The serous coat should then be rolled up in the form of a cuff. A ligature should be thrown around the appendix close to the cæcum, and firmly tied. The appendix is then to be removed, and the end of the stump touched with a piece of gauze wet with sublimate solution. The cuff should then be rolled over the stump and sutured in place. The entire stump should be invaginated into the wall of the cæcum, fine silk and the Lembert suture being used to draw the cæcum over it. In the second procedure, an elliptical incision should be made around the base of the appendix and through the entire thickness of the walls of the cæcum. The entire appendix is thus removed, and the consequent opening in the cæcum can be readily closed with fine silk and the Lembert suture. This is the more thorough course to pursue, as it removes every particle of the affected organ. It is also much easier, and requires less time in its execution than does the stump and cuff method.

After the appendix has been removed, the cæcum should be returned to the abdominal cavity, being prevented from bulging into the wound by a large piece of gauze placed between the wound and the bowel. In closing the simple incision, the edges of the wound may be approximated and held in place either by simple sutures of silkworm gut introduced through all the tissues, including the skin, fascia, muscles, aponeurosis, and peritoneum, or a buried quilted suture may be used. All sutures should be introduced before the remaining piece of gauze shall have been removed. After this has been taken out of the abdominal cavity, the sutures are to be fastened and an aseptic dressing applied.

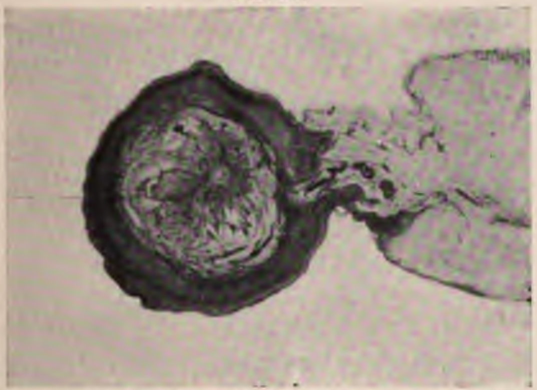
In closing the wound made in the McBurney operation, the several layers should be sutured separately. The peritoneum should be closed with a continuous catgut suture. The other layers in turn should be united with continuous sutures of either fine silk or catgut. The wound in the skin can be closed with a longitudinal buried suture, or with interrupted sutures. This form of operation will give practically four wounds in the abdominal walls, all of which are opposed to each other, and none of which extends through the entire thickness of the parietes. All superficial stitches can be removed in ten or twelve days. An abdominal binder should then be applied, and the patient allowed to sit up. Two weeks is generally sufficient for a perfect recovery from an operation in a case that has been free from pus.

FIG. 21.



Transverse section showing total occlusion of lumen by fibrous connective tissue.

FIG. 22.



Transverse section showing total occlusion of lumen by fibrous connective tissue.

THE SURGICAL TREATMENT OF CONGENITAL ANO-RECTAL IMPERFORATION.

BY RUDOLPH MATAS, M.D.

THE surgical treatment of the congenital deformities of the ano-rectal region which interfere with the partial or complete evacuation of the intestinal contents embraces a great number of procedures. The surgeons who have proposed them have always held two essential indications in view: first, to give immediate relief to the intestinal tension by removing the obstruction, and thus establish the fecal circulation; and, second, to establish an anal outlet that would as nearly as possible imitate the functions of the natural orifice.

Success in accomplishing this purpose must be measured by the fulfilment of certain conditions. The new anus must give easy and painless exit to the fæces whatever their consistence may be. It should be controllable, —*i.e.*, able to close hermetically and voluntarily in the intervals between the acts of defecation. It must be placed in the most favorable situation, considering that for this purpose there can be no better site than that which was originally selected by nature herself. Finally, all these conditions must be permanent and not liable to retrogressive changes in the lapse of time.

The congenital malformations which call for the restoration or the creation of the anal outlet are the subject of numerous classifications, of which Bodenhamer's division into nine species (as stated in vol. iii. p. 322 of this Cyclopædia) is still the most popular and practical. From the clinical and operative point of view, the entire group of ano-rectal defects may be divided into two great classes. The first comprises all the congenital malformations of the anus, including in this category abnormal narrowing or stenosis, occlusion by membranes of varying degrees of density, partial or complete absence of the anus, and other abnormalities of configuration. The second includes the malformations of the rectum: (1) in a cul-de-sac at a variable distance from the perineum; (2) its termination in fistulous or other abnormal opening into the neighboring pelvic organs or channels, such as the bladder, the vagina, or the urethra, the normal anus being present or absent; (3) by more or less imperfect and always anomalous terminations in the skin of the genital or perineal region, the normal anus being absent or imperfectly developed; (4) by a cloaca in common with the vagina; (5) finally, the total absence of the rectum.

It is my object to consider in this article mainly the surgical treatment of the obstructive or atresic malformations of the ano-rectal outlet, in which there is no avenue whatever (not even a fistulous communication with another viscus) for the escape of the intestinal contents, and in which prompt action is required to save the life of the new-born infant.

TOTAL IMPERFORATION OF THE ANO-RECTAL TRACT.

Under this heading shall be grouped all those cases in which the anus and rectum have failed to meet through defective development of either the proctodæum or the enteron, and in which the terminal pouch of the colon or rectum is at a greater or less distance from the perineum. The anus may be well formed or rudimentary, or it may be totally absent; but in either case it does not communicate with the upper intestinal tract. The variations in the relative distance of the rectal pouch from the perineum may measure from a few millimetres to five or more centimetres, or the enteron may be entirely absent and remain out of the pelvis altogether.

An important fact to remember in this connection is that in the vast majority of the recorded cases of ano-rectal deformity the rectal end of the colon or enteron exists and occupies some portion of the pelvis.

The cases in which the rectum is totally absent or is totally inaccessible through a perineo-sacral opening are relatively few. Bodenhamer, whose erudite and laborious researches have made him *facile princeps* among the systematic writers on the subject, collected a total of four hundred and sixty-five cases which he had gathered from all sources up to 1879. Out of this total only Species VIII. and IX. of his classification, in which the rectum and colon are totally absent, furnished the comparatively small contingent of forty-one observations.[1] It is immaterial for our present purpose what these particular variations are. Suffice it to say that we must deal with the group generically, as the differentiation of the species is most frequently impossible at the bedside. The surgeon has but one course open before him, and that is to presume that there is a rectal pouch in the pelvis, and that it is his duty to find it and place it as near the natural outlet as possible or in the region which will most nearly approach the situation of the anal orifice.

CONSIDERATIONS INTRODUCTORY TO THE OPERATIVE TREATMENT OF COMPLETE ANO-RECTAL ATRESIA.

Whenever the diagnosis of total imperforation has been made, it is the duty of the medical attendant to create an outlet for the intestinal contents, and this must be done as quickly as possible. No more delay is permissible or justifiable than the time required in adequately preparing for a aseptic operation.

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[1] *Vide* New York Medical Journal, May 25, 1889, vol. xlix.

The direct causes of death in imperforate infants are stercoræmia, peritonitis, paralysis of the bowel with excessive tympanites, and possible exhaustion from stercoral stasis, which, in addition to the preceding conditions, favors the absorption of toxins and the migration of intestinal micro-organisms. The meconium is sterile at birth (Welch), but it becomes rapidly contaminated after extra-uterine life has begun. The influence of these causes in determining the results of operations is directly proportional to the length of time that has elapsed since birth; hence the emphasis laid by all modern authorities upon operating early. The resistance of the imperforate infant to traumatism is inversely proportional to the time that has elapsed since birth. The longer operative interference is delayed the less will be the resistance to shock. The rapidly increasing experience gathered in recent years in the latest methods of operative relief by sacrectomy or sacrotomy, intra-peritoneal exploration, and the combined sacro-abdominal operations demonstrates that the new-born infant, when free from sepsis, compares most favorably with the adult in the capacity to resist traumatism. Modern experience, therefore, emphatically confirms the dicta of Bodenhamer (1860) and Giraldès (1865), who were foremost among the masters of the past generation to repudiate the tradition that the new-born infant is incapable of resisting severe traumatism. It is not so much to shock as it is to sepsis that the new-born infant is particularly susceptible.

There is no external sign or physical criterion by which we may determine the actual anatomical conditions before operation in the majority of cases. The presence or absence of the anus, the coexistence of pelvic (osseous) deformities, the evidence of fistulous communications with the genito-urinary tract, are all suggestive but unreliable indications of the anatomical situation and relations of the rectal ampulla.

Neither can we depend upon the introduction of guides in the bladder or vagina to determine the presence or absence of the enteron, or to indicate its relative position in the hollow of the sacrum. The use of the trocar or exploring aspirating needle is also fallacious, often positively dangerous, owing to the liability to peritoneal contamination. The cases related by Guersant, Edwards, Curling, Giraldès, Cripps, Jacobi, Wharton, Anders, Czerny, and others are sufficient to convince any one of the dangers of such methods of exploration.

On the other hand, the introduction of guides into the bladder and gina is almost a necessary preliminary in all cases, in order to facilitate e recognition of these parts and to prevent injury to them in subsequent nipulations. The only positive method of establishing the diagnosis of the anatomical condition is by actual operation, aided by digital exploration he pelvic contents. How to accomplish this now engages our attention. The various procedures that have been suggested with the view of overng intestinal obstruction caused by congenital ano-rectal imperforation, according to the anatomical conditions that have been encountered, may be classified as follows:

A. Lower or infra-pelvic operations.
    1. Involving the soft parts only.
  I. Puncture or incision, with dilatation of the membranous septa or diaphragm which separate the anal cul-de-sac from the distended enteron above.
    2. Typical perineal proctoplasty (Amussat, 1835), with or without excision of the anal cul-de-sac, when this exists.
  II. Perineal methods involving the pelvic skeleton, in order of severity.
    1. Coccygeal displacement backward (Delens, 1874).
    2. Median coccygotomy (Polaillon, 1875).
    3. Median sacro-coccygotomy (Vincent, 1887).
    4. Parasacral incision (Vincent, 1887).
    5. Single osteoplastic flap, including sacrum and coccyx (Roux, 1889, Rydygier, Rhen, *et al.*).
    6. Median sacro-coccygotomy with bilateral osteoplastic flaps (Heinecke, 1887, Kocher, Jeannel, Borelius, Morestin, Sieur).
  III. In all of these procedures the peritoneum may be purposely and freely opened for exploratory purposes (Stromeyer's suggestion).
B. Upper or anterior abdominal methods.
    1. Primary median or lateral exploratory cœliotomy, to recognize anatomical position of rectal ampulla and detach it from the peritoneum or mesentery, so as to create a perineal anus, without colostomy (Delagenière, 1894).
    2. Secondary median or lateral exploratory cœliotomy, with same object, after failure of previous perineal exploration (McLeod, Chalot).
    3. Secondary inguinal colostomy (Littre), after failure of perineal exploration, with immediate attempt to restore continuity of rectal outlet to anus, after drainage of bowel, the rectal end being guided to the perineum by a probe introduced through colostomy orifice (Chaput, Stephen Paget, *et al.*).
    4. Primary colostomy without perineal exploration, with secondary attempt at restoration of continuity of bowel to perineal anus at another sitting (Martin, Chassaignac, Lannelongue, McCormac).
    5. Inguinal colostomy with permanent artificial anus (Littre) in left iliac region, or cæcotomy (Pillore) when the sigmoid flexure and colon are missing.
    6. Enterostomy (Nélaton) when condition of patient is too critical to risk further exploration for rectum or colon; this can be applied in sacral as well as in inguinal regions.

FIG. 1.

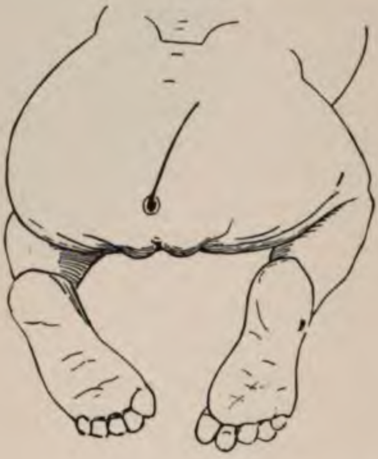


FIG. 2.

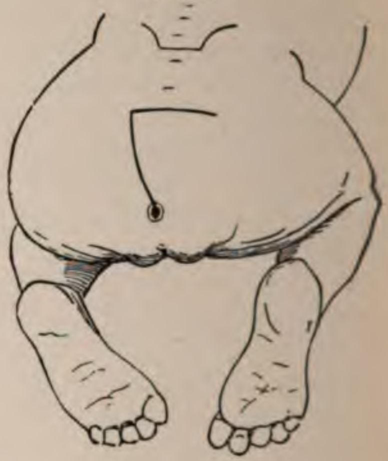
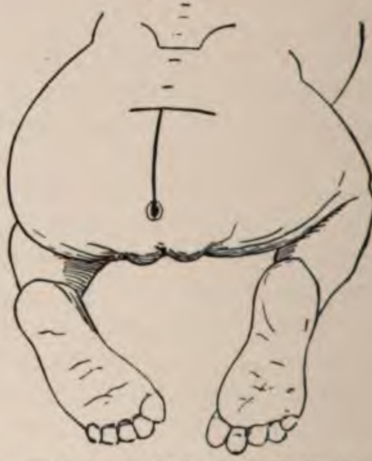


FIG. 3.



Three typical incisions in the sacral region which are applicable to cases of congenital ano-sa[illegible] imperforation.

FIG. 1.—*Parasacral Type* (Vincent, Zuckerkandl, Wölfler). This can be readily transformed b[illegible] a transverse section into No. 2.

FIG. 2.—*Single Solid Osteo-Tegumentary Flap* (Billroth, Rhen, Rydygier, Roux). When mobilized, [illegible] flap is turned over to the right. It may involve complete transverse section of the cartilaginous sa[illegible] on a level with the third sacral foramen, or it may be utilized for lateral sacrectomy.

FIG. 3.—*Median Coccygeo-Sacrotomy* (method of election). Simple median section of coccyx [illegible] sacrum may be sufficient, with retraction, for exploratory purposes. One or two lateral osteopl[illegible] flaps may be readily mobilized, if required, by the addition of an upper transverse section on a [illegible] with the lower border of the third sacral foramen. The same median incision may be utilized f[illegible] a typical Kraske sacrectomy, but this is not recommended.

CONSIDERATIONS ON THE COMPARATIVE MERITS OF THE VARIOUS METHODS OF SURGICAL RELIEF.

Since the ideal result of operative interference in cases of congenital imperforation is the restoration of the intestinal outlet in its normal situation or in the perineum or perineo-sacral region with good functional (sphincteric) control, it is natural that the aim of all surgeons should have been from the earliest historic period to attack the bowel at its terminus with the view of creating a perineal anus. In this respect the surgery of the present day has not modified the dominant surgical rule, which has always been to begin all operative procedures by the perineal route. While the advent of asepsis and the success of intra-peritoneal operations in the modern period have led many surgeons to advocate the primary intra-peritoneal exploration by laparotomy for the recognition of the anatomical seat of the obstruction as an initial procedure, the evidence gathered up to the present demonstrates that the greater chances of success not only in saving life but in securing the best functional results rest with the surgeon who first begins his intervention by the perineal route. The older statistics of Curling, Giraldès, Bodenhamer, Cripps, and Ducuron are confirmed in this respect by the later statistics of Anders, who, compiling his data from cases treated solely in the antiseptic period, demonstrates the greater benignity and success of the perineal method as a primary operation. Thus, up to 1889 Ducuron's statistics, which were a compilation of all previous tables, show that the simple perineal methods (exclusive of sacral operations) yielded 61.9 per cent. mortality and transperitoneal colotomy (Littre) 62.6 per cent. In 1893 the improved technique of the aseptic period enabled Anders to show that in one hundred cases the mortality of primary perineal proctoplasty had been reduced to 28.44 per cent., perineal incisions and other irregular procedures (trocar punctures, dilatations, etc., on the old plan) 32.22 per cent., while the Littre colotomies show 52.8 per cent. mortality. While the superiority of the perineal method as a primary procedure is maintained by the vast majority of modern operations, it is far from my intention to deny the advantages, and, in fact, absolute necessity, of resorting to primary colotomy or enterostomy for the purpose of creating an abdominal artificial anus in certain cases. All that I would insist upon is that the perineal operation by itself, or as modified by recent developments in the surgery of the pelvis, is the method of election in all cases of complete ano-rectal imperforation in which the infant is seen before fecal stasis, septicæmia, and exhaustion have set in, when the prime object of the operator is to save life, regardless of the subsequent functional result. It is plain that when such conditions of extreme constitutional depression exist as to demand the most rapid and certain way of creating an outlet for the retained meconium primary colostomy or enterostomy is the only choice left to the conservative operator, no matter how repugnant this may be to his surgical inclinations. The great argument in favor of colotomy as a primary operation in the past was due to the imperfections and limitations of the perineal operation. The narrow limits of the perineal space and the fear of infecting the peritoneum caused the surgeon to lose much and valuable time in searching for the undescended rectum, so that this bowel was often missed even when, by the modern methods, it could have been easily discovered and opened at the perineal or perineo-sacral wound. This liability to miss the bowel after a long and fruitless search often compelled the surgeons to perform iliac colotomy under the most disadvantageous circumstances, which seriously militated against the success of the final operation (colostomy); hence the disposition to favor colostomy as a primary operation in many quarters. But the recent extension given to the perineal operation by coccygeo-sacral incision and the modified Kraske operation, with the demonstrated advantages of the intra-peritoneal exploration of the pelvis through the perineal incision, as Stromeyer first suggested, has enormously increased the chances of success by the perineal operation, and has made the vast majority of cases of imperforation amenable to this procedure. The contra-indications to the perineal or extended perineo-sacral operation are not based so much on anatomical grounds—viz., the narrowness of the pelvis, the distance of the rectum from the perineum, or the danger of wounding the peritoneum—as was formerly the case, but are grounded almost exclusively on the general condition of the patient as to viability at the time when the surgeon is called upon to interfere in his behalf.

THE PERINEAL OPERATION: PROCTOPLASTY.

The operation of proctoplasty, by which is meant the creation of an anal orifice imitating the natural outlet of the rectum, was not known or understood until 1835, when Amussat laid down the rules for its proper performance. Prior to this time the operation for the relief of ano-rectal imperforation consisted simply in the introduction of a trocar with the view of tapping the rectal pouch, or of perineal incisions of variable length and depth, followed, if necessary, by the use of the trocar and dilating agents. Then the sole aim of the operator was to reach the rectal ampulla, and it was a great triumph if he succeeded in tapping and draining it by a happy puncture or a plunge of the bistoury made in the dark. After this was done, a long, tedious, and usually unsuccessful process of dilatation followed, which had to be kept up for years under penalty of recontraction.

The objections to the method of simple puncture and incisions in complete obstruction—viz., septic peritoneal or pelvic infection and perirectal infiltration, followed by secondary cicatricial recontraction—are too well known to require further consideration. They may be dismissed with the simple statement that they have no place in the surgery of the present, except as historic landmarks in the evolution of the operation of proctoplasty.

The new operation proposed by Amussat, and practised by him in 1835, marks a great step in advance of his predecessors, and is to be regarded as one of the most conspicuous landmarks in the history of this branch of surgery. Amussat proposed that not only should the rectal pouch be sought for by a free and systematic dissection of the perineum, but (and this was the great merit) that after it had been found it should be freely detached from its surroundings, so that it might be dragged to the level of the skin, where, after opening and evacuating it, the mucous membrane could be sutured, without tension, to the skin, where it would unite by primary intention. Thus would a continuous mucous lining be formed for the anal passage which would prevent fecal contamination of the wound and permanently protect the parts from future cicatricial contraction. The manifest advantages of Amussat's operation over the old methods rapidly gained for it general acceptance, so that it soon became the classical operation,—a position which it holds to the present day in all cases in which the rectal pouch is accessible by the perineal route. The essential principle involved in Amussat's procedure—viz., the establishment of a direct mucous channel leading from the rectum to the skin, thus preventing a gap which would be filled with contracting cicatricial tissue—is the one to be remembered in performing a modern proctoplasty (πρωκτός, anus; πλάσσειν, to form), which must be resorted to whenever the rectal ampulla can be brought to the level of the perineum (perineal or normal proctoplasty), of the coccyx (coccygeal proctoplasty), or of the sacrum (sacral proctoplasty).

Presuming that the practitioner is called upon to deal with a case in which there is a total anal occlusion (Species II., III., IV., of Bodenhamer), in which the anus may be totally absent, or exists as a mere vestige, depression, pigmented spot, or is fully formed, like a cul-de-sac, or thimble, the steps of the operation should be as follows: (1) a simple perineal incision beginning from the perineo-scrotal junction and ending on the tip of the coccyx, or starting an inch below the vulvo-perineal commissure in the female (if the anus is well formed, it should be split in the median line on its posterior margin and carried back to the tip of the coccyx); (2) dissection in the median line down to subperitoneal fat, carefully respecting the levator fibres; (3) search for, and isolation of, the rectal pouch; (4) dragging of the pouch to the level of the skin in the perineal wound, followed by (5) puncture and evacuation of the rectum, and (6) suture of its mucosa to the skin by careful suture. Such would be the steps of a typical Amussat proctoplasty in simple cases.

THE ANAL INFUNDIBULUM (PROCTODÆUM): ITS TREATMENT.

I have thus far considered only those relatively simple cases in which the rectal pouch is easily discovered by extra-peritoneal manipulation and is readily dragged down to the perineal wound. This easy termination of the operation is, however, far from being the invariable rule, for, as surgeons all know, often more serious and trying complications combine to thwart the purpose of the operation. Before proceeding further it will be convenient to consider the question whether we should always avail ourselves of the anus and its cul-de-sac (the "anal thimble") and allow these to remain undisturbed when they exist. In simple cases it is unnecessary to increase the traumatism and complicate the operation by its excision, especially when all that may be required to restore the continuity of the recto-anal tract is to perforate a thin membranous partition which alone interferes with the escape of the meconium. But, unfortunately, in the majority this is not the case, and the infundibulum of the rudimentary anus is an impediment to a perfect proctoplasty and must be sacrificed, though its excision must be done in a manner that will respect as closely as possible the subjacent tissues, and especially the muscular fibres of the sphincters, if these exist.

When the rectal pouch is short, and terminates too far from the natural opening to permit its fixation at the normal anal orifice, the only alternative is clearly to create the anus at the lowest possible point and leave the original proctodæum without disturbance. But when the rectal pouch has been freely dissected from its surroundings, and is rendered flaccid by the evacuation of its contents, it is not only long enough to reach the normal anal margin, but leaves a redundancy of mucous membrane which may even exceed the amount required.

In such cases I believe it is a better plan to remove entirely the mucosa of the anal cul-de-sac down to its junction with the skin by careful dissection and substitute for it the mucous membrane of the enteron. It is feasible to dilate the existing anal infundibulum, and, after excising its dome, to suture the bowel to the anal mucous membrane within the edge of the sphincter (circular procto-rectorrhaphy), or, again, to unite the enteron to the posterior margin of the divided anal thimble (lateral procto-rectorrhaphy), as was done successfully in a recent case by myself; or, in an emergency, it may suffice to dilate the anal sphincter, perforate the dome of the cul-de-sac, and invaginate the rectal pouch through it, after which it can be held in place by two anchoring sutures to the skin until peritoneal adhesions form that will hold the invaginated surfaces in a firm joint. But all these methods are untried and, I believe, unsatisfactory, and the best procedure is the one previously advocated,—viz., to excise the original anal infundibulum *in toto*, leaving only the marginal mucosa intact, and suturing it to the rectal pouch at its edge.

The fear that incontinence of fæces may follow this operation need not be entertained. When the anus can be placed in its normal position and the mucosa of the infundibulum alone is dissected away, the sphincter fibres are not injured sufficiently to impair the ultimate functional value of the sphincter apparatus.

STROMEYER'S SUGGESTION, OR THE INTRA-PERITONEAL EXPLORATION OF THE PELVIS THROUGH A PERINEAL INCISION.

Thus far I have considered only those simple cases of imperforation in which the operator is able to recognize the rectal pouch in the subperitoneal tissues of the pelvis at a comparatively short distance from the perineal skin, and is able to anchor the ampulla at the site of the normal anus without serious difficulty. We must now consider the graver problems that confront the operator when he finds (1) that he cannot reach the missing rectal pouch by the ordinary perineal opening; or when, (2) having discovered it, he finds that it is impossible to drag it down without lacerating the perineal wound. It is in trying to solve these two problems that the surgeons of the present day have distinguished themselves and have added new resources to the perineal operation.

The older operators, even up to very recent times, looked upon the accidental opening of the peritoneum in this operation as a great calamity, and if they insisted so formally in following the hollow of the sacrum in their punctures and explorations, it was more to avoid injuring the peritoneum than to prevent accident to the bladder or other organs.

Their fears made them illogical; for after losing valuable time, sometimes hours, in ineffectual subperitoneal manipulations, in which the greatest caution was observed to avoid an accidental tear of the serosa, they abandoned the lower route and proceeded to do deliberately what they had been trying most strenuously to avoid,—viz., to open the peritoneum by another operation, and incur the risk of its infection by creating an artificial anus in the groin.

This inconsistency did not escape the observant mind of Stromeyer, who for the first time in 1844 expressed the opinion that if the rectal pouch could not be found after a careful subperitoneal search in the perineal wound, it was proper to open the peritoneum in the pelvic floor in order that the presence or absence of the rectal cul-de-sac be promptly ascertained.

Stromeyer's operation, as the Germans call this procedure, was never attempted by himself, but Liesrink applied it for the first time in 1872 and obtained a brilliant result. In this case the dilated end of the colon was recognized through the open peritoneum at a depth of seven centimetres; the gut was then seized with blunt forceps and dragged to the opening, where it was sutured and opened. The child made an easy and rapid recovery. After this, Krönlein (1879), Anders (1881), Vincent (1886–87), A. Mayo-Robson (1887), Cripps (1892), Judeau (1891), have reported cases in which this was deliberately done, and the example of these operators, combined with the authoritative advocacy of this procedure by Frölich, Fochier, Commandeur, Durand, Robson, Anders, and others, justify a resort to this mode of exploration at an early stage of the operation whenever the exigencies of the case may demand it. At any rate, it is not likely that we shall read many more accounts of those prolonged manipulations, so exhausting to the patient and the operator, which the French have picturesquely described as "foraging" (*forage*) or "mining" (*travail de mine*), which were, in the main, due to the dread of opening the peritoneum. In this way asepsis has rendered obsolete the old rule that the depth of the perineal exploration should be gauged by fixed measurement. Copeland

Hutchison laid down the rule, which was traditional for a long time with English authorities, that the limit of penetration should not exceed one and a half inches; as surgeons grew bolder, the limit was extended by two, three, four, five, and even six centimetres. Anders (1893) criticises Jacubowitz (1886) for stating that, in general, an exploration beyond the depth of four or five centimetres is dangerous because of the probable injury to the peritoneum, and says he (Anders) has frequently penetrated eight or nine centimetres into the pelvis (presumably through the peritoneum) in his own cases without ill effects. Stronger even than the evidence gathered by the comparatively rare and isolated experience in this class of cases is the convincing argument of modern gynæcological experience, which demonstrates by hundreds of cases of vaginal cœliotomy for uterine, tubal, and ovarian disease that the peritoneum is even more tolerant of trauma and less liable to septic infection in the pelvic cavity than when attacked by the higher hypogastric route. Therefore, to the questions, When should we open the peritoneum? and to what extent should we explore the pelvic cavity? I would answer, just as soon as a free subperitoneal dissection has failed to reveal the presence of the rectal pouch. Then an opening should be made in the pelvic peritoneum sufficiently large to admit the index-finger its full length into the pelvis, and the pelvis should be flexed upon the trunk in an extreme lithotomy position, with the view of exploring not only the contents of the pelvis, but the organs of the upper pelvic strait as well. In this way the possibilities of the perineal route will be promptly ascertained, and the course of the operator will be accurately determined without dangerous experimentation or delay.

EXTENSION OF THE PERINEAL INCISION: THE PERINEO-SACRAL ROUTE.

In operating upon imperforate infants by the perineal method several contingencies may arise which must now be considered. 1. The operator discovers the rectal ampulla by subperitoneal dissection or by intra-peritoneal exploration, but the bowel is too high up in the pelvis to be dragged down safely to the perineum; it can be reached, however, and anchored to the skin on a level with the coccyx, or perhaps higher up on a level with the middle of the sacrum. 2. A congenital deformity of the pelvis may so diminish the transverse diameter of the pelvic outlet that the operator cannot introduce his finger high enough between the tuberosities of the ischia to explore the pelvic cavity. In either case the solution of the problem lies in amplifying the original perineal incision by increasing the antero-posterior or sagittal diameter of the pelvic outlet in the direction of the coccyx and sacrum.

Amussat, who recognized these conditions, was the first to advocate and extend the perineal incision by removing the coccyx in a case that he reported November 28, 1835. His suggestion was forgotten until Verneuil revived it and brought its benefits prominently before the profession in his first paper of 1873. Kocher in 1874, and Lange, of New York, in 1883, independently practised coccygectomy in order to gain more ready access to the pelvis while extirpating rectal carcinoma in the adult; but the credit remains with Verneuil for having familiarized the modern generation with the value of this procedure in dealing with congenital ano-rectal imperforation. Verneuil advised the early excision of the coccyx by prolonging the median perineal incision over this bone, which he removed with great care, so as not to injure the muscles and soft parts that were attached to it. Byrd, of Quincy, Illinois, in 1880 was the first to apply the Amussat-Verneuil suggestion in this country. In 1875 Polaillon reported a successful case in which the imperforate rectal pouch had been reached by splitting the cartilaginous coccyx in two halves. Delens in 1874 succeeded by simply displacing the bone backward without removing it.

After a careful examination of the literature of the subject, I have been able to collect sixteen cases of coccygectomy since 1842, when Amussat performed the first excision of this bone for imperforation. Of these sixteen cases, ten recovered and six died. In four of the six fatal cases conditions existed which would have led to a fatal termination regardless of the operation; this would reduce the mortality to two out of sixteen cases, or 12.50 per cent.

In only two of the sixteen cases the rectal pouch could not be found in spite of coccygectomy; and lumbar colotomy in one and Littre's operation in the other had to be performed to empty the bowel; but even in one of these—Amussat's case—the colotomy would not have been required had the perineal exploration been carried farther.

Nothing was more natural after the extirpation of the coccyx than to advance a step farther, when great difficulties presented themselves in the way of reaching the dilated rectal pouch, or of attaching it to the skin, to increase the gap in the pelvis by cutting through the cartilaginous sacrum, sacrificing a part of this bone. Nevertheless, the important and close relations of this bone to the sacral nerves and vessels, and the fear of opening the sacral canal, caused this osseous barrier to be respected until very recent times. In fact, it was not until the feasibility and advantages of sacral incisions had been proclaimed and demonstrated by Kraske in his papers of 1885, 1886, 1887, and the isolated experience and opinions of previous operators, like Bardenheuer (1880), and the work done in coccygectomy by Verneuil, Kocher, and Lange, that serious interest was aroused in the sacral route as a legitimate surgical procedure.

The first published records that I can find in which the sacral route was utilized for the relief of ano-rectal imperforation are mentioned in the thesis of Mâitre, of Lyons, which appeared in June, 1887, and were gathered from the clinics of Professor Vincent of that city. In Vincent's first case the coccyx and sacrum were divided in the median line up to the middle of the sacrum (coccygo-sacrotomy); in his subsequent operations he adopted a parasacral incision. After Vincent's two cases (1887), the fol-

[illegible] reported for ano-rectal imperforation, in which [illegible] sacral operations were adopted: Ceci, Genoa (1890); [illegible] Chaput, Paris (1892); Poisson, Nantes (1895); [illegible] (1895), two cases; Fochier, Lyons (1894); the writer, [illegible] White, New Orleans (1895); Elliott, Boston (1896); [illegible] (1896); Brault, Algeria (1897); in all, fourteen cases. [illegible] in the sacrum were, in one case, simple coccygo- [illegible] in one, parasacral incision (Vincent); in one, Bar- [illegible] of the sacrum (Ceci); in ten, various portions of the [illegible] the scissors being invariably used; in two, osteo- [illegible] on the Rhen-Rydygier plan (Czerny).

[illegible] the operation: four out of fourteen cases were perma- [illegible] functional anus, or about twenty-eight per cent.; [illegible] cases died at variable periods after the operation (from [illegible] months), owing to causes indirectly connected with the [illegible] condition of the infant (64.27 per cent. secondary mor- [illegible] out of fourteen, death resulted a few hours after the [illegible] directly attributable to it,—*i.e.*, shock or exhaustion [illegible] immediate mortality). In five only out of the fourteen [illegible] coccygeal operation was insufficient to reach the ampulla. [illegible] nine, simple perineal incision failed to discover the am- [illegible] application of the sacral operation was effectual in establish- [illegible] perineo-sacral anus, though in two cases a supplementary [illegible] incision was required to reach the gut and direct it to the perineo-sacral region.

[illegible] purely technical point of view, the sacro-coccygeal route, [illegible] with actual excision of bone on the original Kraske plan, [illegible] osteoplastic flap, or by a parasacral incision, does not appear [illegible] special difficulties in the new-born infant. The only instru- [illegible] in dividing the cartilaginous sacrum were scissors or rongeur. [illegible] operators refer to the ease with which the coccyx or sacrum [illegible] through or excised, owing to its very partial ossification; none [illegible] have been annoyed by excessive or dangerous hemorrhage. Thus [illegible] accidents or evil consequences are reported from opening the ver- [illegible] canal, though in no case have the excisions of, or excisions through, [illegible] been pushed beyond the third sacral foramen.

THE CHOICE OF THE SACRAL OPERATIONS.

It will be observed that, from the especially favorable conditions presented by the new-born infant, almost any of the numerous modifications of the original Kraske operation are applicable to this class of cases.

The various operations performed by the sacral route may be classified into four groups: 1, definitive sacral resections; 2, temporary or osteoplastic sacral resections; 3, median sacral incisions without resection or with osteoplastic flaps (sacrotomy); 4, parasacral incisions.

The parent type of all definitive resections is Kraske's operation, of which a variety of modifications have been described. When Kraske's operation is performed for imperforation, the incision is begun at the anus and is carried up to the middle of the sacrum. The posterior surfaces of the coccyx and sacrum are exposed, and the soft parts covering them are separated and detached on the left border. The coccyx is disarticulated and a portion of the left half of the sacrum is removed. The line of the bone section takes a curved course, beginning below the third sacral foramen, arching inside the fourth, and terminating at the lower border of the fifth sacral vertebra. It is not necessary, however, to excise the coccyx *in toto*. All that is required in imperforate cases is to excise as much of the lateral surface of the bone with the sacrum as will permit of an easy exploration of the pelvis. The remaining steps, which in the adult consist in the isolation of the rectum and the extirpation of the cancerous neoplasm or diseased gut, for which this operation was especially created, do not concern us here. Neither are we interested in this contribution in the various methods recommended for suturing the divided end of the bowel after excision. These are most important and essential details in the surgery of rectal diseases in the adult, but in the operative treatment of congenital imperforation we are chiefly concerned in the methods by which a free, easy, and safe entrance into the lower pelvis of the new-born infant can be obtained. As in the majority of cases the enteron will be found and will be easily brought to the sacral wound by simple parasacral incisions or osteoplastic sections of the sacrum, it is scarcely ever required to sacrifice permanently any part of the sacral wall of the pelvis. One of the chief objections to this procedure is that the removal of an extensive portion of the osseous framework at this point weakens the pelvic floor and favors a procidentia of the rectum. This may occur after simple coccygectomy (Delens's case), and has occurred twice in the fourteen cases of sacral operations in which Kraske's incision has been followed. (Poisson's and my own experience.)

Hence it follows that in adopting the sacro-perineal route for purely exploratory purposes, it is well to select the simplest and most economical operation,—viz., that which, at least, will permit of an easy exploration of the pelvis and a closure of the wound without permanent mutilation. Fortunately, all this can be readily accomplished by either one of three procedures,—viz., (1) a parasacral incision (Vincent's, Zuckerkandl's, Wölfler's); (2) an osteoplastic resection in which a part of the sacrum and coccyx is elevated with the soft parts covering them, *in solido*, as a trap-door opening (Heinecke, Roux, Billroth, Rhen, Rydygier); or (3) by a median sacrococcygotomy as originally applied by Vincent. This last method may be combined with a transverse sacral incision, thus permitting the elevation of two bilateral osteoplastic flaps on the trap-door plan, which will readily give the operator an easy access to the pelvic cavity without any permanent damage to the osseous framework.

THE MEDIAN OPERATION (MEDIAN SACRO-COCCYGOTOMY) IS THE OPERATION OF ELECTION.

Notwithstanding the simplicity of Vincent's parasacral incision, or the modifications to which it may be subjected, I believe that, as a preliminary exploratory procedure, a simple median incision through the coccyx and sacrum (median sacro-coccygotomy) is the preferable operation.

The advantages that can be claimed for the median operation are: First, that it greatly diminishes the risk of injuring the levators and the coccygeus, and thus causes the least disturbance and the least weakening of the pelvic floor. Secondly, that it is less likely to injure the third and fourth sacral pair of nerves which supply the sphincters, levators, and bladder. Thirdly, that the median incision is less likely to be followed by hemorrhage. Fourthly, that a median incision will not interfere with the lateral blood-supply, which is a matter of consequence in making osteoplastic flaps, owing to the risk of sloughing.

These advantages of the median section have laid the foundation for the median osteoplastic temporary sacrotomies devised by Heinecke (1889), Kocher (1889), Jeannel (1890), Gussenbauer (1893), Borelius (1894), and Morestin (1894), who have modified the median or oblique section of the sacrum by the adoption of one or two transverse cross-sections on a level with the highest point of vertical division. The ultimate result of these procedures is that the sacrum and coccyx are divided vertically with the skin and other soft parts, and transverse sections are made, leaving a T-shaped wound which forms two osteo-tegumentary flaps, which can be replaced or sutured after the operation on the rectum is completed.

All these methods, as well as many others, have been devised to meet the conditions found in rectal cancer in the adult. I will not stop to consider them individually, but will simply refer the reader to the admirable and recent contributions of Kammerer, Gerster, Morestin, Taylor, and Sieur. I would also refer to my contribution on "The Surgical Treatment of Ano-Rectal Imperforation in the Light of Modern Operative Procedures," to be subsequently referred to, in which the various operations for imperforation that are practised by the sacral route are more adequately considered. I shall now simply limit myself to a brief description of median sacro-coccygotomy, as suggested by Vincent, and of Morestin's modification by T-section in its application to this class of cases.

Before proceeding with the technique of the operation, it will not be superfluous to consider, if only cursorily, a few points of preliminary importance.

To the inexperienced it is a matter of some difficulty to define by surface markings the safe limit of sacral section. All operators are agreed that excision of the sacrum should not be carried higher up than the lower border of the third sacral foramen. Above this point there is danger from the free opening of the sacral canal and the involvement of the nerve-supply to the bladder, which might be permanently paralyzed; in addition, injuries to the nerves that supply the levators and the sphincters might lead to permanent and irreparable atrophy of the pelvic floor. As a result of personal studies on the cadavers of new-born infants, I have found that the lower border of the third sacral foramen is situated at the distance of one and a quarter centimetres from the sacro-coccygeal joint. I would, therefore, advise that in the new-born infant the transverse section of the sacrum that is required in making an osteoplastic flap on the Roux or Rhen-Rydygier plan should be limited by a line not higher than one and a quarter centimetres or thirteen millimetres above the sacro-coccygeal junction. The same rule applies, of course, to median sacrotomy with or without bilateral cross-sections; this limit is usually amply sufficient for all exploratory purposes. In median sacrotomy the central section can no doubt be carried higher up, even one centimetre, without risk, as the nerves given off from the cauda equina are not disturbed.

Position of the Infant during the Operation.—In my own experience, an extreme lithotomy position is the most advantageous in performing the perineal operation. If the ampulla is not found after dissection of the deeper perineal planes, and the peritoneum is opened on the Stromeyer plan, it will be safer to turn the child on the abdomen, face downward, across the lap of its nurse, with the view of keeping the urine which may be involuntarily discharged from soiling the wound. This is the best position for coccygeal and sacral exploratory incisions. If the perineo-sacral incision reveals conditions that demand laparotomy, then the patient must be turned on the back after careful plugging of the perineo-sacral wound with iodoform gauze. The Trendelenburg position is the best for all explorations which are intended to attack the pelvic contents; but if there is marked tympanites, only the horizontal dorsal decubitus is permissible.

Anæsthesia.—It is safer to operate without general anæsthesia when the patient is seen late after birth, when tympanites, signs of stercoræmia, and exhaustion are manifest. This is particularly true if colotomy is resorted to as a primary procedure. Profound anæsthesia undoubtedly diminishes the chances of recovery in such cases. In robust infants a few drops of chloroform in an Esmarch inhaler will suffice to maintain a state of insensibility which will be sufficient to diminish suffering and relax the patient.

The Operation.—In cases in which the anus is reduced to a mere vestige, or is missing altogether, the incision should commence at the junction of the scrotum and perineum in the male, and the vulvar commissure in the female. The knife must be kept strictly in the median line and carried to the top of the coccyx, the operator being especially careful to separate the posterior fibres of the levator, if they are recognizable, without cutting their coccygeal attachments transversely. If the anus is normally formed, then the incision is begun in the middle of the posterior segment, and is carried to the tip of the coccyx with the same care to avoid dividing transversely the posterior fibres of the levator. This preliminary incision will expose

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the region below and above the lower pelvic diaphragm sufficiently to reach the rectal ampulla, if this exists, in the neighborhood of the pelvic floor, which is the normal condition in many cases. If the ampulla cannot be detected above the plane of the levators by this procedure, the median incision is then carried up vertically to the middle of the sacrum, and the coccyx and the sacrum are divided in the median line with a knife or straight scissors sufficiently to bisect the coccyx into two complete and even halves. The osteoplastic lips of the wound thus created are then pulled apart with retractors, so that the eye and the finger can search for the ampulla at a considerable depth in the pelvis above the levator level. If this opening is not sufficient, the incision in the middle line of the sacrum is carried up to the level of the third sacral vertebra without making transverse section; this will greatly add to the space gained by the bisection of the coccyx, as the cartilaginous elasticity of the sacrum greatly facilitates the free retraction of the wound and exploration of the presacral organs; while retracting the osseous lips of the sacro-coccygeal incision, and by following the median line, the connective tissue and fascial planes in front of the sacrum are divided in the posterior tendinous interspace between the levators, and the cavity of the pelvis is fully explored. Without further amplification of the incision by transverse sections, the pelvic contents, the rectum, the uterus, the vagina, the ovaries and tubes, and the bladder can be recognized without necessarily opening the pelvic peritoneum. The search for the ampulla can be continued subperitoneally up to the promontory of the sacrum by following closely the hollow of the bone. In the majority of cases the rectal pouch will be recognized at this stage of the operation, especially if the pelvis is flexed and the lower abdominal region is forcibly pressed downward and backward so as to direct the pelvic contents towards the lower pelvic outlet. If the ampulla should be completely invested by peritoneum, the parietal serosa can be freely incised so as to facilitate the descent of the rectal pouch in the sacral wound. If more space is required for manipulation or exploration, a transverse section in one or both sides, so as to convert the wound into two triangular osteoplastic flaps which will tend to close in the median line spontaneously, can be advantageously and easily effected with the help of the knife or scissors.

Before this final step is undertaken, it will be the easier plan to ascertain the exact relations of the ampulla and determine the practicability of dragging the rectal pouch successfully to the sacral wound by a careful intra-peritoneal exploration. If a well-formed anal orifice exists, it must be carefully dilated and effort made to suture the rectal ampulla to the margin of the anal mucosa after excising the superfluous portion of the anal cone. If the rectal pouch is rooted at too high a level, and the mesenteric resistance is too great to drag it down to the anal orifice, an effort should be made to suture it (after preliminary tapping and evacuation of its contents) to the coccygeal region, in the interval between the levators, in such a manner that the new anal outlet shall be placed below the plane of the muscular diaphragm formed by these muscles. To accomplish this, the coccyx may have to be sacrificed after a careful enucleation.

By adopting this median procedure the operation can be performed not only rapidly but safely, because the hemorrhage is comparatively insignificant; it is obvious also that if the new anus is placed below the plane of the levators, the prospects of future sphincter control will be greatly increased.

Finally, when the rectal pouch is rooted too high up in the pelvis to be anchored in the coccygeal region, there will be no alternative but to attach it to the sacral region on a level with a higher point in the sacral incision. Under such circumstances it is evident that the technical rules which govern the formation of a perineal anus (proctoplasty) must be carefully applied. The guiding principles in making a *sacral* anus are: (1) To make it as low down and as near the normal outlet as possible. (2) To carefully unite the mucosa to the skin, otherwise a simple fecal fistula results, with its tendency to eczema, dermatitis, and stricture. (3) There must always be enough of the enteron or bowel to permit of an exact suture to the skin without tension. In suturing the mucosa to the skin, Vincent's suggestion can be applied with advantage. This procedure consists in cutting two elliptical pieces of skin as the site of the proposed anus. This increases the raw surface for muco-cutaneous apposition and adhesion, as it allows the mucosa of the enteron to spread out like the normal anus over a firm base of attachment.

It is obvious that in cases in which the enteric pouch is too short to be dragged to the surface of the skin and the strain is too great upon the sutures, it will be safer to perform an iliac colostomy with the expectation that the gut will ultimately descend into the pelvis. *Better, far better, a technically perfect iliac anus than an imperfect contracting and painful fecal fistula in the sacral region.*

Gersuny's Procedure.—To obviate the possibilities of fecal incontinence when the rectal pouch is attached to the sacrum, the enteron should be attached below the insertion of the levators, and when this cannot be done it will be well to give the ampulla a rotary twist upon its axis, so as to narrow the lumen of the outlet and thus favor the control of the alvine evacuations, as suggested by Gersuny. Notwithstanding what Morestin and others have said in condemnation of this procedure, recent experience has shown (Gersuny, Gerster, Chaput, Thomas, Eiselberg, Prutz, Rhen, Keen, *et al.*) that this expedient is by far the most practical and beneficial of all the methods that have been recommended to obviate the risk of fecal incontinence. In applying Gersuny's axial rotation, the open extremity of the gut should be twisted upon its axis until the finger, which is introduced into the lumen of the gut, feels a certain resistance and constriction. The degree of twisting usually required varies from 120° to 270°. The edge of the rectum is then sutured to the skin by two tiers of sutures. If the tension is sufficient, the flow of mucus and other intestinal secretions that is usually observed after the operation ceases at once. Gersuny[1] lays special stress upon the careful suturing of the rectal edge to the skin. If the sutures are not drawn tightly, the bowel will untwist, and the effect of the tension will be lost. In order to prevent the untwisting of the bowel, he suggested that the torsion be made permanent by suturing the rectal folds externally by several sutures applied to the bowel itself before the mucosa is sutured to the skin. Of course, care must be taken that the bowel is not strangulated by overtwisting; but the bowel can stand a great deal of tension without interference to its circulation, and the danger is usually in the other direction,—viz., insufficient torsion.

One of the great advantages that can be claimed for the sacral route in dealing with anal malformations of the kind here considered is that it permits the operator to cope more readily and directly with the complications that frequently exist, such as vaginal, rectal, or vagino-rectal fistulæ. The clinical demonstration of the superiority of the sacral route in dealing with such conditions is still lacking. But, judging merely by the results of experimental exploration of the pelvis in still-born infants by the various sacral operations, I am confident that many cases in which these complications exist (which otherwise would be inaccessible or could be palliated only by colostomy) will be directly relieved in future by the sacral operation. This is more particularly true of the more serious cases in which a fistulous communication exists between the rectum and the bladder. Colostomy has been the only remedy for such sufferers, but this is at best a palliative treatment. The aim of the operator should be to separate the connections that exist between the bladder and the colon. The ease with which access can be obtained to the bladder as well as the rectum, and even the sigmoid flexure, in new-born infants, by sacro-coccygotomy would suggest the feasibility of radically relieving them by this operation.

To conclude, then, with the sacro-coccygeal route, it is indicated: (1) As a primary exploratory measure in all cases of imperforation in which a simple perineal incision will not permit of adequate digital exploration of the pelvic cavity up to its brim. (2) In all cases in which intra-pelvic exploration through a perineal incision demonstrates the presence of the enteron at an accessible point in the pelvis, but which cannot be dragged down. Here the sacral route is applied with a view of reaching the rectal pouch, so as to evacuate it and mobilize it sufficiently to permit its suture at the lowest point in the sacro-perineal wound. (3) In all cases of imperforation in which the enteron communicates by fistulous openings with the vagina, the bladder, or the urethra, the object of the operator being to close the abnormal communication and restore the normal outlet. (4) In all cases in which the enteron has been arrested in its descent above the pelvic brim, but in which a secondary inguinal laparotomy with colostomy permits the operator to drag the enteron into the pelvis or to a convenient point on the sacral surface, where a proctoplasty can be successfully attempted. (5) As a secondary procedure in all cases of primary inguinal colostomy in which exploration through the colon demonstrates that the rectal cul-de-sac can be guided to the pelvic floor, where an anus nearer the natural outlet can be formed.

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[1] Twenty-sixth meeting of the Deutsch. Chirurg. Gesellschaft, Berlin, April 24, 1897.

The Upper or Anterior Abdominal Route (*Cœliotomy*).—When, as in very exceptional cases, the enteron cannot be found in the pelvic cavity or in the margin of the brim of the pelvis, or, again, when the terminal cul-de-sac of the colon is present, but is so firmly attached to the lumbar region that it cannot be dragged down without the risk of tearing and spilling the meconial contents into the peritoneum, there are five paths open to the surgeon who has already begun his operation by the perineal route. His choice between these methods must then depend largely upon the general condition of the patient when this stage of the operation is reached, and upon the anatomical conditions that he may find after thorough intra-peritoneal exploration through the perineo-sacral wound. If exhaustion is marked and life is ebbing fast, and it is evident that any additional traumatism or shock would be fatal, the wisest course to pursue would be: (1) To seize the nearest distended coil of intestine, attach it to the wound, and give issue to its contents. (2) But if the condition of the patient is fair and will justify further operating, a lateral or median laparotomy should be performed with the view of identifying the true terminus of the colon and bringing it down to the perineum. (3) Or should the strength of the patient fail at this moment, it would be preferable to perform a simple colostomy to relieve intestinal tension, with the expectation of completing the operation at some future time,—*i.e.*, making a perineal anus by guiding the rectal cul-de-sac to the pelvic floor with the help of a director introduced into the colon through the inguinal opening. (4) Should the worst anatomical condition be recognized, and it be found that the colon terminates too high up in the lumbar region to permit of its immediate or subsequent descent to the perineum, then the *dernier ressort*, a permanent iliac anus, must be made. (5) Should even the colon be difficult of access (as in truly teratological cases), so as to make a left inguinal colostomy impossible or very difficult, a simple enterotomy on the Nélaton plan, by attaching the nearest and most distended loop of the bowel, would be preferable to making an additional opening in the right iliac region with the view of draining the cæcum. (Pillore's operation.)

Finally, the questions will be asked, Why not perform a primary cœliotomy to determine the exact anatomical conditions? and if these permit, why not guide the rectal cul-de-sac to the perineum, where the physiological anus can be restored by a combined abdomino-perineal operation? or, again, if the anatomical conditions contra-indicate this procedure, why not proceed at once to the performance of an inguinal colostomy?

These questions have all been discussed in detail by me in a recent contribution on the surgical treatment of ano-rectal imperforation,[1] and as a result of this comparative historical and statistical study the following conclusions have been arrived at:

First, as to the propriety of performing a simple enterostomy or creating an artificial anus on the Nélaton plan. This procedure is of course called for, as in the surgery of intestinal obstruction in the adult, only as an emergency procedure. It may be applied in the perineo-sacral wound or in cœliotomy by the upper route. In either case it is a deplorable expedient, but as a life-saving measure in exhausted children it has its justification. It cannot be regarded with favor under ordinary circumstances, because it is certain to exclude a portion of the terminal intestinal tract of unknown length and of probable great physiological value. It has not been applied often in cases of imperforation, but there is at least one case, that of Krönlein (1879), in which the operator was compelled to resort to it by pressure of circumstances, but yet succeeded in saving the child. Not only was he able to save the infant, but Krönlein, seven months after, explored the pelvic cavity through the artificial anus and discovered a mass which he recognized as the missing rectal ampulla. This was reached by a perineal incision where the pouch was opened and sutured to the perineal skin, creating a perfect perineal anus. The artificial anus in the groin was then closed, and the child made a complete recovery. If we may judge by this case alone, there is ample justification for an artificial anus, not only as a temporary life-saving measure, but as a permanent source of relief as well.

The other expedients referred to may be classified and considered as follows:

Primary Perineal Incision (with or without Coccygectomy or Sacrectomy), followed by Median or Lateral Cœliotomy, with a View to recognizing the Rectal Ampulla and guiding it to the Perineal or Sacral Wound.—This suggestion, which originated with Neil McLeod in 1879,[2] has been carried into effect with some modifications in six reported cases,—viz., by Hadra (1884), recovery; Connant (1891), recovery; Chaput (1891–1892), recovery; P. Delagenière (1893), recovery; Elliot (1895), recovery; Chalot (1896), recovery.

In all of these instances the patients recovered from the operation. They are considered as operative successes, notwithstanding the fact that in Hadra's case the child died on the fourth day from accidental asphyxia; in Connant's case, death in three months from entero-colitis; in Delagenière's case, death on the ninth day from broncho-pneumonia; in Chaput's case, death in two months from diarrhœa. Both Chalot's and Elliot's cases were living and apparently in good condition at the time of the report, which was several months after the operation in each instance.

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[1] *Vide* Transactions of the American Surgical Association, vol. xv., 1897, and in the American Journal of Obstetrics, November and December, 1897.

[2] British Medical Journal, 1880, vol. xi. p. 657.

From this evidence we can state that the immediate operative results following McLeod's procedure are satisfactory, as the operation appears to have been well borne in the six cases in which it has been practised. The final history of these cases, however, points to the great mortality that prevails in these as in all classes of defective or ill-developed infants.

PRELIMINARY COLOSTOMY, FOLLOWED IMMEDIATELY OR AT ANOTHER SITTING BY THE RESTORATION OF THE PERINEAL ANUS.

The germ of this suggestion is found in a communication addressed by Martin (le jeune), of Lyons, to the Société de Santé de Lyon,[1] in which he proposed that in cases of congenital atresia ani an incision should be made in the iliac region, and that the sigmoid be pulled out of the wound sufficiently to introduce a guide into its lumen, which would be used to press the rectal cul-de-sac against the perineum. The modern conception of this operation was first clearly stated by Chassaignac in 1856, who advised that after a preliminary colostomy in the left groin the artificial anus could be utilized to guide the terminal cul-de-sac of the colon to the perineum, where it could be opened, the opening left by the colostomy being closed permanently after the formation of the perineal anus.

This useful suggestion, which begins with Chassaignac's case, appears to be well supported by clinical evidence, as in seven cases in which it has been more or less perfectly carried out (Chassaignac, Krönlein, Haynes, Lannelongue, Demelin, Maylard, and Stuart and Bierwith) there were seven recoveries, or one hundred per cent. operative recoveries.

In this group the formation of the perineal anus (proctoplasty) has been considered in the light of a secondary procedure, to be performed at a subsequent sitting, when the colostomy is fully established. It is the operation of election, as Sir William McCormac taught, which we should select in weak, exhausted subjects, simply to meet the immediate demand for the relief of intestinal tension and fecal stasis. It is not to be considered as the method of election when the patient is still strong, resisting, and in condition to permit further explorations and manipulation, when the rectal pouch is known to be long and movable enough to permit its implantation in the perineum. If, however, after the performance of a primary colostomy under these critical conditions, a favorable change should be observed in the condition of the patient (as may possibly follow the relief of a distended bowel), then the question arises, Should we at once proceed to the immediate restoration of the normal anus by Chassaignac's method?

The clinical evidence that I have been able to gather is limited to four cases: reported by Edmund Owen, two cases (1880); Stephen Paget, one (1890); Chaput, one (1892). All these cases ended fatally, but the deaths took place some time (days and months) after the operation, from causes not directly attributable to the operation.

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[1] Recueil des actes, 1798.

The objections to this mode of operating may be summed up as follows:

1. That there is danger of perforating the rectal cul-de-sac by a probe or director while attempting to force it to the perineum, and that peritonitis may follow.

2. That the primary fixation of the colon in the groin may interfere with its subsequent mobilization, as appears to have been the difficulty in Chaput's case.

3. If the formation of the perineal anus is long delayed, fecal stasis with ulceration may take place in the blind end and fatal peritonitis may follow, as in a case reported by McCormac.

4. Serious secondary operations may be required to close the iliac anus, as in Stuart and Bierwith's patient.

On the other hand, the advantages of a reasonable delay before attempting the final perineal proctoplasty are:

1. That the patient is given a better opportunity to recuperate and prepare for a complete operation.

2. That the cul-de-sac may spontaneously descend lower into the pelvis, and thus become much more accessible than at the time of the formation of the iliac anus (Krönlein's case).

As to primary exploratory cœliotomy, whether median or lateral, which was ably advocated by Mr. Paul Delagenière in 1894,[1] with the view of expediting the recognition of the anatomical relations of the enteron, and, if possible, dragging it to the perineum after detaching it from its mesentery, I have found not only little clinical evidence to support it, but I cannot advocate it as a safe procedure, in spite of its theoretical plausibility. In the three cases the report may be summed up as follows:

1. P. Delagenière's case: an attempt was made to drag the colonic cul-de-sac out of its serous capsule by my method, and then to guide it to the perineum; but this failed, and an inguinal colotomy had to be performed. Death on the third day.

2. H. Delagenière's case is a typical procedure of this kind, in which the operator was successful from the operative point of view, but death occurred on the fifth day from causes apparently not directly connected with the operation.

3. C. Remy's case: median cœliotomy; rectal cul-de-sac recognized after great difficulties; the exceedingly short mesentery prevented the operator from satisfactorily attaching the enteron to the perineum. Death in thirty-six hours from shock and sepsis.

The objections to primary cœliotomy, apart from the scant evidence that can be quoted in its favor, are:

1. That in the majority of cases of imperforation the rectal cul-de-sac will be found to be accessible by the perineal or perineo-sacral route, and that therefore a double traumatism and corresponding shock will be

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[1] Archives Provinciales de Chirurgie, t. xi., No. 7, July, 1894.

avoided if the exploration is begun by the perineum, especially if Stromeyer's suggestion and coccygo-sacrotomy are resorted to promptly.

2. The great advantage claimed for primary cœliotomy for exploratory purposes is the rapidity with which the anatomical condition can be recognized. But this argument is fallacious, as is shown in Remy's case and in all cases in which there is much tympanites; in such cases evisceration would be frequently required in order to clearly recognize the terminus of the colon; furthermore, the argument in favor of prompt diagnosis has lost its weight since the intra-peritoneal exploration of the pelvis by Stromeyer's method through the perineo-sacral route has become a typical and practical procedure.

3. Not only are the technical difficulties in the way of reaching the enteron by upper cœliotomy, of identifying it, and guiding it to the pelvic floor much greater than inexperienced operators imagine, but the difficulties in the way of accomplishing this purpose, even after the rectal cul-de-sac has been recognized, are at times almost impossible to overcome with safety. The main cause of trouble is the tympanitic distention of the bowels and the packing in of the pelvic contents, especially at the brim, which is plugged up, as it were, with the distended rectum, bladder, and other viscera. As a rule, the bowels must be tapped early in the operation before the true condition of the parts can be recognized, and before the blind end of the rectum can be pushed into the pelvis to its final resting-place in the perineum. This compulsory enterostomy seriously complicates the situation by increasing the risk of peritoneal infection in the subsequent manipulations. Of course, if the operation is performed early, intestinal tympanites scarcely exists, but even at an early moment the enteron will be found quite full and blocking the way at the pelvic brim, so that tapping has to be resorted to before the blind gut can be guided to the peritoneum.

In conclusion, I would state that the only real advantage offered by primary exploratory cœliotomy, especially if the abdominal incision is made in the left linear semilunaris, is that it allows the operator to proceed to iliac colostomy without serious delay. This would limit its application as a primary procedure simply to weak, exhausted infants, who are brought to the operator at a late hour, when signs of intestinal sepsis and excessive tympany demand the most expeditious method of relieving intestinal tension and fecal stasis. Under such adverse circumstances an exploratory cœliotomy is made in the left groin with the view of determining the relations of the rectal ampulla solely as a preliminary to inguinal colostomy, which is the operation of election. The advantage of exploration before opening the colon in such cases is that the operator will probably be able to determine at once whether he is to perform a permanent colostomy or to provide a mere provisional anus, to be obliterated at a later day when the condition of the patient will permit of radical relief by perineal proctoplasty.

DISEASES OF THE PANCREAS.

BY EDWARD MARTIN, M.D.

ACUTE PANCREATITIS.

THIS affection, the pathology and symptomatology of which have been minutely studied by Fitz, was at one time supposed to be unknown in children. It is extremely rare even in adults, and gives rise to symptoms so like those of acute intestinal obstruction or of an irritant poison that differential diagnosis may be impossible.

These cases have been grouped by Fitz under the headings hemorrhagic, suppurative, and gangrenous. He believes that the disease is a complication of gastro-duodenitis, and that it is a cause of peritonitis which is generally overlooked.

McPhedran[1] has reported a case of hemorrhagic pancreatitis occurring in a child of nine months. The symptoms were those of acute intestinal obstruction, and the diagnosis of intussusception was made, for the relief of which an operation was performed. This was necessarily fruitless, and the true nature of the affection was discovered only when an autopsy was made.

A case representing the typical features of the disease was reported by Cayley.[2] The patient suffered for one or two days from epigastric pain and tenderness associated with constipation. There followed vomiting and rapid collapse, death occurring within five days from the beginning of the symptoms. In such acute cases peritonitis may develop or abscess may form in or about the pancreas, the patient surviving for weeks or months, or even recovering by the spontaneous evacuation of the pus. The rapidly fatal cases show an inflammatory exudate of the pancreas with fatty and coagulation necrosis of the glandular structure and extensive hemorrhage in and around the gland. The rapid and fatal collapse is attributed to compression of the retro-peritoneal ganglia by the inflammatory exudate. The hemorrhagic form is fatal within a few days. The suppurative and gangrenous forms run a more protracted course.

Symptoms.—These are, as has been stated, extremely misleading. Cayley states that the characteristic symptoms of the disease are epigastric pain and tenderness which may be of only moderate severity, resembling the pain of gastric catarrh, or may be very severe from the onset; then vomiting, the matter regurgitated not showing the characteristics of the intestinal contents. There is usually constipation, but sometimes diarrhœa. There may be fulness and swelling in the epigastrium, but no general distention or any signs of the presence of gas in the abdominal cavity. The tenderness is mostly limited to the epigastric region. Fever is constant, and with the collapse the temperature falls. A fatal collapse may set in as early as the second day, more commonly on the third or fourth.

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[1] Canadian Practitioner, September, 1896.

[2] British Medical Journal, September, 1896.

In the case reported by Cayley the diagnosis was based upon the absence of a history of gastro duodenal ulcer and of general abdominal distention. The symptoms were of gradual onset, and when collapse developed it was persistent and profound, showing no tendency towards reaction. Fulness or even swelling of the epigastric region could not be detected in this case.

Diagnosis.—This might be suggested by a history of gastro-duodenitis and by the presence of epigastric tenderness and tumor. Theoretically, sugar might be expected to be found in the urine, but in many of the reported cases this sign has been absent.

Prognosis.—This is extremely grave.

Treatment.—There are no direct means by which acute internal affections of this nature can be controlled. Collapse should be treated in accordance with general principles, by stimulants administered by the mouth, the rectum, or hypodermically, strychnine being particularly serviceable, morphine not being withheld when pain is severe.

Oliver holds that suprarenal extract is an extremely powerful means of controlling vaso-motor paralysis and stimulating the heart; pressure on the abdomen is also serviceable, since it has a tendency to limit the amount of blood which stagnates in the mesenteric vessels. The most important point of treatment is, perhaps, the making of a proper diagnosis, and thus the avoidance of a futile laparotomy. In the more protracted cases with abscess-formation, as shown by hectic, tumor, and leucocythæmia, incision and drainage are indicated.

CYSTS OF THE PANCREAS.

The etiology of pancreatic cysts has been fully described in vol. iii. of this Cyclopædia. A study of the more recent literature on this subject simply accentuates the importance of trauma as a predisposing factor. Thus, Leith[1] has collected seventeen cases in which the history of injury was definite, and a cyst followed so hard upon it that there was strong presumptive evidence as to the relationship between this pathological formation and traumatism. An interval of ten days was the shortest period which elapsed between injury and cyst-development; the longest was an interval of eight years. In this last case there is necessarily some doubt as to the relation between cause and effect. In six cases the interval was between two and four months, in five cases between two and five weeks. Trauma was due to falls, blows, and crushes.

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[1] Edinburgh Medical Journal, 1895–96.

Cathcart explains the formation of these cysts in this way. The injury causes laceration of the gland and necessarily extravasation of blood and pancreatic secretion from the torn vessels and ducts. The quantity of fluid is constantly augmented by the continuous secretion of the gland. The contents of the cyst are somewhat irritating, thus causing the formation of a capsule which gradually increases in size from internal pressure.

Durante records a case of pancreatic cyst on which he operated, caused by obstruction of Wirsung's duct, due to the *Ascaris lumbricoides*. There was sepsis of the pancreatic fluid with softening of the parenchyma.

Symptoms.—Aside from the growth of a tumor in the epigastric or right or left hypochondriac regions lying behind the stomach, often distinctly conveying the aortic pulsations, exceptionally associated with glycosuria, there are no characteristic symptoms.

Diagnosis.—This is practicable only when the cyst has attained considerable size. A history of traumatism or of pancreatic colic, followed by an epigastric tumor of rapid growth, and in exceptional cases associated with sugar in the urine, would suggest pancreatic growth. As the tumor grows it necessarily pushes the stomach forward and ultimately towards the right side, the transverse colon being displaced downward. Inflation of the stomach and colon will readily demonstrate the relations these organs bear to the tumor. When this has reached a large size, occupying the entire abdominal cavity, it is quite impossible to distinguish it from tumors of ovarian or nephric origin.

Prognosis.—This, when a portion of the gland still remains functionally active, is extremely good, provided operation be undertaken before serious lesions result from mechanical pressure.

Treatment.—There have been a sufficient number of pancreatic cysts reported to enable surgeons to clearly formulate the appropriate treatment. This consists in incision and drainage. Aspiration is ineffectual and dangerous, and attempts to cause obliteration by injections of irritating solutions, such as iodine, have resulted in fatal peritonitis. The cyst should be brought forward to an abdominal incision, secured in this position by suture, opened, and continuously drained. This necessarily causes a pancreatic fistula which may or may not close spontaneously.

Leith has reported seventeen cases thus treated with complete success in all but one. Krecke states that of twenty-seven cases all were successful.

Excision has been strongly recommended as the ideal operation. The procedure may, however, be extremely difficult, indeed quite impossible, and from reported cases it is evident that the mortality of the operation is high.

Anterior incision and drainage may be unsuccessful. It is especially serviceable in those movable cysts which can readily be brought to the anterior abdominal wall and are broader in front than behind. It is, however, not well adapted to those deeper-placed cysts which are somewhat fixed and are broad posteriorly. These are hard to reach, and it may be impossible to bring their walls to the edge of the parietal wound.

Leith[1] states that since the method of anterior incision and drainage for pancreatic cysts is open to the disadvantages already given, these accumulations may be primarily reached and drained by posterior operation. After experimenting on cadavers, he found that he could readily cut down on the tail of the pancreas and open the lesser omental sac. A vertical incision about three inches long was made in the outer border of the erector spinæ muscle, beginning above the twelfth rib. After cutting through the skin and fascia, the thin fibres of the latissimus dorsi are first recognized, and then the strong fascia at the outer border of the erector. This is incised, and the upper border of the quadriceps lumborum is next seen running downward and outward. The finger is inserted above it, and feels for the posterior surface of the kidney through the fat and cellular tissue. It then defines the position of the renal vessels. The tail of the pancreas and the postero-lateral wall of the lesser omental sac lie just above and inside them, and by inserting a probe it is possible to enter the cavity of this sac either through the posterior peritoneum or above this gland, according to the upward obliquity given to the probe. In no case was the suprarenal capsule or any other structure in the neighborhood injured. When the lesser omental sac was distended with fluid, fluctuation could be distinctly felt by the exploring finger working upward above the renal vessels, and fluid could be evacuated with ease by means of the drainage-tube made to enter the sac, with the help of a director and a pair of dressing forceps.

This procedure Leith thinks will be found to be quite easy in case of pancreatic cysts and probably of others in this neighborhood. The cyst-wall may be in some cases thick and thus be difficult to penetrate, but this need not cause trouble, while long-existent distention will have brought the cyst within more easy reach. It may even extend downward below the line of the renal vessels, and thus be reached by the surgeon whether he goes above or below these structures.

Cotterill adopted this method of reaching these cysts with complete success, the sac being entered below the renal vessels.

Gould mentions a case in which the cyst was fixed and could not be brought to the anterior surface. Passing a finger into its cavity, he cut down below the twelfth rib and drained it posteriorly. The fistula rapidly closed.

The advantages of this method of procedure are that it is easy, is safe, drains perfectly, and is followed by rapid convalescence. Even should the surgeon fail to reach the cyst, this would in no way prejudice the success of the anterior exploration, and would, moreover, even in such cases, allow the rapid establishment of posterior drainage.

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[1] Edinburgh Medical Journal, 1895.

Richardson[1] records a case of cyst of doubtful pancreatic origin cured by complete extirpation. The patient, a child of thirteen months, had exhibited a tumor in the abdomen since birth. This rapidly increased in size and embarrassed both circulation and respiration. The whole abdomen was filled by a fluctuating tumor, supposed to be caused by hydronephrosis. This tumor was dissected from its attachments back of the peritoneum in the pancreatic space. There were numerous large vessels supplying it, and it was crossed by the splenic vein; the tail of the pancreas was spread out over its anterior portion. Examination of the cyst-wall showed true pancreatic tissue. Enucleation was rapid and easy and, by avoiding the splenic vein and tying the pedicle mass, no hemorrhage ensued. The author holds that cysts of the pancreas, if they can be easily separated from attachments, should be removed, since there is a possibility of reaccumulation of fluid when such cysts are treated by drainage. Attempts at enucleation should, however, be abandoned when it is evident that the cyst is so thoroughly incorporated with the surrounding tissues that it can be separated only by cutting.

Zweifel records twelve cases of complete extirpation, followed by five deaths. It is noteworthy that the cases of incision and drainage heal very slowly, and there often remains a permanent fistula; hence posterior drainage is becoming more popular.

The operation of incision and drainage should not be divided into stages,—that is, the cyst should not be exposed and sutured to the parietal opening at one time and its contents later evacuated. As soon as the wall is exposed, it is grasped by the toothed forceps employed in the treatment of ovarian cysts, is punctured by the canula, and its contents evacuated, the general peritoneal cavity being carefully protected by gauze pads. When the cyst is large and movable, it is drawn through the abdominal wound as soon as a sufficient quantity of fluid has escaped to make this practicable. After thorough evacuation and washing out of the contents with dilute antiseptic solution (1 to 10,000) or sterile normal saline solution, the cyst is incised and the edges of this wound are secured to the abdominal opening by suture. The drainage-tube, the lumen of which should be large, is carried to the bottom of the cyst, sterile gauze is packed in lightly, the skin is protected by a thick boric ointment, and a generous sterile gauze dressing is applied, after which a pressure bandage is used.

CALCULI.

As in the case of biliary calculi, formation of pancreatic stones is important, at least from a surgical stand-point, only when from lodgement in the smaller or larger ducts there result obstruction to the free flow of the secretion, dilatation of the glandular substance, the outlet from which is thus interfered with, and following dilatation either cyst-formation or atrophy, dependent upon the degree of stoppage.

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[1] Boston Medical and Surgical Journal, No. 132.

A catarrhal condition of the ducts of the gland is probably the usual predisposing condition to the development of calculi, although changes in its secretion are undoubtedly important factors. It is worthy of note that mechanical obstruction may also occur from lodgement of a gall-stone in the common biliary duct.

Retention cysts due to obstruction by calculi rarely attain large size.

Brunet, Freyhan, Picot, and others report cases of occlusion of the duct of Wirsung produced by calculi, followed by atrophy of the glandular structure and glycosuria.

Symptoms.—It is worthy of note that the diagnosis of nearly all cases of pancreatic calculi has been made post mortem. The case recorded by Holzmann[1] perhaps exhibits the symptom-complex in its typical form. This patient suffered from severe pain in the hypochondrium, radiating at times to the right shoulder, and followed by salivation. During the attacks distinct traces of sugar were found in the urine. When the pains were slight and abortive in type there was neither glycosuria nor salivation. When they were severe there were both glycosuria and salivation associated with fever. The stools were free from excess of fat or from concretions.

In Minnich's case the pain was referred to the left ribs, the epigastrium, and spread to the left shoulder-blade. Moreover, the characteristic pancreatic calculi were found in the stools.

Diagnosis.—This would be based on paroxysmal colicky pains referred to the left rather than the right side and radiating towards the shoulder rather than downward, tenderness on deep pressure in the epigastrium, traces of sugar in the urine, and possibly fatty stools containing calculi after acute attacks.

Prognosis.—This is probably favorable, except in those cases characterized by frequent recurrences and by progressive emaciation.

Treatment.—The general treatment appropriate to hepatic or renal colic is indicated,—*i.e.*, allaying of pain by hot baths, morphine, or even ether. As a curative agent, pilocarpine has been recommended in fairly full doses. It is probable that the best results will be obtained by the use of large quantities of a mildly alkaline natural water, and by careful treatment of the gastro-duodenitis which is frequently associated with this condition.

TUBERCULOSIS OF THE PANCREAS.

While the existence of primary tuberculosis of the pancreas has been questioned, there can be no doubt that this organ is not exempt from initial invasion by Koch's bacilli.

Involvement in conjunction with tuberculosis of other portions of the anatomy is by no means rare. Mondière states that of one hundred tuberculous children, in five only was the pancreas involved. Coudretski, however, in one hundred and twenty-eight cases of tuberculosis, found the pancreas diseased in thirteen. There was no predominance of involvement in either sex. Age, however, seemed to be an important predisposing factor, since over forty-four per cent. of the bodies of children presented tuberculous pancreatitis.

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[1] Münchener medicinische Wochenschrift, May 15, 1894.

Symptoms.—The symptomatology of this affection is necessarily obscure, and will depend upon interference with function, either from pressure or from degeneration.

Diagnosis.—Until a distinct tumor is felt, a positive diagnosis is impossible. The nature of this tumor would then be suggested only by tubercular family history or by lesions of this nature in other parts of the body.

Treatment.—The treatment is that appropriate to tuberculosis in other regions,—*i.e.*, tonic, supporting, climatic, and, if it be possible to establish probable diagnosis, operative. Because of the rarity of these cases, no formal methods of procedure are recognized. On general principles surgical interference should be limited to extirpation of tuberculous foci, leaving as much of the gland as possible.

SYPHILIS.

The lesions of syphilis appear in the pancreas as a gummatous formation or as a diffuse sclerosis. Usually these manifestations of syphilis are associated. They are common in children subject to hereditary syphilis, and may be found in conjunction with similar degenerations of the liver, spleen, and kidneys. Amyloid degeneration is also observed.

Symptoms.—Since syphilitic pancreatitis is not recognized as an isolated lesion, the symptoms of the affection are masked by lesions of other organs, particularly those of the liver and spleen. Slight glycosuria might suggest involvement of the pancreas, but the accompanying gastro-intestinal catarrh and progressive wasting and fatty stools in case of extensive disease might readily be referred to involvement of the liver.

Diagnosis.—This cannot be formulated. Palpation of the pancreas should always be attempted, but, with the exception of one or two reports, this is generally acknowledged to be futile, unless the tumor is of considerable size.

Treatment.—Inunctions of mercury and baths are indicated during infancy, later associated with iodides in case the symptoms do not yield.

Inunctions are given as in the treatment of other manifestations of hereditary syphilis. A child four years of age should receive from thirty to forty grains of an ointment made by mixing blue ointment with an equal quantity of carbolated cosmoline. This should be spread upon a soft muslin cloth, should be applied to the abdomen, and secured in place by a binder, the skin being carefully cleansed and bathed in alcohol once in twenty-four hours. Iron, preferably in the form of Basham's mixture, tonics, digestants, nutritious diet, and general hygiene are also strongly indicated.

CARCINOMA.

Primary carcinoma of the pancreas has been exceptionally observed in young children. Because of the rarity of the disease it is impossible to formulate the symptomatology of the affection in early life, but there is no reason to suppose that the symptoms differ from those observed later. We should then expect, according to Maxson, cachectic countenance, anæmia, irregular or voracious appetite, steady decline and loss of flesh without any apparent satisfactory cause, diminution of the taste and relish of food with little or no preference as to taste, with overwhelming and progressive weakness. Other symptoms considered by Maxson as subsidiary are certainly more characteristic of pancreatic disease than those already given; they are fatty stools, glycosuria, abdominal pain extending from the region of the pancreas to the right shoulder, the development of a palpable, somewhat fixed, often pulsating tumor behind the stomach, nausea, and icterus.

The most typical symptoms in children would probably be the extremely rapid growth of a rather hard tumor in the pancreatic region, glycosuria, fatty stools, jaundice, and pronounced cachexia.

Treatment.—Extirpation is indicated. This, however, holds out little or no hope, since the neighboring lymphatic glands are always early involved.

WOUNDS.

The position of the pancreas is such that when it is lacerated or contused by external violence other more exposed organs are so extensively involved that the symptoms pointing to pancreatic trauma are completely masked. Richardson states that the pancreas has never been found lacerated from abdominal contusion without accompanying rupture of the liver and kidneys. It none the less happens that the pancreatic lesion may be the most important and life-threatening, and the one which most imperatively demands surgical investigation.

Leith's statistics[1] would seem to prove that the pancreas may suffer alone, as in two of his nine fatal cases lesions of other organs are not mentioned.

Symptoms.—The symptoms of trauma of the pancreas differ in no particular from those which characterize injury to other abdominal organs. Death may take place quickly in collapse, or pain and severe shock may be followed by meteorism and the symptoms of internal bleeding, with the formation of an epigastric tumor due to blood effusion into the omental bursa.

Exceptionally symptoms are delayed, as in the case reported by Jordan Lloyd.[2] For a few days following the injury the patient vomited blood-streaked matter and exhibited slight fever; after a period of apparent convalescence there were recurrent paroxysms of epigastric pain and vomiting, followed by the development of a left hypochondriac tumor.

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[1] Edinburgh Medical Journal, 1895–96, vol. xli. p. 423.

[2] British Medical Journal, November 12, 1892.

A second case, also reported by Lloyd, exhibited the typical symptoms of this lesion. A patient having been kneed, suffered severe pain, vomited, and shortly developed a marked tumor occupying the umbilical, epigastric, and left hypochondriac regions. This was tense, rather sharply outlined, dull on percussion, and distinct from the liver and spleen. A medium laparotomy showed that the greater peritoneal cavity was healthy. The omentum was thickened and enormously congested. The omental bursa was filled with blood.

Diagnosis.—In the case of gunshot, punctured, or incised wounds, the direction taken by the vulnerating body, considered together with the position of the patient when wounded, might lead to a correct diagnosis. When the pancreas is crushed or torn without external wound, diagnosis of the injury is usually impossible without an exploratory laparotomy, though even in these cases the nature of the force and the direction in which it was applied may lead to a suspicion as to the injury. When the violence is applied from in front, there is often no external bruising.

Prognosis.—Trauma to the pancreas is often fatal because of associated injury to other organs. Death, as a rule, is due to collapse, often dependent on fatal hemorrhage. Unless the main artery or veins are ruptured, the substance of the organ may be extensively torn without dangerous hemorrhage, and it is well proved that recovery is possible even after severe injury, as instanced by extensive cicatrices found after death due to other causes.

Even after immediate convalescence there is, however, the danger of cyst-formation, Leith having collected seventeen cases in which injuries to the epigastrium were followed by this condition at periods varying from a few days to many years. In nine of the cases the injury was due to falls, in six to blows, in two to being run over by wagons.

Treatment.—Wounds of the pancreas should be treated in accordance with general surgical principles. So far as this gland is concerned, healing after gunshot wounds seems to be prompt and uncomplicated. The main danger of such injuries is dependent upon excessive hemorrhage and associated trauma to neighboring organs.

In case the pancreas is ruptured, surgical intervention would not be required unless the formation of a tumor, particularly one forming in the epigastric region, and the constitutional symptoms point to internal bleeding. Under such circumstances the abdomen should be opened and the bleeding points secured by ligature, the rupture then being closed by suture.

Senn, basing his conclusions upon an experimental study, recommends that the ends of the glands be separately ligated previous to suture. This effectually arrests hemorrhage and the outpour of pancreatic juice. If the ends thus ligated are approximated by catgut sutures, the nutrient vascular supply is quickly re-established. Ruptures thus treated heal quickly and are not followed by cyst-formation, since physiologically separated portions undergo simple atrophy.

DISEASES OF THE LIVER.[1]

BY JOHN H. MUSSER, M.D.

THE diagnosis of disease of the liver in children is more frequently made than the occurrence of cases of actual liver lesion warrants. Jacobi[1] is the authority for the statement that primary affections of the liver are rare, and that enlargement is frequently assumed to exist when the size of the organ is normal. The fact that the liver is much larger in proportion in the child than it is in the adult cannot be too frequently insisted upon. This normal increase in size is exaggerated because the lower portion of the viscus is not covered by the ribs. Ashby and Wright[53] thus outline the upper margin of liver dulness. At the right edge of the sternum the upper limit of superficial hepatic dulness is in the fifth interspace, in the right midclavicular line it is at the upper border of the sixth rib, in the midaxillary line it is at the seventh rib, and posteriorly it is at the ninth rib. The deep area of hepatic dulness always extends a little higher than does the superficial area. The lower edge of superficial hepatic dulness is always below the costal margin.

Rotch[60] shows that in young children the inferior border of liver dulness is midway between the costal margin and the umbilicus. As the child increases in age the lower border of the liver is gradually elevated in position, until at the age of twelve years it is just at the costal margin.

In palpating the liver of older children, Parkins's method may be used to advantage. The patient sits on a chair and leans forward, placing his elbows on a table and his head in his hands. This position throws the organ forward, so that a larger extent of its superior surface than normal comes in relation with the anterior abdominal wall and the examining hand.

Dauchez[52] says the patient should be placed on the left side, the arm should be lifted, the hand fixed upon the bedstead, the left flank supported and elevated by a pillow. Exact vertical measurements of the limits, determined by palpation and percussion, should be made in the axillary and midclavicular lines. Hepatic dulness, anteriorly, is usually one or two centimetres less than the true height of the liver measured in the cadaver. In pathologic conditions there are three degrees of increase: (1) moderate projection beyond the ribs in catarrhal jaundice and gastric disturbance; (2) decided enlargement in malaria and cardiac disease; (3) great enlargement in amyloid disease, hydatid cyst, syphilis, and fatty liver.

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[1] Dr. Musser desires to acknowledge the valuable assistance rendered by Dr. John M. Swan in the preparation of this article. This contribution is to be looked upon as an appendix to the original article. The subject is presented in the following manner in order that the original text be not disturbed.

[illegible] has observed right-sided œdema in four cases of hepatic disease.

[illegible] JAUNDICE—ICTERUS.

Icterus Neonatorum.—The majority of authors unite in dividing icterus neonatorum into (a) mild and (b) grave cases. Baumel,[3] however, classifies the cases as idiopathic or functional and symptomatic or organic.

According to this author, mild icterus is dependent upon difficult labor with long and violent pressure in the abdominal region, or upon the natural debility of the infant.

Many theories have been advanced to account for the production of mild icterus neonatorum. The three of greatest importance are thus given by [illegible].

(1) The condition may be due to the sudden transference of the blood-supply from the umbilical to the portal vein. This change is temporarily followed by the comparative emptiness of the blood-vessels of the liver, a diminution of vascular tension, and the passage of bile into the blood. (Frerichs.)

(2) The pressure from congestion and œdema, due to the arrest of the circulation in the umbilical vein before the establishment of respiration, will cause the absorption of bile into the blood-vessels. (Weber.)

(3) The areolar sheath surrounding the vessels in the transverse fissure, and prolonged with them into the liver-substance, becomes œdematous and swollen from venous obstruction in the liver during difficult parturition, and by pressure obstructs the flow of bile into the intestine. (Birch-Hirschfeld.)

The yellow color of mild cases of icterus neonatorum is often masked by the rosy tint of the new-born, but the presence of bile-stained urine and the colorless stools serve as diagnostic guides.

Among the causes of grave icterus neonatorum may be placed septicæmia, syphilitic [illegible] in the transverse fissure of the liver, congenital malformation or obliteration of the bile-ducts, compression of the bile-ducts by new growths, and parenchymatous lesions of the liver, as in syphilis and cirrhosis.

The cases of grave icterus of a septicæmic nature are dependent upon an umbilical arteritis or phlebitis.

Kynoch[38] reports the case of a female child who died at the age of three months and four days. This child had been jaundiced since birth. She was emaciated, the abdomen was distended, especially on the right side, and the liver was enlarged. The stools were white and milky, the urine was dark and contained bile-pigment. At autopsy the liver was seen to be enlarged and the hepatic and cystic ducts were normal. The common bile-duct, immediately after its formation, was normal in calibre, but its lumen grew rapidly narrower, having a thread-like appearance in the lower part of its course. On pressing the gall-bladder, bile passed easily into the upper part of the duct, but in the narrow portion the progress of the fluid was much slower.

Thomas[8] reports a case in which five successive children of one mother presented icterus neonatorum. In four of the children the jaundice had appeared a few hours after birth, and death had ensued before the ninth day. There were no autopsies in these cases. In the case of the fifth child, which Thomas saw, the jaundice appeared a few hours after birth, and the child died on the twelfth day. At autopsy it was found that the cystic and common bile-ducts were impervious. From the clinical history of the four preceding cases the author concluded that the same condition had existed in them.

A case of compression of the bile-ducts by a new growth is reported by Earle and Weaver,[15] in which a child aged two years had become jaundiced, and simultaneously had developed clay-colored, offensive stools, anæmia, emaciation, loss of strength, and great enlargement of the liver. The gall-bladder was distended. Pressure on adjacent organs by the growth caused vomiting and troublesome dyspnœa. The patient gradually improved, but finally died in a typhoid state. At autopsy the liver was found to be enlarged and cirrhotic. The gall-bladder was enlarged, and the cystic duct was completely obliterated, except for one-quarter of an inch at its upper end. The hepatic duct was pervious. The cystic duct became lost in a dense, firm mass in its lower portion, which was found to be a sarcoma of the right suprarenal body.

Hayem[14] reports the case of a female child aged two months who presented moderate icterus, prostration, marked anæmia, and a hæmophilic condition. At autopsy the gall-bladder was found ruptured near its neck, and the opening was concealed by the new membrane formation of a circumscribed adhesive peritonitis. There were about two hundred and fifty grammes of ascitic fluid mixed with bile in the peritoneal cavity. The liver and bile-ducts were normal. The bile had evidently been absorbed by the peritoneum, producing the jaundice. There were catarrhal lesions of the gall-bladder in the neighborhood of the ulcerated point.

Cases of jaundice in new-born children are attended by the usual symptoms of that condition in adults. In a large majority of cases the liver is enlarged and the hepatic region is tender. Armitage,[4] however, reports a case of grave icterus in a child aged two years and seven months, in which there was no hepatic enlargement or tenderness. Bile was absent from the stools and was present in the urine.

Surmont[70] has shown that the toxicity of the urine in catarrhal or grave jaundice is normal or diminished.

The prognosis of mild cases of jaundice is good, the majority of cases recovering quickly. Donkin,[33] however, reports the case of a child aged two years who had well-marked and increasing jaundice, with delirium, convulsions, and petechial hemorrhages, and points out that the prognosis should be very guarded in all cases of jaundice, especially when the condition continues for more than a week, increasing in severity. Carreau[12] also points out that simple icterus may be benign, but that in the course of a simple icterus grave jaundice (acute yellow atrophy) may supervene and rapidly become fatal.

Grave jaundice, on the other hand, is nearly always fatal, although Baumel and Boiadjieff[31] report a case of recovery from bronzed icterus which was accompanied by subconjunctival hemorrhage. The child had been born prematurely after a hemorrhage, and the jaundice developed on the third day of extra-uterine existence.

Jacobi[1] thus summarizes the treatment of icterus neonatorum. Those cases due to septicæmia in the new-born may be prevented, but cannot be cured. Those cases which result from syphilitic stricture of the bile-ducts may recover, even after months, through mercurial treatment. Those cases which are due to complete obliteration of the bile-ducts cannot be treated.

The sulphate and the salicylate of sodium, administered for months in succession, have answered best as preventives to guard against a repetition of attacks. In the beginning of the attack the bowels should be opened by fractional doses of the mild chloride of mercury.

Carreau[12] is so convinced that benign jaundice may become grave that he directs all his patients who are attacked with yellowness to avoid with care all fatigue and all excesses, such as too sudden change of temperature. He prescribes for them, to this end, the use of flannel garments and woollen stockings. This advice seems to be particularly adaptable to the case of children, who are, as a rule, particularly susceptible to these changes. Carreau uses essence of turpentine in all cases of grave icterus, on account of its diuretic and hæmostatic properties, and because it acts as an oxidizing body, converting the methæmoglobin in the blood into hæmoglobin. Carreau also advises against the use of quinine in jaundice.

Baumel[9] uses honey as a purge, and advises the use of breast-milk to sustain strength. He says that alkalies are rarely indicated or necessary, and that no extra work should be put upon the alimentary tract.

ACUTE YELLOW ATROPHY OF THE LIVER.

This lesion usually develops insidiously. In a patient who has been suffering from catarrhal jaundice the symptoms increase in intensity, the liver becomes enlarged and tender, and vomiting supervenes. The child becomes irritable; delirium, convulsions, and coma develop, and are accompanied by dilated pupils, ecchymoses, oozing of blood from the gums, and œdema of the feet and of the face. After the primary enlargement of the liver that organ will begin to grow smaller and the urine will contain leucin and tyrosin. Fever is usually present, although it may be absent, as in the case reported by Auché and Coyne.[28]

Carreau[12] believes that the change from benign into grave icterus (acute yellow atrophy) occurs only after the hæmoglobin in the blood has become partially converted into acid methæmoglobin. In the absence of a more satisfactory theory, it does not seem to him rash to advance the supposition that a primary alteration of the blood by a cause productive of methæmoglobin is necessary for the production of grave icterus. The blood, primarily adulterated with bile from polycholia due to excess of function of the liver, contains a certain quantity of acid methæmoglobin and of hæmoglobin dissolved in the serum. In the presence of the altered blood it determines the formation of a certain quantity of hæmatin. The hæmoglobin, diminished in quantity and having undergone a certain degree of reduction, does not suffice to oxidize the organic matters. The products of the intermediate transformation of urea appear in the blood, which one finds charged with creatin, xanthin, leucin, and tyrosin. The urea will be in less quantity after the latter formation. Under the same influence the production of fat will be increased, the rarefaction of oxygen in the blood increasing the decomposition of albumin.

Cases of acute yellow atrophy of the liver in childhood are reported by Yeoman[83] (ten months), Merkel[104] (six years), and V. Babes.[113] The primary cause in Babes's cases was an infection by a pyogenic coccus. Marchand[114] reports the histological findings in five cases,—to wit, the presence of minute canals, resembling canaliculi, formed by the subdivision of epithelial cells that have escaped destruction; a reparative process if destruction is not too rapid for proliferation to occur. He believes that various infections, of which syphilis may be one, may give rise to the disease. Meder,[115] commenting on these cases, believes that the essential anatomic change is a fatty degeneration and necrosis of the liver-cells and of some of the epithelial cells of the bile-ducts. In consequence, the latter are destroyed or obstructed, and hence cause jaundice by preventing the escape of bile. The jaundice is not hæmolytic, but hepatogenous. Meder describes the process of regeneration.

In these days of advance in the knowledge of bacteriology it would be strange if a micro-organismal origin were not sought for in this disease.

Auché and Coyne[28] report the case of a little girl aged ten years who was attacked stealthily by grave icterus and who died. The icterus had not been accompanied by fever. Cultures from the blood of the median cephalic vein, made eight hours before death, resulted negatively. At autopsy there were lesions of acute yellow atrophy of the liver and the bacillus coli communis was found in the bile-paths.

Nepveu and Bourdillon[13] have found streptococci in a case of grave icterus, and they quote Klebs as having also found them in a case of acute yellow atrophy of the liver.

It is probable, however, that the micro-organisms are merely associated with and are not the cause of the disease.

Acute yellow atrophy of the liver or acute diffused hepatitis may be due to an intoxication, as in phosphorus or other metallic poisoning, alcoholism possibly, the poisonous products of the intestinal tracts (ptomaines), the poisons which result from bacterial invasion or the micro-organism itself. It is not necessary to invoke a specific micro-organism. The writer recently saw a case of hypertrophic cirrhosis of the liver take on the symptoms of acute yellow atrophy and die after a brief illness. The autopsy showed that there was present an acute infectious endocarditis, the causal agency of which no doubt produced the acute hepatitis also.

In the beginning of the disease it is impossible to say whether the case is one of catarrhal jaundice or of acute yellow atrophy; but the progress of the latter lesion is rapid, and the true nature of the trouble is soon apparent to the diagnostician. The development of the nervous symptoms in cases of acute yellow atrophy is very likely to mask the correct diagnosis, so closely does the succession of events resemble the progress of tuberculous meningitis. The decrease in the size of the liver and the tenderness over that organ, together with the appearance of leucin and tyrosin in the urine, ought to serve to distinguish between the two conditions.

The disease is always fatal.

Treatment is of no avail. The general measures should be carried out that are in vogue in all severe illnesses. Essence of turpentine, as advocated by Carreau,[12] may be tried in doses of two or three drops every four hours.

II. AFFECTIONS OF THE BLOOD-VESSELS OF THE LIVER.

1. *Congestion of the Liver.*—Congestion of the liver may be either active or passive. Starr[67] points out that habitual overfeeding and the use of too highly seasoned foods, together with insufficient exercise, may induce active hyperæmia of the liver. Chills from exposure to cold or from malarial poisoning may produce the same condition, and Jacobi[1] adds infectious fevers and very high temperatures to the list of etiologic factors. The first-mentioned cause will probably be found to be more frequently operative in the children of the rich than in those of the poor.

The symptoms of this condition are at first not particularly characteristic, consisting of malaise, headache, yellow, furred tongue, anorexia, and nausea. The skin soon becomes sallow or jaundiced, and this is accompanied by relaxed bowels with clay-colored, offensive stools, and by the passage of dark-colored urine which is loaded with lithates. The child assumes a dorsal decubitus because there is pain in the right hypochondrium, which is increased by turning on either side. There is tenderness over the liver, which is aggravated by coughing or by deep breathing. The right lobe of the liver is palpable, and the gall-bladder is frequently distended into a pyriform tumor of variable size. The upper limit of hepatic dulness is frequently as high as the third interspace or the third rib.

This condition is to be diagnosed from disorders of the intestinal tract which are associated with the expulsion of putty-like stools. Enlargement of the liver is necessary before the diagnosis of congestion can be established, and this enlargement must be accompanied by pain and tenderness. (Starr.[67])

Passive congestion of the liver may be and usually is due to cardiac disease, but pulmonary disease may also act as an important factor in the production of this condition. Frequently we have seen, particularly in adults, it is true, cases of pneumonia treated for congestion of the liver because of the onset of the affection with jaundice and other hepatic symptoms. Jacobi[1] has shown that diaphragmatic pleuritis may so constrict the inferior vena cava that hypertrophy of the liver, ascites, and death may result; he also states that pneumonia, in its early stage, impedes the hepatic circulation temporarily and may thus induce passive congestion.

Treatment.—In cases of active congestion of the liver an initial dose of calomel combined with powdered ipecac is useful. This may be followed by the use of the chloride of ammonium, in doses of from one to five grains, to be given after meals. Vichy water may be used, as recommended by Starr.[67]

In cases of passive hyperæmia of the liver one should treat the cause, and use calomel in divided doses frequently repeated.

2. *Suppurative Pylephlebitis.*—An important addition to the literature of suppurative pylephlebitis in children has been made by Maygrier and Chaillou.[11] These authors report the case of an infant aged fourteen days who was seized with fever, convulsions, tender abdomen, and cyanosis. The child died in a convulsion four days after the onset of the symptoms. At autopsy the mesenteric vein was injected, and, on section, exuded an abundant pus, which distended it. The portal vein and its intra-hepatic ramifications were also filled with pus, which flowed back into the patulous portion of the umbilical vein. In the interior of the liver the first branches of the portal vein had a wall fully five millimetres thick. There were metastatic abscesses in the brain and spinal cord.

This child had nursed until the mother was taken with a lymphangitis of the right breast. This inflammation of the lymphatics involved only three or four glandular lobules. The day following the beginning of this infection the baby began to lose weight and refused to nurse. The mother then milked her own breast and fed the milk to the child, and this milk was taken by the child until its death. The micro-organisms had evidently found their way into the portal vein from the digestive tract and had been introduced there with the mother's milk. No cultures of the pus were made.

The authors give the very pertinent advice that in all microbic affections of the breast, howsoever small, the feeding should be suspended until after complete cure.

III. AFFECTIONS OF THE BILE-PASSAGES.

1. *Congenital Obliteration of the Bile-Ducts.*—Thomson[21] reports one case of congenital obliteration of the bile-ducts, and tabulates forty-nine others obtained from the literature. His work is exhaustive, and we give his most important conclusions concerning the pathology and the symptoms of the lesion.

Etiology and Pathology.—In the great majority, if not in all, of the cases there is, to begin with, a congenital malformation of the bile-ducts, due in some way to defective development. This malformation probably affects a considerable extent of the walls of the ducts, and may consist in narrowness of their lumen. The interference with the outflow of bile thus caused gives rise to catarrhal inflammation, and finally to blocking and obliteration of the ducts, owing to the inflammatory process spreading to the walls of the ducts and of the gall-bladder. This progressive inflammation goes on, slowly spreading, the local condition getting gradually worse during many months, if the patient lives. The obliterated ducts or gall-bladder, or portions of them, may entirely disappear, not even leaving a distinct band of fibrous tissue to indicate their original position. The obliteration generally becomes complete at an early, but variable, period of intra-uterine life, but occasionally this obliteration does not occur until after birth. In a few cases the inflammatory process spreads to the peritoneum, and possibly the presence of inherited syphilis may favor this extension. The occurrence of peritonitis is probably always secondary to the blocking of the ducts, and syphilis has nothing to do with the original lesion in them. When the lumen of the duct is so far encroached upon as to obstruct the free passage of bile into the intestine, biliary cirrhosis begins, which, as it advances, causes increasing interference with the most important functions of that important organ. The production of a kind of blood-poisoning is the result of this condition, and upon it depends directly the vomiting, spontaneous hemorrhage, and convulsions. Emaciation, diminished vitality, and death ultimately result.

Symptoms.—The occurrence of congenital obliteration of the bile-ducts produces primarily a jaundice, which begins at or within a few days after birth. The jaundice is extremely deep, greenish, and grows steadily worse. (See Icterus Neonatorum, page 790.) The urine usually contains bile-pigment, one case in Thomson's fifty not showing this symptom. The meconium is sometimes normal in appearance and is sometimes colorless. The bowel movements are white, gray, or cream-colored. Sometimes, late in the disease, the child will have one or two colored passages from the bowel. Vomiting and spontaneous hemorrhages rapidly develop, and are followed by convulsions and emaciation. Infants thus affected live for a few days only, as a rule. One case is recorded in which the child lived eight months. The disease is liable to appear in several members of the same family. (See case of Thomas, page 791.) The fact that the onset of the jaundice is not at all contemporaneous with the blocking of the bile-ducts, and usually begins several days after birth, is best explained by taking into account the effect on the liver-cells exerted by the enormous changes in the hepatic circulation which occur at birth. The presence of colored meconium in some cases and of nothing but white discharge in others is due to the blocking of the ducts having occurred at different periods of intra-uterine life. When green matter is passed along with the colorless motions during the progress of the case, the chemical action of the contents of the bowel on mercury which has been administered may be looked upon as the cause. The tendency to spontaneous hemorrhage is probably due to the occurrence of a state of chronic blood-poisoning, the arrest of the outflow of bile so damaging the liver that its functions are interfered with, and it allows organic products of a poisonous nature to pass into the circulation. The enlargement of the spleen, the convulsions, and the vomiting are probably more or less connected with this same condition of blood-poisoning. The fact that children live as long as they do, and do not usually become emaciated early, is to be explained by the theory that the presence of bile in the bowel is not very necessary for digestion. When the nutrition and general health do begin to suffer, it is probably due to the interference which the secondary changes in the liver-tissue are causing with the more important functions of that organ.

The lesion is fatal, and no treatment can possibly bring relief.

Eshner[110] reported the case of a child aged two years, in whom the gall-bladder was absent.

2. *Catarrhal Jaundice.*—Catarrhal jaundice in children is due to the same etiological factors that determine the disease in the adult. The most common cause is the extension of inflammation from the stomach and duodenum along the common bile-duct. Carpenter and Syers[18] report twelve cases of this affection in children, four of which began with evidences of gastric catarrh. Walker[17] reports an epidemic of catarrhal jaundice occurring in children and youths. Twelve cases occurred in four families.

3. *Cholelithiasis.*—Cases of gall-stones occurring in children are excessively rare. We have been able to find but two cases of the affection. Lieutaud[36] reports the case of a child aged twenty-five days who died from the obstruction caused by the lodgement of a biliary calculus in the ampulla common to the common bile-duct and to the pancreatic duct. Basinger[26] reports the case of a man who had been jaundiced from birth, and who had had attacks of colic from childhood. This man died after an attack of hepatic colic, and at autopsy numerous gall-stones were found in the gall-bladder. The author assumes that the stones were present at birth.

It is not, theoretically, impossible for gall-stones to appear in children, especially if there be aught of practical value in Naunyn's theory[79] that the bacillus coli communis is the factor which causes the precipitation of the bile to form calculi.

Treatment.—The treatment of this condition must remain largely hypothetical in the absence of more cases of actual experience. Cassael[102] and Strizover[103] recommend salicylate of sodium in this affection. The latter author commends salol. In case a stone should become arrested in the common bile-duct or, as in Lieutaud's case, in the ampulla of Vater, a cholelithotomy might be done. This operation, however, has probably never been performed on a patient in early life. In seventy-eight cases of gall-bladder and bile-duct surgery reported by Robson[32] the youngest patient was eighteen years of age.

IV. CIRRHOSIS OF THE LIVER.

Cirrhosis of the liver may occur in one of two forms: first, atrophic, and second, hypertrophic or biliary. To these two forms Gibbons[65] has added a third variety of cirrhosis of the liver, which will be briefly described later.

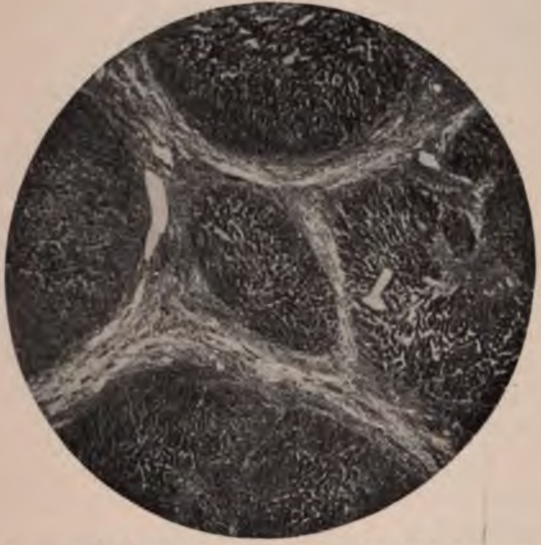
Frequency.—The opinion was advanced in vol. iii. p. 489 of this Cyclopædia "that hepatic cirrhosis is less uncommon with children than is generally thought." The author of this section, Dr. Hatfield, reports one hundred and fifty-six cases obtained from various sources. We can now add to this number five hundred and twenty-nine cases which have been reported since, and we are sure that the list is incomplete. The statement as to the frequency of this lesion in children can unhesitatingly be confirmed. The disease is much more frequent in warm than in temperate climes. Four hundred of the above cases are reported by Ghosh.[73]

Blagowjechtschenski[106] reports a case of atrophic cirrhosis in a boy aged ten years, D'Espine[92] a case in a boy aged six, and Marchand[107] reports a case of alcoholic cirrhosis in a child aged seven years.

Age and Sex.—In papers by W. A. Edwards,[3, 74] in which he tabulates one hundred cases of the disease, the average age at which the lesion has been observed is given as between the ninth and twelfth years. In the same papers boys are asserted to be more often affected than are girls. The author also states that the atrophic form is the more common.

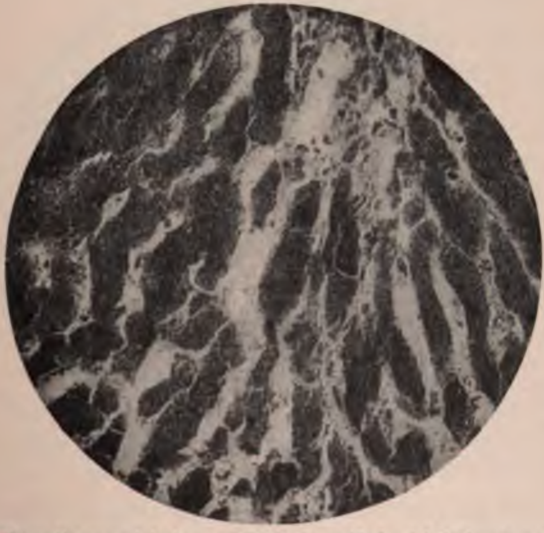
Etiology.—The disease may be due to alcohol, syphilis, tuberculosis, rachitis, malaria, congenital obstruction of the bile-canals, chronic heart disease, disordered digestion forming ptomaines (Gibbons,[47] Bose,[48] Clarke,[52] and Ghosh[73]), duodenitis, burns of the abdomen and the influences of cold and of traumatism (Grisey, quoted by Edwards[3]), and the eruptive fevers (W. A. Edwards[3] and Blanche Edwards[64]). The theory of Gibbons,[47] Bose,[48] Clarke,[52] and Ghosh[73] supposes that the food, instead of passing through the normal chemical changes in the digestive tract, undergoes abnormal fermentation or decomposition with the formation of ptomaines. These ptomaines are then carried to the liver through the portal circulation, and by their irritation produce the lesion. It is possible that this method particularly influences the production of the hypertrophic form of cirrhosis, since it is from hot countries in which the hypertrophic variety is more common that this opinion comes.

Fig. 1.



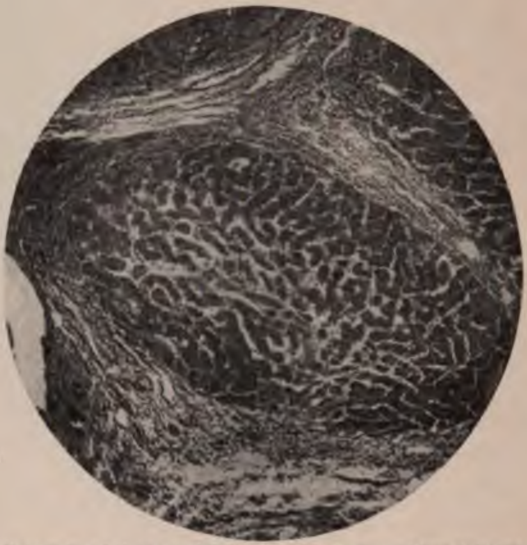
Shows lobule surrounded by the enormously increased interlobular connective tissue. (× 35 diameters.)

Fig. 2.



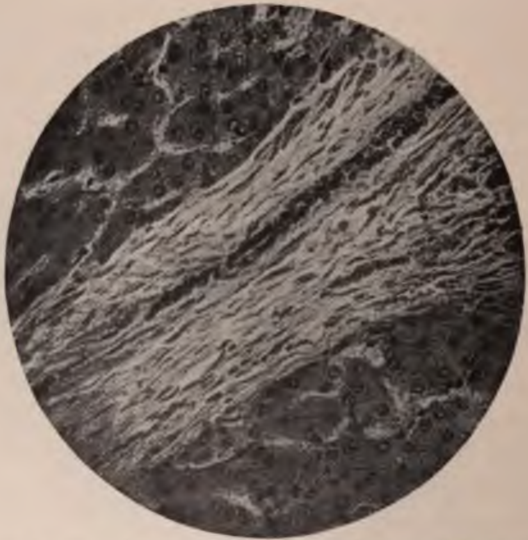
Shows the same lobule more highly magnified. (× 75 diameters.)

FIG. 3.



Shows the centre of the same lobule, showing increased intralobular tissue.

FIG. 4.



Shows the edge of the same lobule, showing the dense character of the increased tissue.

De Giovanni,[84] in a study of many cases, believes that a predisposition exists in the lymphoid structures, particularly of scrofulous or tuberculous subjects.

There is much difference of opinion as to the *rôle* played by alcohol in the production of this condition. W. A. Edwards[3] believes that alcohol holds a minor position as a causative agent, and considers that more importance is due to the influence of the infectious fevers as an etiologic factor. Cases without alcoholic history have been reported by Abbé,[71] O'Carroll,[38] and Hatfield.[2] Stack,[72] after examining the autopsy records of nine thousand cases, selects twenty which undoubtedly present that condition of the liver which is to be seen in the spirit-drinking adult. Stack does not believe that alcohol caused the disease in these twenty cases for three reasons: first, because in only ten of the cases was that substance mentioned; second, because the poor recognize that spirit-drinking is not good for any one, much less for a child; and, third, because the children were too young. On the other hand, cases in which there had been a distinct history of alcoholic habits, either in the parents of the patients or in the patients themselves, are reported by Weber,[35, 39] Mirinescu,[41] Foxwell,[37] Biggs,[63] and Sainsbury.[57] Recently, Goriatchkine[80] has shown that out of 1679 children (841 boys and 838 girls) who were in attendance at Saint Olga's Hospital, Moscow, 506 (282 boys and 224 girls) were in the habit of taking alcohol in some form either as a result of their environment or upon the advice of a physician. In many cases the alcohol was given in the form of alcoholic infusions of various drugs. It would seem, therefore, that while the relation between the acute infectious diseases and hepatic cirrhosis might be that of cause and effect, still the influence of alcohol as an etiologic factor should not be lost sight of or relegated to a position of secondary importance.

It has been observed by the writer in several instances that cirrhosis of the liver is much more liable to occur in a child who is taking alcohol moderately, if the patient is the subject of organic heart disease with or without failing compensation. Failing compensation renders the use of alcohol particularly hazardous, and we should be very chary in its use under these circumstances.

Pathology.—In the atrophic form the increase of connective tissue which characterizes the lesion takes place at the periphery of the lobules, begins around the radicles of the portal vein, and has a tendency to contract. The early stage of this process following an attack of one of the acute infectious diseases is thus described by W. A. Edwards,[3] and graphically illustrated in the accompanying photo-micrographs. The liver is slightly enlarged and presents a variable color, which is grayish on section, often irregular in outline, sometimes of a nutmeg character, and not infrequently presenting a marked fatty change. The liver-cells are infiltrated with fat, which may be uniform in its distribution throughout the lobule or may be limited to the periphery. The intra-lobular capillaries are usually dilated, the connective tissue is much more abundant than normal, the portal veins are dilated and their connective tissue is proliferated. The tunics of the arteries and veins are much thickened. The biliary canals are dilated and are more apparent than normal. The newly formed connective tissue seems to form pseudo canals; the connective tissue formation on the periphery grows centripetally and meets a little islet of embryonic connective tissue in the centre of the lobule, and, uniting around the canals, constitutes a true example of interstitial hepatitis. There is never any modification of the endothelium in the blood-capillaries of the liver.

In the hypertrophic form of hepatic cirrhosis the bile-ducts show the beginnings of the fibroid change in their walls. The new-formed connective tissue may invade the lobules of the liver and lie between the individual cells, but it is usually found only at the periphery of the lobules. The biliary canaliculi undergo proliferation, and the hepatic cells begin to show fatty change and to degenerate.

In some cases areas of cerebral softening may be found accompanying the cirrhotic condition of the liver. This lesion may account for the nervous symptoms seen just before death. Such a case is reported by Ormerod.[68]

It is interesting to note that Senator[105] classifies the different forms of this disease as follows:

I. Laennec's portal granular atrophy of the liver; atrophy of the liver with uneven surface; ascites; periphlebitis of the portal vein; tumor of the spleen; no icterus. A variety of this form may show hypertrophy of the liver,—rare cases in which the fatty infiltration causes swelling. Another variety would be cases in which jaundice occurs, due to accidental obstruction of the biliary duct (inflammation or gastro-intestinal catarrh).

II. Biliary cirrhosis with consecutive atrophy. This form would originate from and be characterized by chronic obstruction of the biliary duct by stones, etc. It may be associated with swelling of the spleen (cause doubtful).

III. Hanot's hypertrophic cirrhosis with icterus. There would still remain a small number of cases that could be classified only as chronic interstitial hepatitis, with or without chronic splenitis,—cases that do not belong strictly to any of the three classes mentioned.

Symptoms.—The symptoms of the atrophic form of cirrhosis of the liver may be enumerated: first, as gastro-intestinal symptoms; second, as symptoms referable to the liver; and, third, as general symptoms. The gastro-intestinal symptoms consist of digestive troubles, abdominal pain which is slightly augmented by pressure, vomiting, and alternate attacks of constipation and diarrhœa.

The symptoms referable to the liver are: jaundice, contraction of the liver, dilatation of the subcutaneous veins of the abdomen, hemorrhoids, and ascites. The ascites may develop slowly or suddenly, and without accompanying anasarca, as in cases reported by Tidey[60] and Hall.[58] In Tidey's case the sudden development of the ascites was due to the presence of two enlarged lymph-nodes at the transverse fissure of the liver, which pressed upon the portal vein, effectually obstructing the flow of blood through it.

The general symptoms are: emaciation, venous stigmata, the occurrence of hemorrhages from the various mucous membranes and beneath the skin, and bronchitis; of these, epistaxis is probably the most common. The heart may not be affected. The blood is usually negative. The urine may be normal or it may contain albumin. The spleen will usually be found to be enlarged. Fever may be irregular or it may be absent (Tidey[60]). According to Surmont,[70] the toxicity of the urine is increased.

Just before death we may observe the development of toxæmia, peritonitis, nervous disturbances, pleuritis, congestion of the lungs, pneumonia, ulceration of the colon, tuberculous meningitis, and profuse hemorrhages.

The symptoms of the hypertrophic variety of hepatic cirrhosis are essentially the same as those of the atrophic form of the disease, except that the liver is larger instead of smaller than normal. The toxicity of the urine is sometimes normal and sometimes increased (Surmont[70]). Cases are reported by Hatfield,[2] Gilbert and Fournier,[23] C. W. Smith,[25] and F. J. Smith.[43]

Diagnosis.—The diagnosis of atrophic cirrhosis of the liver should present no marked difficulty. Hypertrophic cirrhosis may be confounded with amyloid liver or with enlargement due to malaria. Ghosh[40] has tabulated the differences as follows:

	HYPERTROPHIC CIRRHOSIS.	AMYLOID LIVER.
Age	Under two and a half years.	Rare.
Cause	Faulty diet.	Chronic disease.
Enlargement	Left lobe first.	Uniform.
Progress	Rapid.	Slow.
Edge	Thin.	Rounded.
Increase	Downward.	Both upward and downward.
Anæmia	Slight.	Marked.
Cachexia	None.	Marked.
Jaundice	Common.	Rare.
Complications	Spleen enlarged.	Spleen, kidneys, stomach, and intestines involved.
Duration	Short.	Long.
Anatomical character	Disorganization of the liver-cells from the pressure of newly formed fibrous tissue surrounding the hepatic cells. The bile-ducts are primarily affected.	Characteristic amyloid reaction. Walls of lobular capillaries and arteries affected.

	HYPERTROPHIC CIRRHOSIS.	MALARIAL HYPERTROPHY.
Age	Two and a half years.	At all ages.
Sex	Males more than females.	Males and females alike.
Habitat	All districts.	Malarious districts.
Number	Increases yearly.	Increases where malaria prevails.
Climate and hygiene	Little influence.	Much influence.
Premonitory symptoms	Nausea and anorexia.	Fever.

	HYPERTROPHIC CIRRHOSIS.	MALARIAL HYPERTROPHY.
Countenance	Healthy.	Sickly.
Invasion	Insidious.	Sudden.
Fever	Slight in the beginning; higher when jaundice is present.	Persistent from the beginning.
Enlargement	Rapid.	Slow.
Pain	Absent at first; slight when jaundice appears.	Painful on pressure.
Edge	Well defined; becomes rounded.	Never rounded.
Feel	Hard, tense, firm, and resistant.	Soft.
Lobe affected	Left first.	Right first, or uniform.
Constipation	Marked.	Absent.
Stools	Yellow or muddy white.	Muddy, not white.
Urine	Clear, then bile-stained and saffron color; no albumin.	Not so deeply colored.
Œdema	Varies; commences in hands and feet.	Commences with anæmia and ascites.
Ascites	Partial.	Complete when it occurs.
Jaundice	Varies.	Not in all cases.
Spleen	Slightly enlarged.	Considerably enlarged.
Cachexia	None.	Malarial.
Complications	Slight enlargement of spleen, ascites, jaundice, and cholæmia.	Great enlargement of spleen; complete ascites; jaundice and cholæmia rare; liver abscess, diarrhœa, dysentery, lung disease, etc.
Duration	From two to twelve months.	Longer.
Contraction	Late, if at all; sometimes rapid.	None.
Prognosis	Unfavorable.	Favorable.
Death	From jaundice and cholæmia.	From exhaustion.
Etiology	Erroneous diet.	Malaria.
Anatomical characteristics	Disorganization of liver cells, due to pressure of bands of new fibrous tissue surrounding the hepatic cells and impeding the flow of bile.	Portal vein primarily affected, but not so as to obstruct the portal circulation absolutely. The impediment is enough to produce ascites.

Prognosis.—The prognosis is bad. The atrophic cases run much the same course as the atrophic cirrhosis seen in the adult. Millard[86] and Fremont[87] each report a case alleged to be cured.

The cases of hypertrophic cirrhosis gradually perish of toxæmia and emaciation. Out of four hundred cases reported by Ghosh[73] only six recovered. Fremont[87] and Stor[93] report cures of the hypertrophic variety. It must be remembered that the symptoms of cirrhosis may remain in abeyance for a long time, or may disappear temporarily. All cases of so-called cures must be accepted with reserve.

Treatment.—As a prophylactic measure it would be well to avoid giving children medicines in the form of alcoholic tincture or wine. As an additional preventive measure all stimulating food should be avoided, an the diet should consist largely of milk. Rendu[88] advocates milk di strongly.

Mercury with chalk, the syrup of the iodide of iron, and salines m be used (Rolleston[75]). Ghosh[73] lays most stress on a complete change

and says that no benefit can be expected from mercury, iodide of po-
ım, chloride of ammonium, or phosphate of sodium. Bose[48] advises
ıse of euonymin in doses of one-quarter of a grain, combined with
ered ipecac and the bicarbonate of sodium. Our views of treatment
ide with those of Ghosh, unless there is a syphilitic origin for the
ion. Millard believed that his case was cured by milk diet, iodide of
sium, purgatives, and diuretics; Fremont, that his cases were cured by
ınd alkaline waters. Lauenstein[116] observed two cases cured by tap-
Gheorgievsky[85] recommends the balsam and resin of copaiba.

n 1891 Gibbons[65] described a fatal disease of the liver which attacks
g children. This disease, which is a cirrhotic affection, differs from
ırdinary form of biliary cirrhosis in that the fibrous tissue is formed
n the lobules, between the hepatic cells. The multiplication of the
lucts is one of the most striking features of the disease.

'he affection is common among well-to-do Hindoos and Mohammedans;
s never been seen in European or Eurasian children. It is usually
and runs its course in three or four months. The onset of the malady
sidious, and is unmarked by symptoms likely to attract the attention
e parents.

ymptoms.—There is painless enlargement of the liver, accompanied
nguor, fretfulness, and the development of a voracious appetite; this
manifestation soon gives place to a refusal of food. There is marked
t; fever is usually absent, except in cases which are complicated by an
ged spleen. The spleen, however, does become enlarged in the ma-
y of cases. Constipation, with the passage of white, clayey stools, is
l. The liver reaches a huge size and subsequently undergoes contrac-
and, with this change in the dimension of the organ, jaundice appears.
tes develops, being ushered in by œdema of the feet, and then death
s with symptoms of cholæmia.

'he disease is considered to be due to the irritation produced by the
ucts of faulty digestion.

V. ABSCESS OF THE LIVER.

Ve are able to add five cases of this affection, which have been reported
nglish and French literature, to the thirty-four published in vol. iii.
6 of this Cyclopædia.

tiology.—Abscess of the liver may be either single or multiple. The
iple abscesses are usually metastatic in origin, and arise from suppura-
in another part of the body, notably subcutaneous abscesses. Single
sses are the result of ulceration of the bile-passages or of pylephlebitis
nois and Lyonnet[29]).

Ionnier[89] reports a fatal case of multiple abscess with portal phlebitis,
ıbly originating in ulceration of the appendix. The patient was a boy
twelve years.

'his lesion may be due also to traumatism, lumbricoid worms, dysentery, tropical diarrhœa, typhoid fever (Lannois and Lyonnet[29]), umbilical phlebitis, perityphlitis (Leblond[50]), and tuberculous disease (Godlee,[5] Pépin[90]).

Pathology.—The pus of hepatic abscess is usually composed of fragments of hepatic cells, which are often infiltrated with fat; numerous leucocytes, which are more or less altered; granular bodies; red corpuscles, which are sometimes broken down; and micro-organisms. It may be stained with bile, and instances have been reported of the finding of lumbricoid worms in the pus. The organisms which have been isolated are diplococci, amœba coli (Slaughter[59]), the pyogenic organisms, bacterium coli communis (Leblond[50]), and the bacillus typhosus (Lannois and Lyonnet[29]). The pus is sometimes sterile, and Leblond[50] thinks that the bactericidal action of the bile may account for this sterility. On the other hand, Arnaud and Astros[62] have mixed a pure culture of the diplococcus obtained from the pus of a liver abscess with pure bile from a dog, and have found the resulting growth to be abundant. This experiment disproves the theory that mere mixture of bile with micro-organisms destroys the latter. It is more probable that the micro-organisms exhaust themselves in the formation of the pus, thus explaining the sterility of certain cases.

Zancarol[94] concludes as follows on the pathology of hepatic abscess: 1. Abscess of the liver is a micro-organismal disease. 2. The principal factor is a streptococcus. 3. Dysentery is produced by the streptococcus; the amœbæ play no *rôle* in the pathogenesis of the affection. 4. The point of entrance of micro-organisms found in the liver is chiefly the intestinal tract, whence they pass to the liver either with the portal blood or the general circulation.

Loison and Arnaud[95] conclude as follows in regard to the etiology of the trouble: 1. Abscesses of the liver are micro-organismal in origin. 2. They may be monomicrobic or polymicrobic. 3. They develop in the course of diverse infections of the intestinal canal. 4. They are usually pyocholic in origin, rarely pyæmic. 5. They should be opened and drained as soon as recognized.

Godlee,[5] who reports a case of abscess of the liver in a child aged nine years who had tuberculosis of the hip-joint, thus explains the pathogenesis of abscess from such a cause. Septic clots escape into the venous circulation from softening thrombi in the veins at the seat of the primary mischief and lodge in the liver. Such emboli may reach the liver either by the hepatic artery or the portal vein. In an infant these emboli may reach the liver through the umbilical vein.

Symptoms.—In the development of a liver abscess the child will complain suddenly of a sharp pain in the region of the liver, with radiation to the shoulder, tenderness on pressure, and a sense of weight in the epigastrium. Chills, accompanied by fever of a remittent type and by vomiting, develop. The appetite is lost, and there may be diarrhœic stools. The liver increases uniformly in size either towards the thorax or down into the abdomen. The ribs project and a tumor appears, which is dull on percussion, which may become fluctuant, and which is characterized by the appearance of œdema of the overlying skin and by the development of a net-work of superficial veins.

Godlee[7] draws attention to the fact that when an acute enlargement of the liver is caused by the development of an abscess in the interior, the edge of the organ may become so rounded that room is left for the encroachment of the intestines upon its anterior surface. Under such circumstances the extent of dulness by no means corresponds to the size of the tumor, the shape of which may be so unlike that of the liver as to lead to considerable confusion in diagnosis.

As soon as an abscess is suspected, the exploring-needle should be used to establish the diagnosis. Even after the exploring-needle has been used, there may be some difficulty in determining the exact nature of the case, particularly if the abscess is on the upper surface of the liver. Taylor[55] reports a case in which a liver abscess had pushed up the diaphragm to such an extent that the aspirator revealed pus so high in the thorax that the abscess was thought to have ruptured, filling the pleura with its contents. Subsequent events showed that the abscess had not previously ruptured.

According to Godlee,[5] and as previously pointed out, an hepatic abscess may rupture externally through the parietes, usually in the epigastric region, rarely in the axilla. It may rupture into the stomach, into the intestine, into the pelvis of the right kidney, into the lung, into the pleura, into the pericardium, or into the peritoneum.

Diagnosis.—Abscess of the liver has been confounded with hydatid cyst of the liver, cancer of the liver or of the stomach, suppuration of a neighboring organ, subdiaphragmatic abscess, pneumonia, chronic pulmonary tuberculosis, subhepatic collection of pus, purulent diaphragmatic pleurisy, febrile gastritis with congestion of the liver, renal abscess and calculous pyelitis, aneurism of the abdominal aorta, and typhoid fever.

From purulent diaphragmatic pleurisy an abscess of the liver may be told by the previous history of the case; by the more sudden development, the more intense pain, the more sudden and more marked dyspnœa, the existence of the diaphragmatic button and of the scalenic point, the obliquity of the lower ribs, and the form of the upper border of dulness, which, in the former, has the same outline as the diaphragm. The paroxysms of fever are less remittent in the former, and the symptoms are more irregular and generally more grave in character. When the abscess is punctured, if it is a purulent diaphragmatic pleurisy, the flow of pus will be affected by the respirations; if it is a liver abscess, there will be no fluctuation corresponding to the respiratory movements.

From febrile gastritis with congestion of the liver, hepatic abscess is to be told by the character of the fever, which in the former is of an intermittent nature.

Examination of the urine will serve to distinguish hepatic abscess from abscess of the kidney or from calculous pyelitis. Much important information concerning the diagnosis of this affection has been obtained from the paper of Leblond.[50]

Tschernow,[117] in discussing three cases of suppurative hepatitis in children, comments on the infrequency of the affection in early life, and on the similarity of the symptoms to those in the adult. The disease may be absolutely latent and insidious, and the author has seen cases in children where the disease entirely escaped recognition or was mistaken for some other affection. The disease may be mistaken for tuberculous peritonitis, tuberculosis, and typhoid fever. The exaggerated convexity of the right hypochondrium, the dilatation of the base of the thorax at the right side, and the accompanying œdema of the hepatic region must be borne in mind, and may suffice for the diagnosis if they are associated with the febrile phenomena that occur during the development of all purulent foci. A special characteristic, particularly in children, is, that in rare instances there may be no local pain. When the abscess has attained a certain size, the increased convexity of the dome of the diaphragm on the right side and the diminished amplitude of the movements of the liver during respiration may become apparent; but these and other symptoms may be modified by the complication of a right pleurisy. Care must be taken not to mistake the affection for pleurisy.

Prognosis.—In case of a single abscess of the liver the prognosis is good if the proper operative interference is adopted sufficiently early. In the case of multiple abscesses pyæmia is liable to follow, and the prognosis is therefore grave. This fact is mentioned by Gerster,[56] who also indicates that this severe character of the disease is prone to occur when it depends upon chronic intestinal affections, especially of an ulcerative nature, for its cause.

Treatment.—There is no doubt that, after the diagnosis of abscess of the liver is made, the only treatment to be employed is the evacuation of the collection of pus. The question to be settled concerns the method which should be employed. Little advises the surgeon to open the abscess in the same manner that he would open an abscess on the surface of the body, passing through abdominal wall, peritoneum, and liver by one free incision. After the pus is evacuated, irrigation, double drainage, and antiseptic dressing complete the procedure. This method is recommended by Clarac.[51] On the other hand, Leblond,[50] who quotes Little's method, properly shows that it is devoid of danger only if adhesions have formed between the abscess and the abdominal wall. We cannot always be certain that adhesions have formed, and we are also sure that, while the pus contained in an abscess of the liver is sometimes sterile, it is not always so. For these two very obvious reasons the method is not to be recommended. In opening an abscess of the liver, then, the abdominal wall should be incised layer by layer; the liver should be sutured to the peritoneum firmly enough to preclude all possibility of pus entering the general peritoneal cavity. Then the abscess should be opened, irrigated, and drained. This method is advocated by Fontan,[30] Leblond,[50] and Gerster.[56] Fontan[30] goes further, and recommends that the abscess-cavity be thoroughly curetted before the drainage is introduced and the wound dressed. By this procedure Fontan records 86.66 per cent. of successes.

If the abscess projects into the pleural instead of into the abdominal cavity, the pleura should be sutured to the skin, and, if necessary, one or more ribs should be resected to allow free egress of pus, to prevent the occurrence of necrosis of those bones, and to prevent the formation of fistula.

In exceptional cases, in which the abscess is central and near the large intestine, it is possible, by a cœliotomy, to perform an hepato colotomy, anastomosing the abscess-cavity and the colon (Leblond[50]).

If the abscess ruptures into one of the neighboring organs, it should still be opened and drained, and further treatment should be adopted in accord with the organ invaded by the pus from the liver. A perinephritic abscess should be opened, pus should be drained from the pleura or the pericardium, and an abscess of the lung should be incised (Leblond[50]).

As prophylactic measures, Jacobi[1] advises the disinfection of a dysenteric rectum by frequent enemata, the early incision of perityphlitic or pleural abscess, and the prevention of umbilical phlebitis by aseptic dressing of the umbilical cord.

VI. NEW GROWTH IN THE LIVER.

1. *Carcinoma, Sarcoma, etc.*—We are able to add four cases of new growth of the liver to the twenty-three already recorded (vol. iii. p. 456 of this Cyclopædia). Nothing new has been adduced concerning symptomatology or diagnosis.

CASE XXIV.—Descroizilles[42] reports the case of a little girl aged eleven whose father was alcoholic. The patient was emaciated and anæmic, and had an enormous appetite. The bowels were regular. There was no vomiting at the beginning of the case. Pain was present and became violent, particularly in the region of the liver, and then constipation and bilious vomiting developed, accompanied by tympany and cramps in the legs. There was no fever or chill or cyanosis. Palpation of the abdomen revealed a hard, smooth, elastic, and dull mass, which was connected with the liver. There was no jaundice or alteration of the pulse. The urine contained albumin. Autopsy showed a carcinoma of the liver, which was secondary to cancer of the small intestine and of the neighboring lymphatics.

CASE XXV.—Axtell[49] reports the case of a child aged three and a half years who presented a swelling in the epigastrium, which was first noticed after a fit of anger. Ascites, cough, loss of weight and strength, and poor appetite were among the symptoms. The tumor was hard, round, and smooth, and was dull on percussion. It was not movable, and presented neither pulsation, thrill, nor fluctuation. The superficial abdominal veins were distended. The urine contained no albumin, but presented a slight biliary reaction. At autopsy the liver was found to be enlarged and there was much perihepatitis. The upper surface presented three tumor masses, which, on microscopic examination, proved to be small round-celled sarcomata.

CASE XXVI.—Barr and Rénon[24] report the case of an infant in whom the liver seemed somewhat enlarged and in places cirrhotic. On section, no alteration of the hepatic cells was found. The lesions were limited to the biliary canals, which were very much dilated, and were surrounded by a well-defined zone of embryonic and fibrous tissue. In certain of the canals polypoid projections existed, which, on section, proved to be the beginning of those columns which have been described on the surface of large cysts of the liver. The bile canals contained a yellowish mass. There were no cysts, properly speaking, in the liver, but the lesion belonged to a like pathologic process beyond a doubt. The kidneys were cystic.

CASE XXVII.—Lendrop's;[101] primary sarcoma; age four months; no jaundice; enormous distention of abdomen.

Treatment.—Tumors of the liver in children may be removed if they are primary growths. Ponfick[81] has shown that a large portion of the liver of animals may be removed, and subsequently be quickly replaced by a kind of "liver generation." Von Meister[20] confirms Ponfick's results. According to the former, in the dog, cat, and rabbit, removal of more than three-fourths of the liver was not followed by any serious consequences, and in thirty-six days after the operation repair had advanced to such an extent that the weight of that organ was regained. This regeneration is effected partly by hypertrophy of the hepatic cells, but mainly by their hyperplasia. Bile-ducts and blood-vessels share in the new formation.

Keen[45] has removed a tumor from the liver of an adult. In this operation he cauterized the liver-substance around the tumor without previous ligature, except where large veins were encountered. After the removal of the tumor, the flaps of liver-substance were approximated by sutures, which were passed deeply through the substance of the liver. The patient recovered. Keen tabulates nineteen other cases which had been operated on for removal of liver tumor. Of these cases, two died, the result in one was doubtful, and sixteen were successful. Although the operation has not been performed on a child, there seems to be no reason why it is not applicable, particularly in cases of benign growths or single growths which are malignant.

2. *Hydatid Cyst of the Liver.*—In a Paris thesis Duval[54] discusses fully the subject of hydatid cysts of the liver in children. This author divides hydatids of the liver into: first, those which are simple or unilocular, which contain the ordinary clear, characteristic fluid, and which have not been treated; and, second, those which are multiple, which are scattered in the liver or omentum, which contain a number of daughter cysts, and which may be suppurating, either spontaneously or as the result of some former operative treatment.

Gailliard[44] divides intra-thoracic hydatid cysts which spring from the liver into those which project anteriorly and those which project in the dorso-axillary region or postero-laterally. Debove,[96] Laveran,[97] Cullen[98] (boy aged eight), Félizet,[99] and Boinet[108] report cases of this affection. Debove's case was thought to be tuberculous peritonitis. Boinet found a ptomaine in the fluid which caused symptoms in a mouse and rabbit similar to the toxic symptoms from which a patient died,—rapid respiration, paresis of lower extremities, convulsions, and death. The toxin was found more abundantly in cases in which electrolysis and puncture had been performed.

Symptoms.—The symptoms of hydatid cyst of the liver are principally concerned with the character of the tumor which develops. If this tumor projects into the abdomen, it will have the following characteristics: it will in all probability be felt in the right hypochondriac or epigastric region, it will usually be round and regular in outline, and it will present fluctuation. By bimanual examination, placing one hand in front of the tumor and the other hand in the lumbar region, the tumor may be shown to be movable both antero-posteriorly and transversely. This sign constitutes Guyon's ballottement, which has been claimed to be pathognomonic of kidney tumors. The tumor produced by a hydatid cyst will be dull on percussion, and it will be found to be movable with respiration (Duval[54]).

In case the cyst arises from the superior or posterior surface of the liver, it may project into the thorax. If the tumor is in the anterior portion of the thorax, it is fairly easy to recognize if it is not complicated by pleural effusion. An area of bulging will be seen in the mammary region, and around this area the percussion-note will be dull. There will be absence of breath-sounds and suppression of vocal fremitus over the area of bulging. Posteriorly the respiratory murmurs can be heard to the base of the lung. Cysts which project in the dorso-axillary region simulate pleuritis of the right side and are difficult to diagnose (Gailliard[44]).

Superficial cysts will present the so called "hydatid thrill" to combined palpation and percussion. This sign was present in a case reported by Malcolm.[34]

Santoni,[109] by auscultatory percussion, has found that the stethoscope reveals a special and peculiar sound of sonorous quality having a low tone and of brief duration, which ceases abruptly. It may be compared to the sound produced by striking a membrane stretched upon a metallic frame. This sound is so characteristic that, once heard, it can hardly be forgotten. He considers it as a pathognomonic sign of the disease.

Treatment.—According to Duval,[54] two operative procedures appear to be disputing for surgical favor: first, puncture and evacuation of the cyst, followed by the injection of a parasiticide liquid; second, free opening, with extirpation of the cyst when possible.

The technique advised for the treatment of these cysts by the injection of parasiticide fluids has been varied. Baccelli punctures the cyst, does not evacuate the fluid, and injects an antiseptic solution. Debove punctures the cyst, evacuates the fluid completely, and then injects a given quantity of corrosive sublimate solution, which is removed after a few minutes. Bouilly aspirates the cyst to dryness with Dieulafoy's syringe and then injects five grammes of Van Swieten's solution (1 to 1000), leaving it there. He closes the puncture with cotton and iodoform collodion.

The dangers of these methods are numerous. Escape of the injected fluid into the peritoneal cavity may cause fatal peritonitis. The injected fluid may be absorbed from the cyst, causing symptoms of dangerous intoxication. The operation may be incomplete.

The safest method is to open the cyst by a free incision, either in two steps or at a single operation. If the operation is done in two steps, the cyst-wall is stitched to the peritoneum on the occasion of the first interference, and when adhesions have formed the cyst may be opened and its contents evacuated. To accomplish the same result at a single operation, which is the method to be recommended, the technique is as follows. After a lateral incision through the abdominal wall, two ligatures of silk are passed at the two extremities of the cyst in such a manner that it is drawn against the abdominal wall when it is delivered. Guarding the peritoneum by sterilized compresses, the cyst should be opened freely and its contents evacuated. Now, one of two processes may be chosen: the sac may be fixed to the abdominal wall, or it may be extirpated. If the former procedure is adopted, the redundant portion of the sac should be cut away and the remaining cavity should be packed with iodoform gauze. If the latter method is chosen, the sac should be removed by curved scissors and the finger, stopping hemorrhage by the application of the thermo-cautery as the operation proceeds. The edges of the liver from which the sac has been removed may be brought together with catgut sutures.

Duval[54] reports twelve cases. Of these six were treated by puncture of the sac and the injection of parasiticide fluid and six by incision. Of the first six cases three were cured, two died from mercurial poisoning, and one developed suppuration. Of the second six cases all were cured, one of these being the case in the first six which suppurated.

Malcolm[34] reports a case in a child aged five and a half years, treated by free incision and drainage, and cured.

Lloyd Roberts[6] reports a case in a girl aged nine years, in which the diagnosis was confirmed by aspiration. Recovery took place after incision and drainage. The cyst-wall, which was one-quarter of an inch thick, was stitched to the abdominal wound.

VII. TUBERCULOSIS AND SYPHILIS OF THE LIVER.

1. *Tuberculosis of the Liver.*—The lesions in the liver produced by tuberculosis may be either larger or smaller gray miliary tubercles or cheesy nodules. These lesions are usually accompanied by cirrhotic and fatty changes.

Pathology.—The liver may become infected either through the blood or by the extension of peritoneal tuberculosis. If the infection is brought to the liver by the blood, the tubercles will be found in the neighborhood of the branches of the portal vein. The fat in a tuberculous liver begins at the periphery of the lobule.

Symptoms.—Hutinel[61] reports four cases of tuberculosis of the liver which presented miliary tubercles at autopsy. The patients were œdematous and cyanotic, the nails were blackish, and the extremities of the fingers were habitually cold. In these respects the patients resembled cardiac more closely than they did tuberculous cases. The abdomen was uniformly distended, making it large and prominent, and on the skin covering this region dilated veins could be distinguished in the form of a rosette. Palpation of the abdomen was painless; there were neither puffiness, peritoneal thickening, nor adhesions of intestinal loops. If the disease had depended for its development upon extension from peritoneal lesions, examination of the abdomen would have resulted in the detection of these later signs. On percussion, the presence of ascitic fluid, which had been suspected after palpation, was confirmed. The liver was much enlarged, and extended from the third interspace to the umbilicus. In each of these cases the liver was firm in consistence and its edge was sharp. Jaundice was absent. The spleen was hypertrophied.

In these cases the heart is usually somewhat hypertrophied, and will present gallop rhythm or fetal rhythm on the slightest occasion.

H. Köster[112] reports the case of a boy aged three years who suffered from repeated attacks of vomiting and jaundice of varying intensity from the age of fourteen months. Upon autopsy a great number of tubercles as large as a pea, and many of them caseous, were found disseminated through the liver. The portal hepatic glands were swollen to the size of a bean. In the lowest part of the ductus communis choledochus a perforation of the wall some few millimetres in diameter led down to a cavity, the size of a hazel-nut, filled with a sticky, puriform mass, and surmounted by a fibrous, rather firm mass of connective tissue.

Prognosis.—The prognosis is grave.

Treatment.—The treatment resolves itself into a symptomatic battle, the physician constantly endeavoring to make the existence of his patient as comfortable as possible.

2. *Syphilis of the Liver.*—The lesions in the liver produced by syphilis are seen frequently in young children, but in older subjects the condition becomes more and more rare. According to Starr,[67] the disease may take one of three forms: first, a diffuse growth of connective tissue elements; second, localized gummatous change; and, third, peripylephlebitis. In the latter form the inflammatory process is confined to the septa, the proliferation of connective tissue taking place both between the hepatic islands and in their interior. Hector Mackenzie[19] showed a specimen at a meeting of the London Pathological Society in which there was great overgrowth of connective tissue between the lobules and in the lobules themselves, constituting a true pericellular cirrhosis. In the discussion following the presentation of the specimen, Dr. G. N. Pitt reported a similar case. Both of these cases also presented hepatic gummata.

Bar and Rénon[22] report a case in which the proteus vulgaris was obtained in pure culture from the umbilical vein, the liver, the spleen, and the right heart in a child who had died five days after birth, and whose liver contained evidences of syphilis.

Symptoms.—In mild cases the symptoms are few and are not characteristic. In grave cases there will be jaundice, ascites, umbilical hemorrhage, melæna, ecchymoses, subnormal temperature, syphilodermata, and syphilitic lesions of the mucous membranes. The liver will be enlarged and hard.

Prognosis.—On the whole, the prognosis is unfavorable. Cases which are properly treated do, however, recover.

Treatment.—The treatment should be chiefly mercurial. There is much difference of opinion concerning the form of mercury which should be employed. Many authors recommend mercurial ointment, one drachm to be smeared on the binder daily and thus by the movements of the child absorbed through the skin. Other authorities favor the employment of mercury with chalk, in doses of one-quarter of a grain three times daily. We prefer to use the mild chloride of mercury, in doses of one-quarter of a grain three times daily. This dose can be increased gradually until diarrhœa occurs and thus points to the discontinuance of the remedy.

As the case improves the syrup of the iodide of iron may be employed. Large doses of this remedy should be given,—fifteen drops three times a day for a child one year of age. Any of the other iodides may be used. Starr[67] advises iodide of potassium, one grain; chloride of ammonium, one and one-half grains; compound syrup of sarsaparilla, ten drops; and water, fifty drops, three times daily, for a child one month old.

Jacobi[1] advises the alternate use, by the week or fortnight, of the iodide of potassium or sodium and the bichloride or green iodide of mercury.

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ACUTE NEPHRITIS AND CHRONIC NEPHRITIS.

By D. D. STEWART, M.D.

ACUTE NEPHRITIS.

General Consideration.—In discussing the diseases of the kidney commonly known generically as Bright's disease, the writer prefers to employ the term *nephritis*, for the reason that not only in the kidney affections of adults, but also in those of children, forms of disease are encountered, more especially in the chronic malady, in which the urine is more or less persistently albumin-free. Now, as he has remarked in a previous publication,[1] apart from the disadvantages of a cognominal nomenclature of disease, Bright's researches, which led to the affections of the kidney, as they were then understood, being named after him, referred solely to the diseases of these organs having as their chief mode of recognition the invariable presence of albumin in the urine. The term nephritis is for this and other reasons a preferable one, but it must be understood that in employing it, as Councilman has recently remarked, we do not necessarily imply that the existing lesions should fall under the rubric of *inflammation*, the word being used merely to indicate a *disease* of the kidney, for which simplicity renders undesirable the coining of a new term, such as "nephropathy."

In children, unlike what is the case in adults, save as concerns a special form of nephritis, the so-called granular kidney, we have no definite knowledge of a Bright's disease strictly speaking chronic in onset, excepting the nephritis of amyloid disease. This last, as Dr. Goodhart has remarked, is clinically simply a form of chronic parenchymatous nephritis, with symptoms of the advanced stage of this malady, so that separate consideration of it is unnecessary.

Diseases of the kidney in infancy and childhood, other than the acute nephritis accompanying scarlatina, in former years were viewed as of some rarity, they commonly escaping detection through lack of exact knowledge as to mode of causation. Recent work in the lines of bacteriological re-

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[1] "Further Remarks on the Occurrence of a Form of Non-Albuminous Nephritis," Medical News, April 14, 1894.

The kidney is considerably enlarged in the subacute stage, and in the more chronic may be enlarged, of normal size, or slightly smaller than normal. The capsule is somewhat adherent in the subacute form and more tightly in the chronic form. The surface is pale or moderately mottled. Section in the subacute form shows a pallid, wide, opaque cortex, with indistinct markings and pale glomeruli. In the more chronic form the cortex may be wider than normal, or again even smaller. As in the subacute form, it is opaque, pallid, with obscure markings and non-visible or pale glomeruli. The essential lesions are glomerular. These are quite similar in the subacute variety, as regards changes in the tufts and capsules, to those described under the acute form.

In the more chronic form there is wide-spread hyaline degeneration of tufts and glomeruli, with obliteration of capillaries. Every transition can often be noted between the glomerular lesions in these and those in the subacute stage. The capsular epithelium and the intra-capsular connective tissue formation may be increased. There is general wide-spread degeneration of tubular epithelium and destruction of the tubules themselves. What remains of the tubules may be merely thickened, irregular membrana propria. The connective tissue throughout the kidney is much increased.

Etiology of Acute Diffuse Nephritis.—It is interesting that there exists a consensus of opinion among clinicians supporting the fact that acute nephritis, in childhood at least, practically never can be indubitably traced to exposure to wet cold or sudden cold, the most injurious forms of exposure. This is important in view of what later developments have taught us as to the etiology of renal inflammations, cases of which were previously so confidently attributed to cold and exposure because of lack of more fitting evidence. Not only in children but in adults the writer has long thought that in the majority of cases of acute nephritis which are viewed as originated by exposure, the effect of cold or wet, as has been suggested by Vignerot,[1] is to bring to the surface an acute exacerbation of a hitherto existing though unrecognized chronic affection, primarily doubtless of infectious origin. Exposure may also thus, through a sudden lessening of the vital resistance of the individual, give impetus to a morbid condition already latent, which, however, it cannot itself create. H. P. Loomis[2] shares the writer's opinion that many cases of acute nephritis supposedly due to cold are really of infectious origin. He has been unable to induce nephritis in rabbits first shaved and then exposed to hot and cold water. In adults he holds that certain cases of acute nephritis may occur as the result of exposure through the sudden lowering of vitality permitting the action of a toxic agent already present, which in itself would have been insufficient to produce the attack. Exposure could thus be the determining cause, and a toxic agent, such as a toxin of an infectious disease, or a drug, such as cantharides or turpentine, the exciting cause. There is little doubt that all cases of acute nephritis, even in adults, are of toxic origin, due to the direct local irritation of the kidney by a poisonous substance brought to it by the blood. This substance may be and usually is a toxin produced by a micro-organism, or less often a medicinal agent, or, though still more rarely, if ever, in childhood, a toxic waste produced in the organism. Concerning the operation of the various causes, Loomis aptly remarks that what have been styled different forms of acute nephritis are in reality distinct individual developments of the disease under its particular causes, or, it might be well to add, under a special susceptibility of certain histological elements of the kidney to that cause. Unquestionably the overwhelmingly important cause of acute nephritis in both children and adults is the action on the vascular secretory and intertubular structure of the kidney, *via* the blood, of a soluble toxin generated by a micro-organism, the natural route of the elimination of the former of which is largely the kidney. Apparently the bacteria themselves are little concerned directly. The interesting experiments of Roux and Yersin in diphtheria are in agreement with the observations of other bacteriologists as to the causation of nephritis in certain other infectious diseases. Roux and Yersin in a large number of observations on this point have never met the Klebs-Loeffler bacillus in the kidney, nor were they able experimentally to induce a nephritis by the injection of Klebs-Loeffler bacilli into the renal artery, but intravenous injections of bouillon culture, free from Klebs-Loeffler bacillus, have always produced typical diphtheritic nephritis. (Loomis.) Acute nephritis may be thus originated in the course of any infectious disease, the most usual of which to produce it, with frequency approximately in the order named, are scarlatina, diphtheria, erysipelas, measles, acute endocarditis, acute pneumonia, infectious tonsillitis, acute rheumatism, typhoid fever, small-pox, varicella, malaria, tuberculosis, syphilis, and gonorrhœa. The degree of frequency with which it is encountered in any particular infectious disease depends upon whether the kidneys are especially concerned in the elimination of the toxin of the disease. Thus, as Loomis points out, kidney complications are of extraordinary frequency in scarlatina, but are much less often encountered in typhoid fever, the removal of the toxin of the latter taking another channel.

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[1] Arch. Gén. de Méd., October, 1891.

[2] American System of Practical Medicine, vol. ii.

Of the above ailments, scarlatina and diphtheria are the most common causes of acute nephritis. Apparently in scarlatina there is no special relation between the intensity of the fever and the development of nephritis. In scarlatina acute nephritis is more common and is attended with a higher mortality in male than in female cases. The most frequent age at which this complication is encountered is between five and ten years. It is more apt to occur during the period of desquamation, somewhat late in the disease. In diphtheria, unlike scarlatina, nephritis is not common in the lighter attacks, and usually develops early.

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[2] American System of Practical Medicine, vol. ii.

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Should the case show a more chronic tendency, there are developed in the course of the disease pallor, dropsy, anæmia, and probably cardiac dilatation.

With a favorable issue, after from approximately five days to several weeks of fever and a tendency to the slight or decided uræmic symptoms before indicated, these lessen and finally disappear, coincidently with increase in the amount of urine and of the output of urea, with diminution, though much more tardy, in the percentage of albumin present and the microscopic evidences of subsidence of the inflammatory condition. After some weeks or months, complete disappearance of all symptoms of the disease is common in favorable cases, with the gradual return to the normal of the urine. Should the case assume a chronic form, the child may to all appearances recover its usual health, although a tendency to puffiness of the face or eyelids and extremities, with pallor, anæmia, and disturbed cardiac action, may be steadily manifest. The urine then continues to show more or less marked persistent indications of existing disease, and the case becomes an ordinary one of chronic nephritis, in which albuminuria, debility, bacteria. Among medicaments are such drugs as cantharides, turpentine, potassium chlorate, squill, and corrosive sublimate. Most of these drugs, even in moderate doses, if too long continued, have a special tendency to set up an acute degeneration of the epithelium of the tubules of the kidney of the form first described. Cantharides and turpentine in full doses set up a diffuse inflammation, involving tubular and intertubular structure. Potassium chlorate in overdose not only causes disintegration of the blood, but a severe diffuse acute nephritis which is apt to result fatally. It is little known that chloral hydrate has a most pernicious action on the kidney, causing in certain cases, even in small doses, if long continued, degenerative changes. Experimentally, on dogs, Cavazzini[1] was able to produce, by a single injection of chloral hydrate into the abdominal cavity of dogs, acute granular degeneration of the tubules, "with other symptoms of acute parenchymatous nephritis." This action of chloral is important in view of the rather free administration of the drug in cases of scarlatina by certain physicians. Other drugs sometimes administered over long periods in nephritis, such as strontium lactate, with the idea of beneficial effects, have been found experimentally to induce nephritis. (See, as to this effect of strontium, the *American Journal of the Medical Sciences*, p. 191, February, 1894.)

The prolonged administration of naphtalin, especially the impure variety, and of impure α- and β-naphtol, has set up nephritis. Instances of this have occurred in the writer's observation. Salicylic acid, especially that derived from carbolic acid, even in small but long-continued doses, has an injurious action upon the kidney, which eventually would tend to produce a degenerative nephritis. The too free ingestion of alcohol is regarded by some as a cause of nephritis. Alcohol cannot be regarded as often operative in the production of nephritis in children.

In the newly born, uric acid infarctions in the tubules of the kidney are a frequent cause of a mild form of nephritis, and the same may tend to induce interstitial nephritis in childhood.

Symptoms.—The symptoms of acute nephritis in childhood depend somewhat upon the variety of the affection present and its severity. The notion that all cases are apt to be ushered in by such typically objective signs as scanty, smoky urine, high fever, violent headache, convulsions, and dropsy is most unfortunate, permitting, as it often has, many cases of acute nephritis occurring in the course of an infectious disease to go unrecognized. Thus, in the acute degenerative nephritis which is very common in infancy and childhood accompanying scarlatina and diphtheria, there may be no obvious symptoms other than those of the original disease, so that, unless one is on the watch, it may altogether escape detection. This is the more probable in diphtheria, in which, as before remarked, unlike scarlatina, the renal complication appears at the height of a severe form of the malady, the symptoms of the last of which may entirely mask those of the renal complication. With the disappearance of the original illness the kidney involvement may undergo resolution, or may, and much less frequently, although not rarely, continue as a low grade of nephritis, to be subsequently brought into activity by the occurrence of a second attack of an infectious disorder, or, with impaired bodily health, by exposure to wet cold. There are frequently reported cases of nephritis in both children and adults, noted as those of the acute variety, in which the indications of the urine and, when such are later obtainable, the post-mortem records show undoubted evidences of a long-standing affection, of which a severe intercurrent attack had alone caused its recognition. In more pronounced cases of the acute affection, such as the frank form of glomerulo- or parenchymatous nephritis occurring in childhood, the onset is apt to be outspoken. There may then occur more or less prominently such symptoms as a chill or chills, with a temperature rise of several degrees, a rapid, irregular pulse, dyspnœa, cough, coated tongue, anorexia, nausea and vomiting, loin pain, aggravated by pressure, and, if the child is sufficiently old to interpret intelligently its symptoms, severe headache and perhaps vertigo. Convulsions, limited or general, succeeded, if generalized, by stupor or coma, are not uncommon. The urine may be, and often is, diminished in amount, of smoky hue, and will contain an abundance of albumin and, microscopically, blood-cells, and casts of the same, with epithelial and hyaline casts.

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[1] Deutsche medicinische Zeit., September 7, 1891.

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There are other cases of acute nephritis of more moderate severity that can scarcely be overlooked, showing many of the above-mentioned symptoms, though in minor degree, which, receiving prompt treatment, at no time assume a grave aspect, but usually progress to complete though gradual recovery. Thus, as is more common, the fever may be of a very moderate grade, with initial chill absent or unnoticed. There are malaise, anorexia, and perhaps nausea and vomiting and loin pain, with diminution in the amount of urine, which has become of high color and perhaps smoky hue, and now contains an abundance of albumin, blood-corpuscles, blood and epithelial casts, and degenerate renal epithelia or their nuclei.

A discussion of certain of the usual symptoms of acute nephritis is important, such as the *state of the urine*, the *gastric* and *nervous* symptoms, the *dropsy*, and the *condition of the vascular system.*

The Urine.—The urine is commonly diminished in amount from the onset, and this diminution may proceed to complete suppression. The color is high, due to the presence of more or less blood. It may be of a smoky or actual blood tint. The sediment when it contains blood is copious and of brownish hue, and consists of, besides blood and blood-casts, epithelial and other cylinders, renal epithelium, urates, and granular débris. In the early stage, erythrocytes and casts of the same, epithelial casts, and degenerate isolated renal epithelia or their nuclei crowd each field of the microscope. Blood-casts are in excess of the other elements at first; at a later stage these become less frequent, and epithelial, granular, and hyaline casts with renal epithelium come more into prominence.

The scanty, smoky urine is of acid reaction, and at first is of high gravity (1020 to 1040). Albumin is present in large amount, by weight equalling from one-half per cent. to two per cent., and by ordinary sedimentation, when precipitated by heat and acid, from one-third to three-fourths of the total bulk of the urine in the test-tube. Urea is always much diminished in amount. Concerning the output of urea in children in health, it is important to note that for the body-weight there is a considerable increase over that present in adults, so that while in the healthy male adult there is on full diet excreted somewhat over a gramme for each five pounds of body-weight, in young children it approximately equals one gramme for three pounds, or five grains for one pound. This is especially true of children between the ages of three and ten years.

With lessening of the inflammatory condition there occurs increase in the amount of urine, with diminution in the albumin and in the number and character of the casts.

It is important to note that, especially in the nephritis of scarlatina, the presence of casts may and often does precede any decided albumin reaction or the occurrence of obtrusive symptoms of nephritis. So also in milder forms of nephritis the urine may be but slightly altered in appearance, containing little blood macroscopically and not a great deal microscopically. Albumin may be present in but small quantity, and casts may not be numerous. In a severe and very fatal form of primary acute infectious nephritis occurring in infants, described by Holt, in which with high fever and pronounced nervous symptoms there were rapid pulse and peculiar respiration, but exceptionally dropsy, albumin was frequently absent early in the attack, and, although present later, was rarely in large amount. Casts were never numerous, but were always found when carefully searched for: these were of the epithelial, granular, and hyaline varieties.

Casts in many cases of acute nephritis are not only found to precede more than a faint albumin reaction to the commonly applied tests, but may continue to be encountered after the disappearance of albumin. H. P. Loomis calls attention to the interesting observation of Sehrwald that casts may be undetectable in the urine in nephritis, or be found in very small amount, for the reason that their solution may occur in acid urine through the influence of the trace of pepsin it normally contains. Sehrwald also showed that the prolonged sojourn of the urine in the bladder causes disappearance of casts, and that the higher the temperature the fewer the casts.

Should the case become chronic, the urine increases in quantity, with a diminution in its gravity and a slight increase at first in the output of urea. The amount of albumin remains from the first high, approximating from one-quarter of one per cent. to two or more per cent. by weight. Casts continue numerous, and are chiefly epithelial, finely and coarsely granular, and hyaline. In an advanced stage of the chronic variety, diminution in the amount of urine with continued low urea output is more habitual, with a large percentage of albumin, and the constant presence of epithelial casts and of cells from the tubules showing fat granules.

Gastro-Intestinal Symptoms.—These, when manifest, are coated tongue, anorexia, nausea, and, with the occurrence of other uræmic symptoms, vomiting.

There is usually at first constipation. Attacks of diarrhœa are more common in the chronic form of nephritis.

Nervous Symptoms.—In cases of gravity, if the child is old enough to indicate its sensations, violent headache with vertigo may be complained of. There may then be marked restlessness, delirium, and local and general spasms, the last succeeded by more or less pronounced coma. Convulsions at the outset may be due to the severe constitutional disturbances ushering in the disease. If pyrexia is not marked, and considerable diminution in the amount of urine has occurred, the convulsions are probably uræmic, whether occurring early or late.

Dropsy.—This is not an invariable sign, even in cases ending fatally. It, however, is usual, and is apt to be of sudden onset and may become general in a few hours. Commonly it is at first manifest in the loose cellular tissue of the eyelids, face, and neck, and about the ankles, and subsequently the scrotum or labia; thence more decidedly in the extremities, and perhaps now invading the serous cavities, such as the peritoneum and pleura, and, more rarely, the pericardial sac. Œdema of the lungs and glottis may occur, and is of ominous nature.

Vascular System.—In the milder cases there are no phenomena here worthy of note. In those of more gravity, disturbed cardiac action, a tendency to rapid cardiac enlargement in the direction of dilatation, and a quick, jerky pulse—commonly in children not of raised tension—are usual.

Complications.—Other than the subcutaneous œdemas and visceral dropsy and the cerebral symptoms, pulmonary and glottic œdema, acute pericarditis and endocarditis, and pneumonia, chiefly lobular, are among those to be on the watch for. Enlargement of the heart with dilatation in excess of hypertrophy is common in severe attacks, and during convalescence marked anæmia is usually manifest.

Pulmonary œdema, a complication of urgent gravity, simulates, as concerns the physical signs, acute bronchitis, and, as regards the more obtrusive symptom,—urgent dyspnœa,—capillary bronchitis.

Diagnosis.—The diagnosis of acute nephritis is unmistakable in an outspoken case, displaying several of the symptoms detailed, such as the rather abrupt onset, the fever, vomiting, diminution in the amount of urine, with characteristic macroscopic and microscopic appearance, facial or more general œdema, or pronounced dropsy. The point to be constantly borne in mind in dealing with the diseases of childhood is, as Goodhart expresses it, that most of the usual symptoms of acute nephritis that one might expect to encounter may be absent, and others present tending to lead one even more astray, and that the younger the child the more likely this is to be the case. Few conscientious observers with much experience in diseases of children have failed to verify more than once this observation of Goodhart's. Error in diagnosis can nearly always be obviated by establishing the routine habit of urine examination not only in all cases of infectious diseases in childhood which may be complicated by nephritis, but in any illness the diagnosis of which is doubtful. The presence of albumin may not, of course, strictly speaking, mean nephritis, as in certain cases of diphtheria, but its recognition is at least a certain danger-signal and places one more on the watch. It is highly important to examine further than for albumin, lest otherwise a variety of cases of even pronounced nephritis be overlooked. Often the presence of casts precedes, perhaps for several days, the occurrence of albumin in the urine in scarlatina and in certain cases of glomerulo-nephritis. Acute as well as chronic diffuse nephritis may occur without the presence of albumin in the urine. Such cases are now known to be of no uncommon occurrence. In these casts are commonly present in the urine, and at least slight dropsy with often marked diminution in the amount of urine may be looked for. Henoch, quoted by Goodhart, refers to a grave form of hemorrhagic nephritis seen by Litten, the only recognizable signs of which were some œdema of the face and the presence of hyaline casts in the urine. Cases of this sort, fortunately not so common as those ordinarily seen, occurring in infancy, are doubtless more often overlooked than recognized, as is that form of acute nephritis occurring in infancy, before alluded to, studied by Holt. In this dropsy was exceptional, the onset rather abrupt, with decided fever and with gastric symptoms. Nervous symptoms with disturbed respiration were prominent. Later anæmia occurred. Albumin was frequently absent early in the attack, although invariably present in moderate amount later. Casts were always found when carefully searched for, but were not numerous, and were of the epithelial, granular, and hyaline varieties. Blood-casts did not occur, though erythrocytes, pus-cells, and renal epithelium were present in moderate number.[1]

This form is here dwelt upon in outline, since its recognition is often difficult unless watched for, and its prognosis when occurring in infancy is grave. In Holt's opinion, many cases of less severity doubtless altogether escape recognition.

Of course it must be recalled in any case presenting indications of acute nephritis that the acute seizure may be but an exacerbation of a chronic nephritis, perhaps long existent. This should be well considered in all cases of presumed primary acute nephritis resulting from exposure or arising otherwise somewhat obscurely. The previous history, if obtainable, usually throws light on the case, especially the occurrence of preceding scarlet fever or of diphtheria during which dropsy has occurred, or a tendency to puffiness of the eyelids or ankles, or to diminution in the amount of urine, and of symptoms suggestive of uræmia.

Cyanotic induration of the kidney, as a result of a long-existing mitral or tricuspid disease, originating certain of the symptoms of nephritis, can scarcely be mistaken for primary acute nephritis.

Prognosis.—This is always grave, save in the slighter cases, unless energetic treatment has been begun early before marked involvement of the secreting structure of the kidneys has occurred. The appearance of uræmic symptoms, such as violent headache, stupor, and local convulsions, renders the aspect serious, as does likewise a tendency to anuria or the occurrence of a grave complication, such as pulmonary or glottic œdema, pneumonia, or endocarditis. The younger the child the more unfavorable such symptoms are, death almost invariably resulting in cases under three years of age. On the contrary, older children are less apt to succumb to the disease than adults, probably because in the latter, especially when the disease occurs in a case over forty years of age, there have already existed chronic changes in the kidney.

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[1] In older children the onset is less abrupt. with only moderate fever and, unusually, dropsy. There is slight diminution in the output of urine and only a small amount of albumin.

In a certain number of cases of acute nephritis apparently complete recovery occurs, the child remaining perhaps for a number of years in seemingly perfect health, when symptoms of nephritis assert themselves gradually or suddenly, as after a chill. Such cases as these are not uncommon, and, as before remarked, are often misinterpreted. Of course the urine has not been normal through the interval, but the absence of recognizable symptoms prevented proper examination.

Treatment.—If we are dealing with a case of infectious disease such as scarlatina, in which acute nephritis may supervene, prophylactic measures are of the utmost importance. On the occurrence of the earliest indications of nephritis, such as the presence of casts and a trace of albumin in the urine, active measures should be taken that will often tend, if not to abort, at least to diminish materially the severity of the impending attack. The same measures should be pursued if the case is seen when the ailment is well under way.

Efforts should be directed to lighten the work of the kidneys, to assist otherwise in the elimination of the toxin of the disease, and to divert as far as possible such elimination into another channel. The patient should remain in bed between blankets. The temperature of the room must be maintained day and night at approximately 70° F. He should be sponged under the blanket with tepid water two or three times daily. The sponging should be followed by friction with a dry towel and subsequently with the hands. On no account must a chill be risked; so that at first, unless most cautiously administered, even a warm bath is to be prohibited.

A full dose of calomel (from two to five grains) should be administered at the onset, preferably on an empty stomach, followed in four or five hours by a few ounces of the solution of magnesium citrate. Subsequently moderately free bowel action is to be maintained by the daily employment of a saline, such as solution of magnesium citrate, or Epsom, Rochelle, or Glauber salt, with, at intervals of a few days, as judgment dictates, an additional dose of calomel. Danforth suggests that, apart from the extreme value of laxatives in lightening the work of the kidney, they have a derivative action, causing as they do hyperæmia of the intestinal mucous membrane, and thus "counter-irritating," as it were, the engorged kidneys.

From the onset the diet must be of the simplest. Unquestionably *the* food in acute nephritis is milk, than which nothing else should be allowed during at least the acute stage of the disease. There exists overwhelming evidence as to its extreme diuretic food value in acute nephritis and as a prophylactic against disease in scarlatina. If the stomach is irritable, milk should always be given in the form that experience shows will best agree: it may be lightly peptonized, or diluted with an aerated water, such as Seltzer, or Wilhelm's-Quelle. Apollinaris or lime water may be added. Milk so prepared may be alternated, if desired, with buttermilk, junket, whey, and, if much gastric irritability exists, with koumiss. In any case, milk itself has a more wholesome effect on the kidneys when taken largely diluted with a slightly alkaline water, such as Seltzer or Wilhelm's-Quelle.

The patient, from the first, if the gastric condition permits it, should be encouraged to drink freely also of plain water or an alkaline water, in order to assist in maintaining the renal secretion. Cream of tartar lemonade (a drachm of potassium bitartrate to the quart), taken in doses of a half-glass to a glass every four to six hours, is also very efficient if well borne by the stomach.

The action of the skin, stimulated from the first by maintaining the patient in bed between blankets, and by the equitable room temperature, tepid sponging, and free water-drinking, is further excited by the employment, should such seem to be demanded by the presence of fever, of a mild diaphoretic mixture which at the same time has a slightly diuretic action, such as a mixture containing, for a child of ten years, five grains of potassium citrate, from one to two drops of tincture of aconite, and ten minims of spirit of nitrous ether, with syrup of lemon and water up to a drachm. This is administered every three to four hours in from two to three ounces of water. More decided skin action, rendered imperative on the occurrence of uræmic symptoms and dropsy, is obtained by the hot pack or vapor-bath. A hot pack is administered as advised by Goodhart. The child, divested of all clothing, is wrapped snugly from chin to foot in a thin blanket which has been wrung out of very hot water. A dry blanket is quickly wrapped about this, and loosely over all a mackintosh. After from one to three hours the child is removed from the pack and quickly swathed in a dry blanket. The *continuous* employment of the hot pack is not advisable, and the bodily temperature must be watched during its action. (Goodhart.) By this means, or by the use of the steam-bath, very marked action on the skin is produced.

The steam-bath is given either by using one of the portable, inexpensive apparatuses now on the market or by erecting a tent in the bed by the use of half barrel hoops and conducting the hot air from an alcohol lamp under the covering by means of a readily constructed tin pipe. The bed steam-bath is preferable, as it can be continuously applied for a long period with the patient at rest in recumbency. The covering is snugly drawn about the neck of the patient, so that all parts of the body except the head are brought under the influence of the hot air. In a case related by Danforth, which he had regarded as well-nigh hopeless previously, the hot-air apparatus was kept in action almost constantly for ten days, the patient hus making a good recovery. Such a continuous employment of the steam-ath is not generally to be recommended, on account of the prostration it casions.

Free skin action may also be obtained by placing about the patient, who between blankets, several large bottles filled with boiling water and ed in long woollen stockings previously wrung out in hot water; or, t preferably, the warm bath suggested by Liebermeister may be employed. The temperature of the water on entering should be 37° C.; this is then increased to from 40° to 45° C. The patient remains in the bath from ten to thirty minutes. Cool water may be poured on the head during this time, if headache is induced by the bath. After the bath the patient is wrapped in warm blankets.

Pilocarpine is a very unsafe and not always certain remedy in the treatment of nephritis. It is regarded as a trifle less apt to cause toxic symptoms in children than in adults; but in the opinion of those who have most used it, and have thus seen accident result, it cannot be given in the uræmic condition, either in children or in adults, without apprehension. The chief dangers from its use are sudden œdema of the glottis and of the lungs, the last of which, despite treatment, is apt to be rapidly fatal. Few who have much used the drug have failed to see such cases. The writer several years ago, when he employed it largely, had sufficient experience in this direction to render him wary of its use. Pilocarpine is decidedly contraindicated in cases of valvular disease or with simple enfeeblement of the heart's action, or in those that show a tendency to pulmonary œdema or bronchitis, or in which a patch of pneumonia exists. There is less danger from it in cases with strongly active heart,—rarely, however, encountered in the acute nephritis of children,—free from any complication. It is more certain in effect when administered hypodermically. The dose is about one-twentieth to one-twelfth of a grain for a robust child from five to ten years of age.[1]

Its action may be much assisted and less diverted to other parts by the induction of a slight preliminary diaphoresis before the administration of the dose, as by drinking very hot lemonade, the patient being snugly tucked between blankets. It is perhaps wise, unless the child be quite robust, to administer coincidently a small dose of strychnine or of sparteine sulphate, to obviate the depressing effect of pilocarpine upon the heart and the respiratory centre. Atropine cannot, of course, be given with it, although it is the best antidote should untoward effects arise.

The Treatment of the Most Important Complications.—*Marked Diminution in the Amount of Urine, or Suppression of Urine.*—The skin should be freely acted upon by the hot pack or the vapor-bath, or by the very cautious use of pilocarpine hypodermically. Water or any pleasant diluent drink that may be craved, such as lemonade to which cream of tartar is added, is given freely by the mouth. Flushing the colon with water is of great value. It has rendered good service for years in the writer's hands in the treatment of uræmia in adults. Hypodermoclysis on lines now well understood is of even greater value. The bowels are stimulated into activity by a full dose of calomel, followed by a saline. The loins should be freely dry-cupped, after which large hot flaxseed poultices should be applied. The inclusion of digitalis leaves in the poultice is doubtless of some slight additional value. It is important to ascertain by the employment of the catheter whether the suppression is not merely apparent.

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[1] Danforth, whose experience with pilocarpine in acute nephritis in childhood has apparently been exceptionally favorable, advises as a medium dose one-eighth of a grain for a child aged from seven to eight years, to be repeated in a half-hour if sweating is not induced. He thus employs it in conjunction with the hot-air bath. His treatment in this particular is not to be generally commended. He holds that three very desirable results follow its employment in acute nephritis: reduction of arterial tension, reduction of body temperature, and increase in urea excretion. Arterial tension is rarely so raised in the acute nephritis of children as to be harmful, and high fever is not very common. Free urea excretion does not necessarily imply a similar free excretion of toxins inducing uræmia

General anasarca is treated similarly to the above, as concerns the employment of the hot pack, vapor-bath, and diuretics and brisk purgatives. Dropsy of the inferior extremities in the subacute and chronic stages is benefited by massage and bandaging. The use of Southey's tubes, under antiseptic precautions, is of great value in obstinate cases. The condition of the heart should always be carefully inquired into in these cases, and digitalis intelligently employed if dilatation exists. Dropsy of the serous cavities not readily yielding to the foregoing treatment may require tapping; this is proceeded with on well-known lines.

Œdema of the glottis necessitates very prompt measures, such as immediate scarification of the swelling. If this is ineffectual, tracheotomy is required. Pilocarpine should on no account be used if a tendency to œdema of the glottis or lungs exists. Œdema of the lungs is treated by brisk purgation, the free use of dry cups, and strychnine and perhaps also atropine hypodermically.

The approach of convulsions should be met by the administration of a brisk purge. In infants and small children the use of a warm bath with cold to the head is often of great benefit. In all, should there be time, a mixture of sodium bromide and chloral should be administered by the mouth or the rectum. Morphine hypodermically is scarcely to be recommended in the treatment of the uræmic convulsions occurring in infants or very young children, although its use is (rashly, in the writer's opinion) advised by some.

In a robust infant or a small child a few leeches to the temples, or in an older child the removal of from two to four ounces of blood from a vein, succeeded in each case by hypodermoclysis of a warm normal salt solution, cannot but be of immense utility. The infusion into the loose cellular tissue of the axilla or of the interscapular space, or into the loins, of from a few ounces to a pint of normal salt solution, the amount depending upon the age of the patient, is certain to be of signal service, diluting the poisoned blood and stimulating into activity the eliminating power of the crippled kidneys. During the convulsive seizure, inhalation of a little chloroform or of amyl nitrite should be employed.

Violent headache is treated by the administration of sodium bromide, leeches to the temple, and an ice-bag to the head.

In persistent vomiting, fluids by the mouth should be withheld. Enteroclysis, or even hypodermoclysis, is advisable. Half-drop doses of equal parts of tincture of iodine and carbolic acid in mint water may be tried at intervals of a half-hour until a few doses are given, or a small dose of carbolic acid with bismuth subnitrate in mint water may be used. Ten per cent. menthol in olive oil, of which a few drops are given on crushed sugar, ingested with a sip of water, is often of great utility, taken when nausea is experienced. Small doses of nitroglycerin are of value in uræmic vomiting, as in that of gastric catarrh.

During the subacute stage and into convalescence the diet should remain as far as possible a weakly nitrogenous one. Milk should be continued unless a strong distaste for it exists. It may be prepared with various farinaceous substances, or in the form of milk toast. With the approach of convalescence, the various digestible carbohydrates and farinaceous foods, tapioca, farina, or rice, in puddings, if desired, are allowed. Until well into convalescence, meat should be withheld. At first soups made of mutton and chicken are permitted, and after a time eggs and beef. Unless a special indication exists for meat, such as the presence of pronounced anæmia and the occurrence of indigestion from the farinaceous dietary, it should be allowed with caution in the subacute stage. The greater the intelligent caution as to the diet at this period the less will be the chance of the kidneys remaining damaged.

Anæmia succeeding an attack of acute nephritis, as in the chronic affection, should be combated by the employment of iron in any form that experience has shown agrees well with the stomach and that rapidly improves the state of the blood. The writer has for several years been sceptical of the value of Basham's mixture in nephritis, which scepticism was engendered by many years' faithful routine, generally resultless, trial of the remedy. He does not regard the acetate of iron as a proper form to administer in the anæmic condition. Tincture of the chloride, however, remains, as always, the standard preparation, which may be given in several palatable forms. Dree's albuminate of iron, which he has long used with success in the anæmia of gastro-intestinal disease, he regards with great favor in the treatment of the anæmia of nephritis, and as of much more value than some of the recent much-vaunted iron peptonates.

With iron, the use of strychnine is commonly indicated for its effect not only upon the heart, but also upon the general condition. Dilatation of the heart, as remarked, is not uncommon as the result of acute nephritis. Indications of this persisting call for special treatment. Rest is of the greatest utility, combined with the use of strychnine and full doses of digitalis or digitalin. Strophanthus is of much less value.

Intelligent use of the Schott method by baths and carefully graded passive exercise are the best means of combating cardiac dilatation which shows a tendency to persist.

The treatment of chronic nephritis is on similar lines to that pursued in adults. Space does not permit a special consideration of it here. The steady persistence of albumin in the urine, despite an apparent complete return to health, must always be a matter of grave concern. In the vast majority of cases, even if casts are present in but small number and of the hyaline variety, and the general health is apparently perfect, a chronic nephritis is, nevertheless, in all probability in process.

CHRONIC INTERSTITIAL NEPHRITIS.

This affection is that known also as the granular, the contracted, or the gouty kidney. It is presumably chronic in character from the outset, and is of infrequent occurrence in infancy and childhood, although of less rarity in older children than was formerly supposed. Guthrie,[1] who has recently reviewed the subject, has collected seven cases from medical literature in children between the ages of five and fourteen years, which were followed to autopsy. Few clinicians of experience have failed to see a case or two in children presumably of this malady. In the experience of the writer and others, its occurrence in young adult life is not infrequent.

Etiology.—This, if in many cases obscure in adults, is still more so in young children. In the writer's opinion, the mechanical effect of persistent uric acid precipitation in the tubules of the kidney has much to do with its causation in children and in adults; this, with an inherent tendency to early degeneration of the renal structure in subjects in which there exists a family leaning towards frank gout with coincident granular kidney, may readily explain its occurrence in older children. In these, too, lead poisoning, as in adults, may sometimes be a factor. Other causes operable in adults, such as alcoholism, over-indulgence in the pleasures of the table, with insufficient exercise and the occurrence of prolonged mental worry, are here without the pale.

Certain of the cases presenting symptoms of granular kidney may have originated in the mild acute nephritis which has occurred in the course of an infectious disease, such as scarlet fever, diphtheria, and typhoid fever. In a case of presumed granular kidney in a young girl aged seventeen, under the writer's observation (symptoms of which first became prominent when aged fifteen), scarlatina had occurred at the age of six, following which slight general œdema lasting about a week had been noticed by the parents. In a second case in a young man aged twenty-nine, scarlatina had occurred in childhood, but a history of dropsy or of other symptoms of kidney implication was not obtainable.

Pathological Anatomy.—Space does not permit a discussion of this, which is in all respects similar in detail to that of the disease in adults.

Symptoms.—These are much the same in typical cases as in the disease occurring in adults. They are often few, and not such as to attract attention to the kidney unless the physician is wary.

The urine is commonly free, pale, and of low gravity. Albumin is often absent for days at a time, and casts are few in number, though usually found if intelligently searched for. They are chiefly of the hyaline variety, though finely granular casts are not uncommon. Epithelial casts are rare. These last may occur, however, merely as the result of the long-continued irritating action of spicules of uric acid precipitated in the tubules (this condition is described in the section on Lithuria).

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[1] Lancet, February 27 and March 13, 1897.

Dropsy, until towards the end, is very uncommon. Even then it may consist merely of local œdemas. High blood-pressure is apt to be present in granular kidney in children as in adults, and commonly when the case comes under observation it is associated with at least slight cardiac hypertrophy. Arterio-sclerosis occurs in the later stages. Dickinson encountered this symptom well marked in a case aged six years.

Nervous and mental symptoms may be met with, such as severe headaches and vertigo and, in an advanced stage, uræmic convulsions, local or general. In a later stage, too, in certain cases with long-continued high blood-pressure, degeneration of the cerebral vessels may occur, resulting in hemorrhage. Filatoff (cited by Holt) has reported a case of cerebral hemorrhage in a child aged eleven years.

Certain other symptoms noted in this disease in children are *wasting*, the patients being commonly undersized and wizened. (Guthrie.) *Dryness*, *inelasticity*, and *pigmentation* of the *skin* are usual and were called attention to by Eustace Smith. Pigmentation of the skin not unlike that occurring in Addison's disease has been remarked in cases of granular kidney both in children and in adults. The writer has under observation a case of undoubted chronic nephritis in a young adult in which albumin is persistently absent from the urine, and in which pigmentation of the skin is quite well marked. With it there is also habitual prostration of strength. Vertigo is common, as in Addison's disease, but here it is a symptom of a toxic (uræmic) blood state. High blood-pressure with its accompaniments, and very low urea and the presence of casts, indicate that the primary trouble is renal.

Involvement of the adrenals in the sclerotic process, having primary onset in granular kidney, is the not unlikely cause of the pigmentation in these cases.

Although anæmia is common in granular kidney in adults, it by no means occurs to the extent to which some would have it supposed. In children it is perhaps always present. Not so, however, in young adults. In three cases studied by the writer over some time more than slight anæmia was absent. These three cases represent a special group first reported by him,[1] in which, with symptoms quite unlike those described as usual in granular kidney, a form of chronic diffuse nephritis apparently microscopically similar to ordinary cases of this affection (see the report of the examination of a section of kidney removed from one of these cases in life, Transactions of the Association of American Physicians, 1897, and the Lancet, September 4, 1897) is probably present. In these the chief symptoms and the urinary conditions agree in certain important particulars, and are totally unlike those observed in chronic parenchymatous nephritis or in the granular kidney as commonly described. In these, as in the others of the group, the urine is persistently diminished in amount, though of a hue and specific gravity normal if the output were that of health. The urinary solids, notably urea, are very low. Casts are constantly present, but in small number, and are chiefly hyaline and finely granular; epithelial and coarsely granular casts are also occasionally observed, but in small number. Dropsy is totally absent, as is more than slight cardiac hypertrophy, although the blood-pressure is persistently high. Symptoms of a uræmic blood state, such as headache, vertigo, confusion of thought, malaise, disturbed sleep, and, in one, local spasms, are more or less usual and severe.

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[1] See American Journal of the Medical Sciences, December, 1893: "On the Occurrence of a Form of Chronic Bright's Disease other than Typical Fibroid Kidney, without Albuminuria." Medical News, April 14, 1894: "Further Remarks on the Occurrence of Non-Albuminuric Nephritis." See also the Transactions of the Association of American Physicians, 1897, and the Lancet, September 4, 1897: "A Further Communication on the Occurrence of a hitherto Undescribed Form of Chronic Nephritis."

The writer regards cases belonging to this group as of not infrequent occurrence, although frequently overlooked through too blind faith in the notion that the occurrence of chronic nephritis without albumin in the urine is out of the question.

Diagnosis.—Enough has been said in the foregoing to indicate the chief diagnostic features of this form of chronic nephritis.

The point to bear in mind as an aid in the recognition of cases of nephritis, and this applies to all varieties in any stage, is that a careful urine examination for traces of albumin and for casts, and, if the importance of the case seems to demand it, quantitatively for urea, as a matter of routine in every doubtful case of disease, should be made whether nephritis be especially suspected or not. If the findings are suspicious only, such examination should be repeated from time to time and a renewed careful physical examination and a thoughtful re-inquiry into the symptoms made which may elicit certain diagnostic features hitherto undeveloped, putting one on the proper track.

It is needless to say that the prognosis of granular kidney is unqualifiedly bad, and that little hope of cure can be extended.

Treatment.—The treatment of chronic nephritis in infancy and childhood is in all particulars similar to that of the affection occurring in adults. Paucity of space forbids its discussion here on this account, however much such consideration is desired by the writer. In the cases supposedly originating from long-continued uric acid irritation, the general line of treatment is quite similar to that laid down under the discussion of the treatment of lithuria, which see. In the treatment of all forms of chronic nephritis, while attention to the diet is very essential, meat, at least in adults, is not to be strictly prohibited, nor a milk diet too rigidly enforced. In chronic nephritis—totally unlike what is the case in the acute and subacute forms—

also, at least as concerns adults, authorities are agreed that a full diet is not more liable to lead to uræmia than any other, while a strict milk diet often does so.

A mild climate, with low humidity, in which the patient may live out of doors without liability to cold or chill, will tend greatly to mitigate the symptoms of the disease. Flannel should always be worn next the skin. Much benefit may result through heightening the tonicity of the skin by a cool or cold sponge- or spray-bath in the morning, immediately preceded, if reaction from the cold bath is poor, by a few moments' immersion in hot water (from 105° to 110° F.). Brisk friction is used after the bath. The bath-room should, of course, be warmed, and not the slightest suggestion of chill should be felt at any time after the bath.

Tonics should be given when they seem indicated. Prolonged high blood-pressure to an extent which is regarded as likely to lead to cerebral accident is met by lessening the nitrogenous intake, occasional free purgation, and the administration of nitroglycerin or its new and promising congener, erythrotetranitrate.

SURGICAL DISEASES OF THE KIDNEY.[1]

By HENRY MORRIS, M.B., M.A., F.R.C.S. (Eng.).

CONGENITAL HYDRONEPHROSIS.

By congenital hydronephrosis is meant hydronephrosis of the fœtus and new-born; not hydronephrosis which occurs some time after birth and is due to a congenital cause.

In by far the larger number of cases of hydronephrosis found in the fœtus and new-born both kidneys are involved, the most common cause being an imperforate urethra. It may be due to minute cysts or membranous septa in the urethra, or to cysts in the ureter or pelvis of the kidney. The subjects of this disease may be born dead, or may live for a few weeks, months, or even years.

The urine removed from some of the cases of congenital hydronephrosis has contained little or no urea. The size of a hydronephrotic fœtus has proved a serious impediment to labor in several cases, and has rendered parturition impossible until the abdomen of the child has been reduced by tapping.

Congenital hydronephrosis is frequently associated with some other congenital deformity, such as harelip and club-foot. It proves that the secretion of urine goes on to a very considerable extent during the latter half of intra-uterine gestation, and that when any obstacle to the outflow of urine exists, the same pernicious effects of distention of the ureters and kidneys occur before birth as are commonly known to arise from urethral stricture, calculus, and other causes of destruction after birth.

A remarkable example of hydronephrosis developing during gestation in connection with abnormality of the ureter came under my care at the Middlesex Hospital in the year 1893. The child was born at six P.M. on November 29, and was brought to the hospital at four P.M. on November 30 by an attendant who had nursed the child from birth, and who stated that, though the baby had passed a small quantity of fæces, there had been no urine discharged. The child was brought to the hospital in the belief that he was suffering from "water on the stomach." He was small for a full-time child, well formed about the face and head, except that the right ear was somewhat misshapen, the legs and arms were very small, and the feet, as in most new-born children, were in the position of equino-varus. The abdomen was very greatly and irregularly distended, the distention being most marked in each anterior lumbar region, extending thence downward towards the symphysis pubis. The lateral enlargements gave the impression of being irregular oval cysts. They were dull on percussion, greatly distended, and became tense when the child cried. The remains of the umbilical cord were dried and ulceration was commencing at the navel, and extending downward in the middle line towards the symphysis pubis there was felt a hard cylindrical swelling connected with the abdominal parietes. The penis was enlarged, elongated, bent upon itself, and altogether of a size more in accordance with that of a boy of five. The gut was patent. A catheter slipped into the normal position of the bladder could not be freely moved about, and only a drop or two of blood-stained watery fluid escaped. The catheter was kept in, anæsthesia was induced, and an incision made in the middle line above the umbilicus large enough to admit the index finger. The intestines were not distended, an enlarged urachus was felt coursing downward towards the bladder, and the two large lateral swellings were found to lie behind the peritoneum. On compressing the abdomen, about an ounce of clear fluid was discharged through the catheter, but the flow could not be maintained and made no appreciable difference in the swelling. The laparotomy wound was then closed, an incision made in each loin, a large hydronephrosis containing clear fluid evacuated on each side, and the cut edges of the cysts stitched to the margin of the skin. After this the abdominal distention disappeared. The fluid drawn off from the cysts was clear, pale, faintly acid, contained a trace of albumin, with phosphates, chlorides, and sulphates, and .24 per cent. of urea, and had a specific gravity of 1004. There was a free discharge of urine from the nephrotomy wounds, and on December 3 urine was passed twice *per vias naturales.* The discharge from the loin was slightly bile-stained, and the skin was jaundiced. A fortnight later some urine was passed through the umbilicus by way of the urachus, the umbilical cord having separated two days before, and three days later this was followed by pus, which also escaped in small quantity from the penis, while a probe could be passed three inches down the track into the bladder. A month after admission two distinct swellings were noticed in the abdomen, on pressing which pus and urine came through the umbilicus and also through the nephrotomy wounds, but no urine passed by the umbilicus after the last week in January. Notwithstanding that the course of the disease from the surgical stand-point was satisfactory in spite of the severe measures resorted to and the tender age of the patient, ultimate success was not attained, owing to the difficulty in feeding. Though every expedient was tried, the child gradually wasted, and life was finally cut short by a sharp attack of diarrhœa ninety-four days after the operation, the weight having diminished from seven and a half to four and a half pounds. An examination after death showed both testes lying high up in the inguinal canal. The urachus was dilated and filled with thick creamy fluid, but no communication could be found between it and the bladder. The circumrenal tissue was condensed and the kidneys firmly bound down. Both ureters were enormously dilated and ran a tortuous course. On the right side the fistula led directly into the first convolution of the ureter; on the left side a similar track led into the renal pelvis. The right kidney was somewhat cuboidal in shape, and was rotated round a vertical axis, so that its posterior surface looked inward and somewhat forward. The right suprarenal capsule appeared to be normal, and lay at a considerably higher level than that on the opposite side. The right ureter at first coursed downward behind the lower border of the kidney, and was 1.2 centimetres in diameter; in this position it was firmly bound down to the abdominal wall and marked the site of the urinary fistula; it then took a convoluted course downward to the right iliac fossa, gradually increasing in calibre, at its widest part being 2.5 centimetres in diameter opposite to the iliac crest; it then turned sharply on itself, running upward and getting narrower, and finally bent down abruptly against the rectum and opened into the bladder at the usual situation. The left kidney was of normal shape, but the pelvis of the kidney had been pulled backward by the fistulous orifice and formed an acute angle with the ureter. The left ureter coursed at first behind the lower half of the kidney, in this situation being of the same diameter as that on the opposite side; it took a somewhat straighter course downward into the left iliac fossa. It then bent upward on itself, forming an extremely acute angle, turned over the left hypogastric artery, and ran downward again, coming in contact on the left side also with the rectum. It opened normally into the bladder. On the left side also the widest part of the tube was opposite the highest part of the iliac crest, and measured 1.8 centimetres in diameter. The walls of the bladder were much hypertrophied and its cavity contracted. The other organs presented normal appearances.

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[1] The present communication is a supplement to my article, vol. iii. p. 559 of this Cyclopædia, published nine years ago, and is to be read in conjunction with it.

The following is an excellent example of hydronephrosis occurring in a child. The case was published in full in the *Lancet*, vol. i., 1894, p. 205:

A boy aged seven was first admitted on November 18, 1892, with the history of hæmaturia at irregular intervals for a year past, the attacks usually coming on after exertion, and being unaccompanied by pain, gravel, or difficulty in micturition. On examination the urine proved to contain blood- and pus-corpuscles; it was acid, had a specific gravity of 1020, and did not contain more albumin than would correspond with the morphotic elements. There were no symptoms, and an examination of the bladder and renal region being attended with a negative result, the boy was discharged on the disappearance of the corpuscles from the urine a few days later. On January 10, 1893, he was readmitted. He stated that the day before his father had given him a whipping, and that shortly afterwards he was seized with severe pain, which he likened to the pricking of a pin in the right loin, lasting only a few seconds.

In the morning the urine was found to be blood-stained, and on examination it proved to contain blood-corpuscles in considerable quantity, with albumin and a microscopical amount of pus. On January 13 anæsthesia was induced, the bladder again sounded with a negative result, and the lower ends of the ureters examined per rectum without discovering any stone.

An incision was then made in the right loin, and the kidney drawn out and found to be dilated, flabby, and sacculated. An incision was made into its convex border, but no stone could be felt either in the renal substance or in the pelvis. The pedicle of the kidney was then transfixed with kangaroo tendon, and the vessels and ureter tied in separate halves. The kidney was detached and the pedicle dropped back into the abdominal wound, which was sponged out and closed.

The kidney itself was only slightly enlarged; the capsule stripped readily and exposed a smooth cortical surface. On section there was seen to be a marked degree of hydronephrosis, the whole of the medullary portion having disappeared and the cortical portion likewise being thinned. The infundibulum was greatly dilated, its mucous membrane much injected, and its wall considerably thickened.

Twenty hours after the operation the pus and blood had practically disappeared from the urine, though a few corpuscles were seen on January 30 and again on March 22; but recovery was complete, and in December the general health was reported by his doctor and friends as being excellent.

In a small proportion of cases, after nephrotomy or nephrectomy, suppression of urine follows, indeed is caused by the operation. This may occur even though no mechanical obstruction whatever is present on the opposite side, but is much more to be feared if the opposite kidney is diseased. A remarkable example of reflex anuria following upon an operation occurred in a girl of thirteen, whose kidney I explored for tubercular disease in June, 1894. The patient was admitted on April 25. Her father had died of consumption, and she had always been delicate. In December of the previous year she began to suffer from incontinence of urine, at first only at night, but afterwards during the day as well. On examination nothing abnormal could be felt in either loin, nor was there any tenderness. The urine was acid, specific gravity 1020; it contained blood and pus. On May 1 the patient was anæsthetized and the bladder sounded, but no stone was found; the urethra was dilated and the bladder examined, but nothing abnormal was detected. As the patient was losing flesh and her condition was daily becoming worse, the temperature having assumed the hectic type, it was decided to explore the kidneys. There was nothing (no pain, swelling, or tenderness) to indicate which was at fault, so the right was selected to be first explored. On June 22 an incision was made in the right ilio-costal space, the circumrenal fat torn through, and the kidney exposed and brought out through the wound. An incision was made along the convex border and the finger introduced into the pelvis, but nothing abnormal was found. The incision was closed with catgut sutures, the external wound stitched up, a drainage-tube introduced, and dry dressing applied. After the operation the patient had constant vomiting, which continued through the following day, the pulse being weak and rapid (120); temperature 102.6°. She was fed by nutrient suppositories and enemata alternately every three hours. On June 24 the vomiting had not ceased, and the patient had passed only a very small quantity of urine. A catheter was passed, but the bladder was empty. She was ordered a hot-air bath, and a hypodermic injection of morphine (one-eighth grain) was administered. She died the same evening. At the autopsy there were tubercular disease of the left kidney and a fistulous opening from the left ureter into the vagina. The opening into the vagina was very small, and situated high up in front of the cervix uteri. There were right pyosalpinx and matting of the left Fallopian tube, a patch of miliary tubercle in the right lung, and cretaceous bronchial glands.

In another case, in which exploratory laparotomy was performed for nephralgia on the same afternoon, and upon a woman in the same ward, followed by an identical procedure in the right loin, there ensued a similar state of continuous vomiting and anuria ending in death on June 25. There was a chronic ulcer of the stomach, but the kidneys were found to be healthy. These are typical cases of reflex anuria due to the disturbance of the nerve-plexuses of the kidney. I have not as yet attempted operative treatment for such a case, nor do I know of its having been used; but it has been proposed by Meyer, under certain specified conditions, to treat cases of anuria coming on after nephrectomy by "an artificial direct depletion" of the remaining kidney, with the view of reducing the hyperæmia which follows the ligation of the renal blood-vessels on the side operated upon originally.

I do not think we can be sanguine of the result of such depletion, though the proposal seems worthy of trial.

Some of the latest developments in operating upon the higher urinary organs relate to stenosis, valvular stricture, and valve formations in the ureter. Plastic operations after resection of a portion of the ureter, longitudinal division of stricture and transverse union of the longitudinal wounds, and excision of the ureter in the whole or greater part of its length after nephrectomy for tuberculous disease have been performed with success by Küster, Christian Fenger, Reynier, myself, and others. It is highly probable that one or other of the plastic operations may advantageously replace nephrectomy or nephrotomy in certain cases of moderate degrees of hydronephrosis, more especially when there is any doubt about the healthy condition of the opposite kidney.

The making of a wound in the loin sufficient to admit of the introduction of the finger so as actually to feel the kidney is a necessary part of diagnosis and treatment in some painful and chronic disorders of the organ which are found not to be amenable to other methods of cure.

FLOATING KIDNEY.

Though true movable kidney, where the dislocation takes place within a lax capsule, is a disease of adult life, the clinically allied state in which the organ is furnished with a mesonephron and floats freely within the cavity of the peritoneum is a congenital malformation, and may give rise to symptoms any time after birth.

The abnormally attached organ also may be the seat of chronic morbid changes, such as pyelitis, hydronephrosis, or calculus; such an organ is also liable to attacks of acute congestion or strangulation, directly or indirectly attributable to displacement of the organ, with kinking or twisting of the structures entering the hilum.

Such symptoms as headache, furred tongue, vomiting, or jaundice, with other intestinal and stomach disturbances, have been attributed to dragging on the duodenum by the mesonephron, while frequency of micturition and tenesmus, renal colic, and suppression of urine may be referred to transitory hydronephrosis or kinking of the ureter, and constipation to dragging on the colon. There is dull aching or dragging pain in the loin, shooting down the thigh and towards the umbilicus, aggravated by exertion or constipation and relieved by lying down.

As to the severity of the symptoms excited by movable kidney, and the relief afforded by nephrorrhaphy or nephropexy, no one who has witnessed the severest forms of renal colic, the great general disturbance of health, and the nervous anxiety caused by the feeling of dragging or of something dropping from the loin to the groin, can any longer doubt either the reality of the suffering or the benefit derived from the operation.

The curative effects of the proceeding are most gratifying if the sutures are made to pass into the kidney-substance and the loose fibro-cellular capsule is shortened and stitched also to the muscles and fascia of the loin. Tuffier stated at the Surgical Congress in Paris, when speaking on the distant results of renal surgery, that in every case of nephrorrhaphy the result was perfect when the operation was clearly indicated.

RENIPUNCTURE IN ALBUMINURIA.

In several instances in which exploratory incisions or punctures have been made in a kidney in the presence of albuminuria, but on account of symptoms of other local disease, even where no other condition than that of nephritis has been found to exist, disappearance of the albuminuria has been noted.

In three cases recorded by Harrison, of London,—two by Newman and another by Hoeber, of Hamburg,—disappearance of the albumin was noted to have followed operation in every instance.

Harrison argues from these examples that inflammatory tension is the cause of albuminuria in acute nephritis, and that its relief by surgical means in the instances recorded explains the disappearance of the albuminuria by the relief afforded to the engorged kidneys.

All the cases were instances of nephritis due to scarlatina, cold, or influenza, in which the kidneys on exploration presented evidence of great tension and engorgement, suggesting the presence of pus, and the recovery both from the acute symptoms and the albuminuria was in each case complete and permanent.

The mode of operation recommended is that pursued in simple exploration of the organ by a small lumbar incision and the introduction of the finger; further procedure involving division of the capsule or renal substance depending upon the degree of tension as manifested by the plumpness and hardness of the kidney.

The time for performing the operation is determined by the course of the symptoms, and especially by an arrest of improvement as regards the quantity of urine passed and the degree of albuminuria after the acute signs of fever and hæmaturia have subsided.

In the cases recorded the reduction of tension in one kidney appears to have relieved the opposite one, as the general improvement in the patient and in the composition of the urine could be attributed only to restoration of the integrity and the functions of both organs.

NEPHRO-URETERECTOMY.

Tubercle, like cancer, must be removed completely if any permanent good or even temporary relief of a satisfactory character is to be effected. A case recorded by H. A. Kelly in the Johns Hopkins Hospital Bulletin, 1896, illustrates a proceeding which may advantageously be adopted in those instances, usually of a tuberculous character, where it is necessary to remove both the gland and the duct. Several published cases by myself and others further contribute to the elucidation of the subject. (See *British Medical Journal*, April, 1895. Hunterian Lectures by the author.)

The ureter is frequently the seat of active tubercular disease concurrently with infection of the corresponding kidney.

RESECTION OF A PORTION OF THE KIDNEY.

In view of the difficulty of determining with accuracy the condition of the opposite kidney, in cases where local disease is discovered, by an exploratory incision in the loin, as well as on account of the liberty afforded for operative interference on the other side should occasion demand it, resection of a portion only of the kidney is a measure to be encouraged when possible.

Instances where partial operations can be undertaken with advantage are afforded by the development of non-malignant growths, such as adenoma, especially when they occur at one extremity of the organ, traumatisms attended by hemorrhage, and in strumous or inflammatory deposits which partially affect both organs. I have in several instances removed wedges of kidney-tissue affected with tubercle from the human kidney with excellent results, in one case as many as four wedges from the same kidney.

The fear that was at one time entertained of the difficulty in restraining hemorrhage has been found to be unfounded, and the experiments of J. Rose Bradford show that, far from sections of the kidney exerting an inhibitory influence on the secretion of urine, the removal of wedges from the kidney in the living dog has been followed by an actual increase in the amount of urine passed.

O. Bloch, of Copenhagen, has published in the *British Medical Journal* for October 17, 1896, a report of such a tumor removed from the kidney of a youth of seventeen, with complete recovery, which was maintained nine months after the operation. Brief notes of ten other cases where various degrees of resection were practised without removing the entire organ are appended. The decision as to whether the whole or part of a kidney is to be extirpated turns upon much the same principle as the surgeon applies in the case of tumors of the extremities involving bones or joints. Partial operations in cases of malignancy are not to be recommended, and the possibility of recurrence is more to be dreaded under these circumstances than the possibility of insufficiency in secreting power in the opposite organ.

RENAL COLIC IN INFANTS.

An explanation of severe paroxysmal attacks attended by pain and disturbance of micturition in infants of from a few months to three years of age, in whom no evidence of true calculus could be found, has been put forward by Dr. Gibbons, physician to the Grosvenor Hospital for Women and Children in London. The cause of such attacks he finds to be excess of uric acid, which agglomerates into masses the size of a hemp-seed with the aid of mucus, and gives rise to spasm in its passage down the ureter.

The condition occurs in both boys and girls the offspring of gouty parents, and affects members of the same family, the paroxysms taking place at intervals of a few months and lasting from one to three days. In a typical attack the child is seized quite suddenly, while apparently in good health, with a severe screaming fit.

The legs are drawn up on the abdomen, vomiting occurs, the testicle is drawn up in boys on one or both sides, the temperature is usually raised, though it may be normal or even subnormal during the period of collapse which succeeds, and the pulse and respiration are hurried.

The child lies on the healthy side, and is extremely apprehensive of movement or palpation, especially of the affected loin, which appears to increase the suffering. The urine is clear and scanty, and is either passed at frequent intervals with great pain or altogether withheld till there is comparative distention of the bladder.

On examination it is found to contain mucous corpuscles, blood, and albumin, with epithelium from the kidney and ureter and abundance of uric acid crystals, lozenge- and rosette-shaped, and agglomerated into masses with mucus the size of a hemp-seed. These latter features of the urine are commonly more marked in the later stages of the attack or after it has subsided. The disorder appears to be uncommon among the children of the poor, and is therefore not met with in hospital. The association with gout and lithic acid manifestations, including calculus, in other members of the family is very constant. The remedies which appear to be efficacious are hot bathing, poultices, and a mixture containing compound tincture of camphor, ammonium bromide, and carbonate of lithia.

VESICAL CALCULUS.

By B. FARQUHAR CURTIS, M.D.

THE USE OF CYSTOSCOPY AND THE RÖNTGEN RAYS IN DIAGNOSIS.

SINCE the publication of Dr. Hunt's article (vol. iii. p. 596) little has been added to our knowledge of the causes of stone, but the use of the *cystoscope* has become general and has cleared up the diagnosis in many doubtful cases and given much positive information about others. The small size of the urethra in boys, however, will greatly limit its usefulness in them. The recent discovery of the application of the *Röntgen rays* to surgery also offers a new means of diagnosis of urinary calculi which would be especially suitable in the case of children, as the very translucent pelvic bones would interfere less with the shadow of the stone than the thick and opaque bones of the adult. The best method of avoiding the confusion of the shadow of the calculus with that of the bone is to take the skiagraph in an oblique direction, the tube being placed above the umbilicus instead of directly over the bladder, so that the shadow shall be projected obliquely towards the anus in the space between the tuberosities and the coccyx. The method will undoubtedly prove useful, but needs much improvement before it can become really practicable.

ADVANCES IN TREATMENT.

The operative treatment of vesical calculus both by litholapaxy and by suprapubic cystotomy has received wonderful impetus of late years. It cannot as yet be said, at least in this country, that any definite conclusion has been reached as to the relative merits of the two methods, and we shall endeavor to enable the reader to judge for himself by putting him in possession of the latest advances and the results claimed for each. The choice of operation will depend largely upon the locality where the surgeon lives, for this will influence the number of cases which he may have under treatment, and upon which depend his experience and skill. The most important contributions in regard to litholapaxy have come from the English surgeons in India, for calculus is so prevalent in the latter country that among boys alone over two thousand cases a year come under treatment. It is to these surgeons that we owe the present popularity of the crushing operation in boys, an operation which was formerly considered very difficult and dangerous, but which they have put upon a substantial footing for daily use. The difficulties of the operation in boys are caused in the first place by the small size of the urethra, which compels the use of very delicate instruments and prevents the introduction of a tube sufficiently large for rapid evacuation of the fragments; and, secondly, the long etherization, which is not well borne by young children. That these very serious difficulties do not prevent the success of the operation in the hands of experienced surgeons is evident from the reports which follow.

RESULTS OF LITHOLAPAXY IN BOYS.

The popularization of the crushing operation for stone in children is largely due to Surgeon D. F. Keegan, who since 1890 has been publishing his results, and in the spring of 1894 had performed the crushing operation in two hundred and thirty-nine cases with only five deaths. The average age of the boys was 6.4 years, the average weight of the stone removed was 98.44 grains, the rate of mortality was only 2.09 per cent., and on the average only 4.16 days were spent in hospital after operation. Keegan also reports the statistics of J. Forbes Keith, a surgeon at Hyderabad, who has operated six hundred and ninety-eight times in native children and boys under fifteen years of age with only four deaths, his last four hundred and twenty-seven cases having been all successful. This series includes some crushing operations through a perineal incision.

Surgeon P. J. Freyer records his results in the treatment of children in the second edition of his work on "The Modern Treatment of Stone in the Bladder by Litholapaxy," and they are almost as good as those of Keegan. Freyer has operated upon one hundred and sixty-five boys by litholapaxy, the average age of the patients being seven years, the average stay in the hospital being five and two-thirds days, the average weight of the stones being ninety-five grains, and there were only two deaths. He quotes a personal communication from Keegan to the effect that the latter has records of seven thousand six hundred and ninety-four cases of litholapaxy in boys, with a mortality of 3.45 per cent. The conversion of Freyer to litholapaxy is all the more remarkable in that he has operated one hundred and ninety-seven times in male children by lateral lithotomy with only one death. Surgeon George W. P. Dennys reports eighty-nine cases of litholapaxy in children with only three deaths, and these he ascribes to want of experience on his part. Finally, Keegan reports on all the operations for stone performed on boys under fifteen years of age in the Punjab, India, during the year 1895. In five hundred and nine cases of litholapaxy the average age of the patient was six and one-third years, the average weight of the stone removed was 151.54 grains, and the mortality was only 2.35 per cent. This is a very remarkable showing, because, as he states, "not a few of the operators were native assistant surgeons and hospital assistants," and therefore the result may be taken to be that obtainable by the average practitioner in India. An Anglo-Indian surgeon writes to Keegan that an operator of experience would remove the stone by litholapaxy in children in nineteen cases out of twenty, only the twentieth case requiring a cutting operation.

Complications and Dangers of Litholapaxy.—The bad condition of the patient, extreme youth, and great size of the stone appear to present but little difficulty to these expert lithotritists. Thus, Freyer states that he never refused to operate upon any patient, no matter what his condition might be, some having been apparently moribund when placed on the operating-table; and although kidney-disease was frequently present, this was no barrier to the operation of litholapaxy. He once operated upon a child only eighteen months old, passing a No. 5 (English) canula with ease and removing a stone which weighed only three grains. Recovery followed uneventfully. He has removed stones weighing two hundred, three hundred, and even seven hundred and sixty-five grains from young children, the last being in a child of nine years. In one of his cases, in a lad of fifteen, the stone weighed more than three and a quarter ounces, and he states that the amount of manual labor necessary in dealing with these large calculi was so great that his hands were often blistered and his arms frequently ached for days afterwards. Of the stones removed by Keegan many were of large size, and in illustration of this fact we may quote the case of a boy five years of age who had suffered from stone for four years. The urethra received a No. 10½ (English) lithotrite, but it was very difficult to grasp the stone, and only after many attempts, chipping pieces from it here and there, was it finally broken. The stone was exceedingly hard, and the operation lasted two hours and a half; the fragments, however, were finally removed; they consisted of phosphate and oxalate of lime, and weighed when dried seven hundred and three grains. For two days there was much pain on passing water, and also tenderness over the region of the bladder and in the urethra, but the urine remained clear, and on the third day the pain had become trifling. Five days after the operation the boy had no urinary symptoms of any kind, and left the hospital cured. In one case Keegan removed a stone of oxalate of lime weighing four grains from a boy five years of age without an anæsthetic, the operation lasting eight minutes; but he says that this is exceptional, and that a general anæsthetic is almost invariably necessary, cocaine being of little value.

To illustrate some of the difficulties that are met with by surgeons of considerable skill, we may instance one of Keegan's cases which was sent to him after another surgeon had unsuccessfully attempted to crush the stone four days previously, the calculus proving to be a very hard one of oxalate of lime and uric acid, and weighing about one hundred and ninety-eight grains. Keegan completed the operation, but we mention the case to show that even in India difficulties may be present which the average surgeon is unable to cope with. It may be well also to relate one of Keegan's fatal cases. A boy four years of age had had symptoms of stone for three years, and the meatus was so small that it had to be enlarged in order to pass a lithotrite. A hard mulberry calculus weighing one hundred and eighty-four grains was crushed in fifty-four minutes without difficulty. The next day there was a rise of temperature in the evening to 101.6° F. and some pain, but the urine was clear. The following day the temperature reached 104.4° F. On the fourth day after the operation there was pain in the abdomen, and the latter became tympanitic. The boy died with symptoms of peritonitis eight days after the operation. The post-mortem examination revealed "a low form of pyæmic inflammation in the peritoneum" with a large quantity of pus, and pus was also found in both the pleural cavities. The bladder would seem to have been healthy, for it contained no pus; the ureters were not dilated, but one kidney was slightly granular. There was no evidence of inflammation in the bladder or its ligaments, and Keegan was inclined to think that the pyæmic process began in the incision made to enlarge the meatus.

The fatal cases are particularly useful for consideration in this country, where experience on the scale obtained by the Indian surgeons will never be possible, and therefore it is worth while to note also the details of some described by Dennys. In one case the hilt of the aspirating canula broke off, and, no other being at hand, the operation was abandoned, leaving some fragments in the bladder. A fragment became impacted in the mouth of the bladder, convulsions set in, and although lithotomy was performed, the operation was too late to save the child's life. In another case a fragment of the calculus became impacted in the eye of the canula, where it acted like a ball-and-socket valve, allowing water to enter the bladder but not to escape. He removed the canula, but while he was trying to clear it with a piece of wire, the bladder burst, and the child died in consequence. In another case, working with a damaged rubber bulb in the aspirator, some of the fragments were not removed from the bladder, although it was thought that the latter had been emptied, and subsequently a small fragment became impacted in the urethra, convulsions set in, and the child died. This last case illustrates one of the dangers of the operation in these little patients and the necessity for completing the operation at one sitting, for the urethra may be blocked by a piece of the stone if all the fragments are not removed, and fatal convulsions may result.

RESULTS OF SUPRAPUBIC LITHOTOMY.

As with litholapaxy, so with suprapubic cystotomy, the results obtained have greatly improved. Although we agree with those who object to its universal adoption as the sole operation for stone, we do not think that the statistical results as published correctly represent the facts as to the mortality of the operation, especially in children. Americans should feel the same national interest in suprapubic section as in Bigelow's operation of litholapaxy, because it is better suited for the general surgeon who does not have a large number of cases in which to obtain great dexterity with the lithotrite, and because Dulles's paper written in 1875 has always been one of the classics in the literature. Dulles's paper reported on four hundred and seventy-eight cases, one hundred and thirty-three of which were in persons under twenty-one years of age, and the mortality was twenty-one per cent., which fairly represents the results in the preantiseptic era. In 1894 Barling published the results of lithotomy and litholapaxy obtained in a considerable number of English hospitals from the five years 1888 to 1892, making a total of seventy-two cases under twenty years of age, with fifteen deaths, of which three were not due to the operation, and deducting these three cases, the mortality is 17.4. It is worth while to note the causes of death in these cases. Two died from shock, two from urinary infiltration around the bladder, three from degeneration of the kidneys, two from peritonitis, and one each from pyæmia, septicæmia, bronchitis, and pneumonia.

The similarity of this rate of mortality to that of Dulles's tables, and even the mere inspection of the list of the causes of death, suggest that the operation has been charged with many deaths which do not rightly belong to it, and which were due to lack of proper antisepsis. It is also to be remembered that in England, where litholapaxy is the favorite operation, only bad cases are reserved for suprapubic section. Barling's figures in children under ten years of age give one hundred and sixty-six cases with fourteen deaths, or a mortality of 8.4 per cent. It is a very curious fact that the mortality appears to have increased of late years in children, for, according to Thompson, who collected eight hundred and fifty cases in children under ten years from 1790 to 1840, with forty-nine deaths, the mortality of this early period was only six per cent. Barling erroneously attributes this rise in mortality to the revival of suprapubic lithotomy, whereas it is evidently due to the popularization of litholapaxy, for lithotomy used to be performed even in the simple cases, but now it is reserved for the difficult cases, while the small stones in healthy bladders are generally crushed. J. William White, in Dennis's "System of Surgery," combining the statistics of Barling and Cabot, estimates the mortality for the suprapubic section in patients under puberty at 13.1 per cent., calculated upon a total of six hundred and thirty-seven cases.

Even this figure we believe to be incorrect, and in support of these views we cite the following series of cases from various European surgeons showing the possibility of better results. Thus, Assendelft, in a total of seventy-six cases of suprapubic lithotomy in children under fifteen years of age, had only two deaths; Couvée reports fifteen cases with no deaths; Alexandroff, quoted by Rasumowsky, reports eighty-five cases with four deaths; and Rasumowsky reports nine cases with no deaths.

We regret that no general statistics are available, especially from American experience; but it appears to us certain that if such large series of cases can be treated with such excellent results, the rate of mortality quoted previously must be incorrect or must be due to some special cause. These good results just mentioned are not to be explained by any such consideration as the great skill of the operator, because suprapubic section is not an operation requiring any great skill, and its results depend upon a careful training in asepsis and in the principles of correct after-treatment. Nor can it be charged that they are due to the nationality or race of the patient, although it is well known that Russian patients have great resisting power to surgical operations. In England one series of thirty cases in children without a death is reported from a single hospital.

In our limited experience, the only dangerous symptoms in quite a number of operations by suprapubic section have arisen in operations for tumor of the bladder in which injury is done to the bladder itself, and large wounds and prolonged operations are necessary, or in cases of very large stone, or when the bladder was in a very foul condition from long-standing cystitis, or the kidney was damaged. In children the cystitis is usually not of a very infective type and the kidneys are comparatively healthy, so that these complications are not likely to arise, and we have generally seen the patients not only survive, but recover without a rise of temperature. In the majority of instances of suprapubic section, especially in children, it is possible to secure primary union of the bladder wound, and in such cases the time of healing will be shortened to a fortnight, or at least the abdominal wound will be reduced to a mere granulating surface by that time. Inquiry among a number of surgeons in New York has resulted in unanimous agreement of experience upon these points. It appears to us, therefore, self-evident that the high mortality of the English operators in suprapubic section for stone rests mainly on the fact that they select only the bad cases for this operation, as they hold that cutting for stone is indicated only when the stone is large, when the bladder is in bad condition, or when the patient's health is such as to render an operation with prolonged anæsthesia out of the question. Even of these cases the simpler ones with the small stones are selected for lateral lithotomy by the majority of English surgeons, and the suprapubic operation is reserved for the worst cases. Under these circumstances it is not surprising that the mortality is so high in comparison with that of those surgeons who submit every case to the cutting operation, and in whose results the true mortality of the latter is shown. A side light is thrown upon this question by the fact that whereas the general mortality from lateral lithotomy is five per cent. when the easy cases are submitted to litholapaxy, Freyer performed the lateral operation one hundred and ninety-seven times with only one death when he was not using litholapaxy at all in these cases. It is probable, therefore, that if all cases in children were subjected to the suprapubic operation, and no stones were crushed, the mortality would be about the same as that of litholapaxy. In support of this view we quote the opinion of Dittel, who has operated for stone one hundred and ten times by the suprapubic route, selecting his cases according to indications very much in accord with the English ideas. He had a mortality of fifteen per cent. in his last series of thirty-two cases, but he estimates that if he had also done the suprapubic operation in the uncomplicated cases of stone which he preferred to reserve for litholapaxy, the mortality would have been reduced to five per cent.

RECURRENCE OF STONE.

It may be as well to remark that the relative mortality of these operations is the most important point to settle in any discussion of their respective merits, for the probability of recurrence after either is not very great. Recurrence after operations for calculus may be the result of overlooking another stone or a fragment of a stone at the time of operation and failing to remove it, or it may be caused by the formation of a second stone. The former accident is much more likely to occur in litholapaxy, but of course no operation can guard against the latter danger. As a matter of practical experience, however, statistics go to prove that recurrence is no more liable after litholapaxy than after lithotomy, probably because the recurrence is usually caused by the formation of another stone.

THE CHOICE BETWEEN LITHOLAPAXY AND LITHOTOMY.

Next in importance to the rate of mortality and the liability to recurrence in deciding the choice of an operation for vesical calculus is undoubtedly the experience of the individual surgeon. It should be remembered that American surgeons will probably never have the experience in crushing stones which has been obtained by their Anglo-Indian brethren, and it cannot be denied that litholapaxy, which always requires a special training, is much more difficult in children than in adults, in spite of their relative good health and endurance of operative procedures. Even Keegan says that "the crushing of a stone in a boy's bladder is not an operation which every surgeon in this country [England] is expected to be able to perform. It is an operation for those who have enjoyed unusual opportunities for perfecting themselves in the use of the lithotrite, and it is not on all-fours with operations of emergency, such as tracheotomy. . . . No surgeon who has been deprived of opportunities for learning the use of the lithotrite need be ashamed to confess that he feels himself unable to perform litholapaxy in young boys." He accompanies this statement with the claim that such patients should be transferred to the care of one skilled in performing litholapaxy, an argument which will hardly hold in America, because the opportunity for gaining great experience in that practice here is rare. In another place Keegan says that "the surgeon who meets with cases of stone only at rare intervals during his career will be acting more wisely if he adheres to lateral lithotomy or suprapubic cystotomy," and adds that "no one should attempt to perform it [litholapaxy] until he has first gained some practical experience of it in adult males."

There can be no doubt of the fact that a cutting operation is abhorrent to the lay mind, whereas the traumatism from crushing a stone, although it may be equal to or far greater than the cutting operation, as it is concealed inside the urethra and bladder, does not excite the same feelings, and therefore crushing will always be more popular than cutting. But it should not for that reason alone win our professional approval; and, without attempting to decide this doubtful question even for the moment, we cannot avoid stating our belief that the average American surgeon will attain better results from the suprapubic operation for stone in children than by attempting the crushing operation, but that the latter is allowable if the surgeon has had considerable experience in litholapaxy in adults.

Lateral and Suprapubic Lithotomy compared.—When a cutting operation is decided upon, should the lateral or the suprapubic route be given the preference? The choice between suprapubic and lateral perineal lithotomy depends not merely upon the mortality but upon the after-consequences to the patients. The mortality for the two from existing statistics would seem to be very nearly equal, that for lateral lithotomy being 5.2 per cent., according to Barling and also according to Keegan's statistics of operations done in the Punjab in 1895, embracing a series of three hundred and seventy-three cases altogether. We have already given our reasons for believing that the mortality of the suprapubic operation would not be greater than that of the lateral method, and would probably be less. So we may at least assume that there is nothing to favor lateral lithotomy in this respect. Of the immediate dangers which are to be charged against the suprapubic operation, those of peritonitis and urinary infiltration have generally been given the first place, but even such an opponent of this operation as Barling admits that they have been overestimated. Urinary infiltration also occurs after perineal lithotomy, and probably the difference between the two in this respect is not great.

What secondary consequences are to be charged against these operations? In suprapubic lithotomy there is the possibility of a urinary sinus which may persist for months, but is of rare occurrence. Its formation probably depends upon too long maintenance of suprapubic drainage, and it has a tendency to close spontaneously; but if it should persist, a trifling plastic operation would undoubtedly effect a cure. A urinary fistula is certainly more frequent after the lateral operation than after the suprapubic, and it is very much more difficult to cure by a plastic operation. Incontinence of urine persisting for some time is a frequent consequence of perineal lithotomy, and wounds of the rectum and of the seminal vesicles or their ducts are also not infrequent. As has been considered in Dr. Hunt's article (vol. iii. p. 596), the frequency with which impotence follows this operation is probably overestimated, but there can be no question that this lamentable condition is not very uncommon.

On the whole, we may conclude that suprapubic lithotomy is an easier operation than the perineal operation, involving no more danger to life and considerably less danger of disagreeable consequences.

Vesical calculus is rare in the female. Freyer estimates that it occurs once for every thirty cases in the male. Litholapaxy is the best method for removing stones from the bladder in girls, unless the calculi are of unusually large size or very adherent to the walls of the organ. If a cutting operation is necessary, the suprapubic method is to be preferred. In litholapaxy it may be necessary to have an assistant compress the urethra around the instruments in order to prevent leakage of the fluid from the bladder.

OPERATIVE TECHNIQUE.

Preparations.—It will be well to give some details as to the performance of the modern operations. In the first place, all the precautions of aseptic surgery must be observed. The instruments must be thoroughly sterilized, and it is most convenient to do this by boiling in a one per cent. carbonate of sodium solution. The hands of all persons who are to take part in the operation, as well as all irrigating fluids, lubricants, silk, catgut, sponges, towels, and so on, must be sterilized by the ordinary methods.

The patient must be prepared for operation by a thorough shaving of the parts, including the perineum, washing them with soft soap and applying a dressing wet with a solution of 1 to 1000 bichloride of mercury, which should be left in place from two to six hours, being inspected every half-hour to see that the skin is not too much irritated. In babies this preliminary sterilization should be omitted because of the danger of the absorption of the mercury, and also because it is impossible to keep the parts free from urine. In these cases the pelvic region and lower part of the abdomen should be thoroughly washed with soft soap and water at least four times in the twenty-four hours preceding the operation, and covered with dry aseptic gauze in the interval, which must be changed whenever it is wet by the urine.

The bladder and urethra should have been thoroughly cleansed by frequent irrigations for at least twenty-four hours beforehand, and if a foul cystitis is present, several days should be devoted to this preparation and also to the administration of salol, which is excreted with the urine and assists in rendering it aseptic. This preparation is even more important in the crushing than in the cutting operation. The fluid used in the irrigation may be the normal salt solution or Thiersch's boro-salicylic solution in one-quarter or one-half strength. After the anæsthetic is administered, the bladder should be given a final washing before the operation is begun. It is a matter of indifference in children whether ether or chloroform is employed as the anæsthetic.

The Operation of Litholapaxy in Boys.—The lithotrite used must necessarily be small because of the small size of the parts. Freyer states that the lithotrites should vary in size at the angle from No. 5 to No. 10 or even No. 12 of the English scale, and the evacuators from No. 6 upward. The evacuating-tube should not be over seven inches in length, as an increase in the length of a tube may impede the flow of fluid through it as much as a reduction of its diameter. The size of the urethra in children varies very much, and it is stated that it is not unusual to see a child of five or six years of age in whom a No. 10 lithotrite can be passed with case. It has been remarked that the early occurrence of puberty in India has undoubtedly favored the use of litholapaxy in that country. Freyer states that he has found that the narrowest part of the urethra in these small patients is the anterior two inches of the penile portion. The largest possible lithotrite should not be employed in any case, because the urethra is apt to swell from the congestion, any irritation making the instrument fit more tightly towards the close of the operation. It is considered absolutely necessary to have the lithotrite constructed with a large fenestra in the female blade, so that there shall be no danger whatever of clogging by fragments. The evacuators employed are slightly curved at the tip, and a great variety of both lithotrites and evacuating-tubes should be at hand, in order to enable the surgeon to use the largest possible instrument in every case. The Indian surgeons seem to dread the division of the meatus, and resort to it only when it is absolutely necessary, although it is difficult to understand how it can add materially to the danger if asepsis be thoroughly carried out. It would, perhaps, be well to make this incision a few days previous, and thus lessen the likelihood of dangerous consequences. The lithotrite is necessarily very delicate, and may be ruined during an operation upon a very hard stone, so that a second instrument should always be at hand.

The operation is done in the ordinary way, but the stone should be crushed as completely as possible before any fragments are removed with the evacuator, in order to diminish the number of times which a tube needs to be inserted into the urethra, and so lessen the traumatism to that canal. The operation should be completed at a single sitting, no matter how large the stone may be, and the necessity for this precaution has already been indicated by the cases related in which blocking of the urethra by a fragment produced a fatal result. Freyer states that in his experience he has almost invariably found the stone lying with its long diameter in an antero-posterior direction, and in the case of very large stones it may, therefore, be necessary to turn them around before they can be taken in the grasp of the lithotrite. Milton, who has had a large experience of litholapaxy in Cairo, Egypt, but mostly in adults, points out that in very hard stones the best plan is to screw the lithotrite firmly upon the stone, wait an interval, tighten the screw a little and wait again, and repeat this process until finally, and usually in one of the intervals of waiting, a loud report will be heard and the stone will be found to have split. He points out that a hard stone may be expected when its growth has been slow without cystitis, the stone being movable in the bladder and ringing very clearly under the stroke of the sound. In cases in which the bladder is tightly contracted around a large stone it can usually be separated from the latter by slowly acting hydraulic force. A fountain syringe with a very small nozzle should be employed, and a pressure of twenty inches above the bladder tried first and gradually increased to thirty and even fifty inches in some cases, but allowing abundance of time for the bladder to dilate, for time is a very important element of success. It is seldom that the complication of a stone in a diverticulum or projecting into the ureter is met in children, but it is not uncommon to have a part of a stone project into the urethra. The stones which project into the bladder from a diverticulum or the ureter can sometimes be seized by their vesical end with the lithotrite and slowly twisted out of the pocket and crushed, but such cases are not satisfactory for litholapaxy, and suprapubic section is preferable. Stones which lie partly in the urethra can generally be pushed back into the bladder and then crushed as usual.

If spasm of the bladder takes place during the crushing, the lithotrite must be left at rest until the spasm is over, and if it occurs while the evacuator is in the bladder, the fluid should be allowed to escape from that organ. If these precautions are not observed, there is danger of rupture of the bladder. Sometimes the last fragments are very difficult to find, but they can be enticed gradually towards the orifice of the urethra by employing the suction force of the aspirator, when the canula is to be withdrawn and the lithotrite introduced and made to seize the fragment as it lies at the neck and to crush it at once. In some cases it may be necessary to introduce a finger into the rectum to remove the stone from some pocket or direct it into the jaws of the lithotrite, or to displace some fragment which has gone to an outlying part of the bladder. At the beginning of aspiration the canula should be kept in the centre of the bladder above the base and moved about slightly in order to completely evacuate the fragments, but towards the end it should be depressed towards the base, and finally the canula should be turned over so that the eye on the concave side can be directed towards the fragments which may lie in this situation. Towards the end of the evacuation the hand should rest a few seconds after each compression of the bulb, in order to give the débris time to settle again about the catheter, and then the bulb may be allowed to expand and aspirate them. Freyer also points out that in these little patients, on account of the small size of the evacuating-tube and the comparative weakness of the stream thrown through it, the click of the fragments against the tube is not so distinct as in the case of adults, and a very careful search is necessary in order to make sure that all fragments have been removed, for complete removal is particularly necessary in children, as has already been explained. The after-treatment of a case of litholapaxy is simple, consisting merely in abundance of drinking-water, rest in bed, and irrigation of the bladder daily or twice a day until the urine becomes clear. The active treatment will be needed only for a few days.

Suprapubic Cystotomy.—Distention of the rectum by the rectal balloon is seldom necessary, as a mere distention of the bladder, especially in a child, will usually carry the peritoneal fold up above the pubic bone and bring the extra-peritoneal part of the anterior wall of the bladder within reach. Some surgeons have claimed that it is not necessary for the bladder to be very completely distended if the incision through the abdominal wall be carefully made so as not to wound the peritoneum, even if it should present itself, for the peritoneal fold can then be sufficiently retracted with the finger-tip. Other surgeons prefer air for distending the bladder, but it is difficult to measure the amount of inflation. It is preferable to place the child in the Trendelenburg position, although this is not absolutely necessary. A table can easily be improvised for this purpose by taking a wide board long enough to reach from the patient's knee to the head, and elevating one end of this upon a suitable support (a chair or box) resting upon the operating-table, so that it shall be held at an angle of about forty-five degrees. The patient is placed upon the inclined plane with the head downward and the legs, bent at the knees, hanging over the upper end, which should be covered with a folded blanket.

The incision is made in the median line, and probably the surgeon will prefer to stand on the left side of the patient, so that the right hand shall be in position to introduce into the bladder if necessary. The incision through the skin should extend from the upper border of the pubic bone for an inch and a half or more directly upward, and should be carried, if possible, through the linea alba. A slight opening of the sheath of the muscle upon either side is a matter of indifference. The pyramidal muscles must often be separated by the scissors on account of the interlacing of their fibres. The incision should be cautiously deepened until the properitoneal fat is reached. The finger is then inserted into the lower angle of the wound, close behind the pubic bone, and worked downward through the fat until the anterior wall of the bladder is felt, when the tip of the finger is hooked backward against the anterior wall and, with the finger-nail, the properitoneal fat, and with it the fold of peritoneum if it extends down so far, is stripped up from the anterior wall of the bladder and pushed into the upper angle of the wound. The wall of the bladder, when it is uncovered by peritoneum, can be recognized by its pale fleshy color, the large veins on its surface, and sometimes by the distinct muscular fibres.

The anterior wall and fundus being thus well exposed, a short, strongly curved needle threaded with stout silk is passed through it on one side of the median line. The ends of the silk thread are then knotted so as to make a long loop, and the needle introduced again on the other side of the median line in the same manner. In passing the needle large veins must be avoided. Tension being made on these two loops, an incision is made between them in the median line or a little to one side in order to avoid a vein which is often found in this situation. The loops of thread serve as retractors to hold up the bladder when it collapses, and the finger must be inserted immediately when the incision is made, to allow thorough exploration of the interior of the bladder before all the water escapes. The size of the stone is ascertained, and if it is small enough to pass through the incision, a pair of lithotomy-forceps is inserted and the stone removed. The incision should be made larger if necessary to permit easy extraction of the stone, as it is important that the edges of the bladder wound should not be injured. The wound may be extended upward as far as the peritoneal fold, which can usually be recognized by careful inspection of the parts in the upper angle of the wound, and also downward through the anterior wall of the bladder towards the neck, but not too far, for troublesome hemorrhage may take place from the circular veins which lie very near the sphincter. The wound should be enlarged with the scissors and not torn with the stone. If the stone is very large and the wound in the abdominal wall too narrow to admit of easy removal, the insertions of the rectus muscle to the pubic bone on each side should be divided transversely close to the bone. The transverse incision is recommended for all cases by some authorities, but it is not necessary unless the stone should be of immense size, and it involves the danger of a hernia developing later in the scar.

If the urine is very foul, before opening the bladder the edges of the wound should be protected by covering them with sterilized vaseline and packing gauze into the cellular space between the bladder and the pubes, or by powdering the raw surface with aristol, which will form an impenetrable film; the urine, however, should not be very foul in these cases, as the preliminary treatment of washing out the bladder removes the greater part of the mucus and pus.

SUTURE OF THE BLADDER.

After the stone has been removed, if the mucous membrane of the bladder is found to be in good condition and is free from ulceration, the wound of the bladder may be united by sutures introduced in the Lembert fashion. The needle should be a full-curved one, either a Hagedorn or an ordinary surgical needle, and the finest silk should be employed. The edges of the abdominal wound must be strongly retracted and the bladder held up by the strong sutures already passed. The first suture is introduced at the lowest angle of the wound on the anterior wall of the bladder, the needle picking up only the muscle and taking only a small bite on each side. This stitch is tied, but the ends are left long in order to hold up the anterior wall of the bladder. A series of these sutures is passed about an eighth of an inch apart until the wound has been completely united. The ends are then cut short and a second tier of sutures passed outside of them, reinforcing the first tier. The two loops of silk thread should then be cut and removed, and if they have torn the bladder-wall during the retraction, two or three Lembert sutures should be introduced at those points. The external wound should be packed with iodoform gauze and an external dressing applied.

After-Treatment.—The after-treatment will vary according as the wound in the bladder is sutured or left open. If it is closed by sutures, a catheter should be secured in the urethra and connected with a siphon-tube the end of which lies under the surface of some fluid in a jar by the bed. Some surgeons prefer to have the catheter passed at regular intervals,—every two hours at first. Others allow the patient to evacuate the bladder in the natural way. Catheter drainage, however, appears to be the safest method, and daily irrigation should be carried out in any case. If the bladder is to be drained by the wound, we prefer to suture the wound in the bladder around the tube, as this hastens the closing. The best drainage-tube is a soft rubber catheter of large size, secured so that it will just reach to the base of the bladder and yet not touch it. It can be tied in by the silk retracting threads and then connected with the siphon-tube. The rest of the wound should be firmly packed, and the dressing must be changed every six hours at first.

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MALFORMATIONS OF THE PENIS, URETHRA, AND BLADDER.

By De FOREST WILLARD, M.D.

WHILE the operative treatment of malformations of the genito-urinary organs has not materially changed since the issuance of the first volumes of this Cyclopædia, yet the psychical and other manifestations produced by any alteration of these organs are more and more appreciated as clinical experience has demonstrated the potency of their influence.

The subtle and far-reaching influence of the sexual apparatus has been noted for many years, but the mental perversions (sometimes permanent in their influence) have been but recently fully comprehended. The relief, therefore, of malformations becomes the duty of every practising physician and surgeon, and such relief should be offered before the child arrives at an age when he is subjected to the annoyance of thoughtless and unkind playmates. Well-planned and skilful surgery can often greatly assist in improving the physical, and with it the mental, condition of these unfortunates, and the family physician should be prepared early to give sound, sensible, and judicious advice.

Greatly as the removal of sound organs is to be deprecated under ordinary conditions, yet, where childbearing is absolutely impossible, the removal of potential organs before the age of puberty will prove a blessing both physically and psychically.

Absence of the scrotum is seldom found without other deficiencies, but the testicle may be in the canal, as in cryptorchidism, or this organ may be displaced into the perineum or into the fatty tissue of the buttocks; again, both organs may be found in one sac when the other is vacant. When in this position they must be uncovered by operation and brought into the scrotum if this sac is present, the cords being left intact.

ADHERENT PREPUCE; ELONGATED OR CONTRACTED PREPUCE; PHIMOSIS.

Added clinical experience confirms the assertions made in volume iii. that adherent and contracted prepuce, both in the male and in the female, has a most decided influence in producing, through irritability of the genital nerves, those neurotic conditions manifested by malnutrition, choreic or irregular muscular movements, uncertain or staggering gait, vesical irritation, incontinence, and many kindred disorders.

By interference with the growth of the glans, and by retention of the hardened smegma, both neurotic and erotic tendencies may be developed. Fortunately, the mania for the removal of this useful preputial protection of the glans penis in the young male has largely disappeared, and its sacrifice is not indicated unless resultant symptoms show that it is exercising a deleterious influence. Early attention to the freeing of the glans from the adherent fold of the prepuce has abundantly demonstrated its feasibility in the first few weeks of life. Subsequent cleansing by the mother will be sufficient to prevent readhesion.

The methods of operation have been fully described in volume iii.

Adhesion of the prepuce in the female is sometimes the cause of defective as well as of excessive sexual desire, and freeing of the hood from the glans clitoridis is followed by normal conditions.[1]

Both in males and in females the most healthy condition, and the one least likely to lead to masturbation and irritation, is a freely movable prepuce which can readily be cleansed.

In feeble-minded children, where the power of self-control is weak, special care should be taken that the glans penis be freed from any adhesion, in order that genital irritation may be lessened. When this cannot be obtained by stripping, dilatation, or incision, circumcision is warranted. The local application of cocaine, or of eucaine, is often all that is required for operation, especially when the primary puncture of the hypodermic needle is rendered painless by the free use of ice and salt, or by ethyl chloride, or when infiltration anæsthesia by Schleich's fluid is employed.

HYPOSPADIAS.

The question of necessary operation will be governed by two considerations: first, the primary importance of securing marital fertility to the individual; second, the appearance of the organ and the psychical effect of the deformity.

When the meatus is near the glans penis the semen can be deposited within the vagina, and fecundation will be probable. When the corpus spongiosum is short, and the meatus is in the anterior portion of the urethra, the semen may be discharged by recurvation in an outward direction. The canal can sometimes be straightened by the individual himself by traction upon the dorsum during coition.

Lengthening the corpus spongiosum is an exceedingly difficult matter, as recontraction often occurs for a considerable period after the performance of a plastic procedure.

When the opening is far back, even in the anterior scrotal portion of the organ, and particularly when it is posterior to this point, fecundation becomes impossible, and operative treatment is imperative. The building of a urethra along the under surface of the penis is beset with many difficulties. The frequent exit of urine, the disturbing power of erections, the extremely attenuated condition of the skin secured for flaps, and the difficulty in maintaining asepsis, all enter into the causes which so frequently result in failure.

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[1] Bernardy, Transactions of the Philadelphia County Medical Society, 1894, p. 427.

The urine may be temporarily brought out through a suprapubic or perineal opening.

The operative treatment as advised by Duplay is described at length in volume iii., and still continues to be a favorite method of procedure; but partial failure must be expected in a very large proportion of cases. The patient's friends should be impressed with the fact that repeated attempts, even five or six, may be necessary before success is assured.

Zoller reports twenty cases, requiring fifty-two operations, twenty-nine per cent. of which were secondary; seven were completely cured, eleven were partially cured, and two were absolute failures. Failures are often due to lack of complete and free dissection of the flaps which are brought from the sides of the penis. There must be no tension upon these flaps, or the stitches will give way in the thin, attenuated tissues which are involved in this operation.

If single silver wire quill sutures are employed, the shot must be exceedingly small, lest sloughing be produced by their weight. The tissue must not be drawn too tight, or the same result will ensue.

The safest plan is to maintain the existing urinary orifice until after the anterior portion of the tube has been manufactured.

Laurent[1] describes an operation in which the lateral borders of the defective urethral walls are dissected free and sutured together in the median line, after which the penis is slipped beneath a flap cut from the thigh and abdomen, which flap is left temporarily as a bridge. The under denuded surface is then turned up and fastened to the fleshy surface of the thigh strip.

EPISPADIAS.

Two methods of procedure are possible: to unite the freshened edges of the gutter of the penis, as planned by Dieffenbach, Duplay, and Krönlein,[2] or to close the gap by flaps taken from the neighboring tissue, as proposed by Nélaton, Dolbeau, Thiersch, etc. Krönlein divides his operation into two parts: first, the formation of the glandular portion of the urethra according to the method of Thiersch; second, the formation of the penile urethra, at the same time closing in the fistula by thorough freshening of the surfaces. He employs knotted sutures with intermediate platted sutures.

Thiersch's method consists, first, in the creation of a perineal fistula in order to divert the stream of urine. Second, closure of the gutter in the glans, the incision being made upon both sides with a sharp angle inward, so that in the middle there is a wedge-shaped piece. The border of the gutter is freshened on the side of the incision, and the flaps are drawn over and united by twisted sutures. Third, the sides of the penile portions of the gutter are dissected back, forming a right-angled flap on both sides of the gutter; one flap has its base inward, the other outward. The flap is then turned over so that the skin side is underneath, and the two flaps are brought across and sutured by means of knotted sutures in such a manner that the faces of the flaps are brought into apposition. Fourth, closure of the remaining defect between the canal of the glans and the canal of the penis. For this purpose a perforating window is made through the entire prepuce. Longitudinal sections of the area of the break between the glandular and penile urethra are freshened, thus leaving a floor for the tube, after which the glans is pushed through the preputial window, the edges of which are stretched asunder and sutured to the freshened surfaces. The posterior part of the prepuce is stitched to the border of the wound. Fifth, the closure of the posterior part of the canal is effected by two flaps taken from the abdominal wall, the first flap being in the form of an equilateral triangle with the base at the upper half of the crevice. This is turned over with the skin side inward and united with the freshened upper margin of the penis. The second flap is taken from the abdominal wall of the other side, and is four-angled. This is turned across the raw surface of the first flap and sutured there, the resulting raw area on the abdomen being allowed to heal by granulation. Sixth, after the completion of these operations, which usually requires four months, the perineal fistula is closed.

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[1] Bulletin Académie Royale de Médecine, Belgium, Bruxelles, p. 629, 1891.

[2] Beiträge fur klin. Chir., Tübingen, Bd xi, 1894, H. 1.

EXSTROPHY.

Exstrophy is so commonly associated with epispadias that the subjects must naturally be considered together. Complete closure in cases of considerable magnitude is uncommon, but a sufficient protection for the prolapsed posterior bladder-wall can often be secured, and at the same time the orifice is narrowed so that some appliance for receiving the urine becomes feasible. In such cases, as in hypospadias, several operations are required, the flaps being formed from the region best nourished, and which can be most satisfactorily turned into the opening.

Rein turned the ureter into the rectum in a female, the ureter being reached through an abdominal incision while the patient was in the Trendelenburg position.

Sonnenburg[1] dissected the bladder from above to below, almost to the level of the ureters, and resected the posterior wall of the bladder. He then detached the terminal extremities of the ureters and attached them to the inferior angle of the wound at the base of the penis.

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[1] Arch. Gén. de Méd., September, 1894.

DISEASES OF THE TESTES AND PENIS.

BY JOHN CHALMERS DA COSTA, M.D.

IN this section we will discuss some points in regard to Hydrocele, Retained and Ectopic Testicle, Gonorrhœa in Children, and Urethral Stricture in Children, the discussion being supplementary to Dr. Sturgis's excellent article in volume iii. of this work.

HYDROCELE.

In dealing with a hydrocele bear in mind that the case may be acute or chronic, and that the mere term hydrocele does not indicate to which group of cases, anatomically considered, a patient should be assigned. A child may have an ordinary hydrocele of the tunica vaginalis; a congenital hydrocele, in which the funicular process opens freely into the peritoneal cavity; an infantile hydrocele, in which the funicular process is closed above but open below; a congenital funicular hydrocele, in which the funicular process is closed below and open above; or an encysted hydrocele of the cord, in which the funicular process is closed above and below, but patent between the two points of closure. Gosselin tells us that encysted hydrocele of the testicle and epididymis is not encountered in children.

Acute Hydrocele, or Acute Vaginalitis.—This is an acute inflammation of the tunica vaginalis alone or of the tunica with the epididymis or testicle. Vaginalitis is the preferable term, and acute hydrocele is an undesirable designation. It may be caused by wounds, bruises, or the continued pressure of a firm truss. Various febrile maladies can produce it, sometimes causing inflammation of the serous membrane alone, sometimes attacking both this membrane and the testicle. We occasionally find acute hydrocele during or after scarlatina, typhoid fever, influenza, mumps, acute rheumatism, and erysipelas.

Morris tells us that infantile masturbation occasionally develops it. Intertrigo is a cause, though an infrequent one. Urethral inflammation by affecting the epididymis may lead to acute hydrocele. The condition often passes away practically without treatment, but in some cases it persists and leads to a chronic hydrocele. An old hydrocele may inflame, but such a condition is spoken of as inflamed hydrocele and not as acute hydrocele.

An acute hydrocele is treated by rest, elevation of the part, the application of lead water and laudanum or ichthyol, the use of an ice-bag, and occasionally by multiple punctures made with a clean tenotome after asepticizing the scrotum. If punctures are made, we must afterwards seal them with iodoform collodion.

In treating chronic hydrocele it is necessary to know with which form of the complaint we are dealing.

Congenital Hydrocele.—In these rare cases apply a truss in order to obliterate the free pathway into the peritoneal cavity. It is not wise to puncture until the neck is obliterated. In Mr. Poland's case of early tapping death followed. After the neck is obliterated, if the hydrocele does not pass away, multiple punctures aseptically applied will cure the case. If the attempt to obliterate the neck by pressure fails, asepticize the parts, incise, ligate the funicular process at the internal ring and cut it off, cut it off above the testicle, remove the process, and sew the cut edges of the process below so as to form a tunica vaginalis.

Congenital hydrocele is often associated with hernia. If the hernia is reducible, it is pushed up and the neck of the sac is obliterated by truss-pressure. If it is irreducible, the parts are exposed by incision, the funicular process is opened, the hernia is restored, and the operation is completed as in ordinary congenital hydrocele, with the addition that the cord is transplanted to between the external oblique above and the internal oblique and transversalis below (Bassini's operation for the radical cure of hernia).

Congenital funicular hydrocele is treated in the same manner as congenital hydrocele, except that stitches are not necessary to make a tunic, because of the tunic being already formed by the closure below effected by nature.

An *infantile hydrocele* often gets well spontaneously. It is held by some surgeons that absorption is favored by the administration of iodide of potassium and by the local use of collodion, ichthyol and lanolin, lead water, vinegar and water, or a solution of sal ammoniac (of a strength of gr. x to ℥j). Morris tells us to build up the general health of the mother and the child.

If these simple means fail, multiple punctures aseptically made will cure the case, although it may be necessary to repeat the operation once or twice.

Encysted hydrocele of the cord can usually be cured by multiple punctures. If these fail, incise with aseptic care, paint the interior of the sac with pure carbolic acid, and drain for a day or two with iodoform gauze.

Hydrocele of the tunica vaginalis has been treated by many methods. In rare cases multiple punctures or simple tapping bring about a cure, but such a result is not to be anticipated. Many surgeons tap and inject an irritant. A great variety of materials have been used as injections (iodine, port wine, corrosive sublimate of a strength of 1 to 5000, alcohol, etc.).

The author does not believe in this plan. It often fails to cure. Occasionally it produces violent inflammation or even sloughing of the scrotum or sac, or suppuration of the sac.

Tapping without drainage is not safe. We can tap and then drain by introducing a strand of catgut through the trocar and into the sac (Quattro-Ciocchi's method).

The surest and safest method is incision. Asepticize the parts. Open the sac, dry its interior, paint it with pure carbolic acid, pack it with iodoform gauze, and dress the parts with sterile dressings. In a couple of days withdraw the gauze packing and allow the wound to granulate. This simple operation is usually all that is necessary in children, because the sac is thin and collapsible. If the sac happens to be thick, the surgeon should excise the parietal layer of the tunic by the method of Volkmann.

A hydrocele with a retained testicle is cured by the operation which is performed to transplant or remove that organ.

Undescended and Ectopic Testicle.—Surgical opinion has shifted of late in regard to the advisability of attempting the transplantation of undescended or ectopic testicles. The older surgeons leaned almost unanimously to the view that the gland should be let alone if it caused no trouble, and should be removed if it became painful or inflamed. The elder Gross was of the opinion that attempts to place the organ in the scrotum were certain to prove futile.

One reason for this pessimistic view was the general conviction that a retained or ectopic testicle was not capable of becoming truly functional. So Curling asserted, so Gross taught, so Holmes, Godard, Folin, and others stated. This idea has apparently received confirmation from the veterinary surgeons, who tell us that in cryptorchid stallions and dogs the testicles are invariably functionless. Lately Griffiths has reaffirmed the old view on the strength of experiments upon dogs. He replaced the testicles into the abdomen in several young dogs, and found that whereas the glands continued to develop up to puberty, they failed to produce spermatozoids. Broca does not believe the organs to be functional. Curling acknowledged that such persons might become capable of intercourse, but believed that they were invariably sterile.

A mass of evidence of another character has accumulated which, in our opinion, renders the usual view untenable. That in many cases the gland is not active may be with confidence asserted, but that it is functionless in all cases cannot be for a moment admitted. In most instances the gland is at first normal, and may continue normal for many years, although in the long run it usually becomes functionless because of pressure and repeated attacks of inflammation. It may be functionless at puberty, and it may not be functionless at the age of forty-five (White's case), but the older the patient the greater the likelihood that the testicle has been destroyed.

The author operated in the Jefferson Hospital upon a boy of fourteen in whom the right testicle was in the abdomen and the left was in the inguinal canal. The left testicle was brought into the scrotum by operation. It has remained there ever since (four years), and the boy is now virile, although the right testicle still remains in the abdomen.

Beigel reported the case of a man of twenty-two with double cryptorchism who had perfect power in connection and whose semen appeared healthy. (Quoted by Jacobson in "The Diseases of the Male Organs of Generation.")

Valette removed a testicle which was lodged in the vaginal canal, and on examining the vas found that it contained healthy semen.

Jacobson says that there are at least five reported cases in which persons with bilateral retention of the testicles have had children. Curling cynically suggests that in such cases there was a real and a supposed father, but this is only a suggestion, and the proofs of paternity would satisfy a court of law. Monod and Arthaud assert positively that in many such cases the testicles are functional. White and Martin state that an undescended testicle which they removed from a man forty-five years of age in the course of an operation for strangulated inguinal hernia was found to be small but fully functional, although "it had been subject to a number of inflammatory attacks." ("Genito-Urinary and Venereal Diseases," by J. William White and Edward Martin.)

Johnson studied the records of eighty-nine post-mortems on supposed cryptorchids. In eight no testicles were found. In sixteen cases the glands were examined with the microscope, and in three cases contained semen. Ten of these individuals had had children.

It seems to us certain that in a boy with ectopic or retained testicles these glands may be normal, but as years go on there is a great risk that they will be destroyed by pressure, by attacks of inflammation, or by sarcoma; hence in some cases we should try to transplant them into the scrotum, and should do so, if possible, before puberty,—that is, before destruction is apt to take place. The glands are less likely to be destroyed in abdominal retention than in inguinal retention or in ectopy.

In cases where the testicle is retained within the abdomen transplantation is often impossible and always difficult. It is quite impossible when the gland is far posterior; it is possible when it is in the iliac region or back of the abdominal ring. In a case of bilateral abdominal retention Guelliot succeeded in bringing one testicle into the scrotum. Two years later the youth possessed sexual power. Nevertheless, when we consider the risk of operation and the fact that testicles retained within the abdomen are not exposed to injury as are testicles retained in the canal or ectopic testicles, and the fact that such subjects may be sexually active, we are loath to advise operation. It occasionally happens that the testicle will descend even after years of retention. Curling prevented such descent, which he regarded as a misfortune, by the use of a truss. It is wiser to let the testicle descend if the tendency to do so exists, and then try to transplant it if the descent fails to become complete. If there be a hernia in a case of abdominal retention we should certainly operate to cure the hernia, and can determine during the operation for hernia if transplantation is possible. If in a case of abdominal retention a tumor forms or severe attacks of pain occur, the testicle must be exposed by incision. The gland is transplanted if it is in good condition and if transplantation is possible; it is removed if badly diseased, and even if not badly diseased when it cannot be brought into its normal position. If the scrotum is deficient on the side of the retention, transplantation is not feasible.

In crural ectopy some surgeons try to push the testicle into the abdomen and keep it in by the use of a truss. Other surgeons remove the testicle. It has been suggested by White and Martin that there is no insurmountable surgical obstacle to transplantation, and we would be disposed to make the attempt if the gland was apparently healthy, the cord was long enough, and a scrotal cavity existed. It would, of course, be necessary to cut Poupart's ligament. If the testicle is diseased or cannot be transplanted, remove it.

In perineal ectopy transplantation is usually possible. In this condition the testicle is much exposed to injury, and if hopelessly damaged it should be removed; otherwise it should be placed in the scrotum, if there is a scrotum to receive it. In this form of ectopy the cord is always long enough to permit of shifting the position.

The incision in Annandale's case was carried from the external ring down the side of the scrotum for one-half the length of that bag; the end of the testicle was found, and, after adhesions were divided, the gland was drawn up into the wound, placed in the scrotum, and anchored by catgut sutures. (Annandale, *British Medical Journal,* 1879, vol. i.)

An inguinal retention may pass away as late as the sixteenth year, but it is unsafe to wait. An inguinal retention may be paroxysmal, the testicle appearing at times outside of the ring, and retiring at times into the canal. In such a case it is wise when the testicle appears to draw it down as much as possible and prevent its return by applying a truss over the ring. (Wood and Holmes.) It is in cases of inguinal retention that stroking is supposed to cure, but it may be laid down as certain that a testicle which does not sometimes appear itself cannot be brought down by stroking or traction. In inguinal retention it is possible in most cases to transplant the gland. All do not agree to this. Broca is opposed to operation if there is no hernia and if no complications exist. ("Maladies de l'Enfance," Paris, 1892, p. 531.)

Jacobson says, "We can operate when the testicle is normal, when the cord is not too short, and when a scrotal pouch exists." It is hard to tell when a testicle is normal. Bierbaum held that when there is normal testicular sensation the gland is not atrophied. When there is any doubt about the condition of the testicle, do not sacrifice it, but save it on the chance of success. When a hernia exists, there is a unanimity of opinion that the hernia should be cured by operation, but a difference of opinion as to whether the testicle ought to be transplanted or removed.

When to operate upon a retained or ectopic testicle is a question in dispute.

Broca operates after the third or fourth year when hernia exists, but operates at once when complications exist.

Terillon operates from the ninth to the fifteenth year. (*La Semaine Médicale*, January 13, 1893.)

On this subject, as in everything he has dealt with, Jacobson speaks most lucidly and logically. Recognizing that we are operating to *prevent damage*, he is in favor of operating between the eighth and tenth years. He operates earlier in poor people, as they cannot have proper care, in the victims of hernia because of the great danger of strangulation, and in those in whom complications arise because complications may destroy the testicle. He never operates before the second or third year, because the stitches are apt to tear out and the wound is likely to become infected.

The Operation of Transplantation, or Orchidopexy.—The operation is often successful. Julaguier operated on fifteen cases, and the operation was successful fourteen times. Many plans have been tried. Wood and Annandale incise, make a bed in the scrotum, place the testicle in place, and suture it.

Cheyne, after replacement, antagonized the tendency to slip back by placing a thread through the cord and fastening this thread to a wire frame, thus making traction.

Keetley sutures the divided tunica vaginalis above the testicle and then stitches the tunic to the fascia lata.

Lucas-Championnière, after replacing the testicle, narrows the ring and upper part of the scrotum by suturing.

Jacobson directs us to proceed as follows. Open the serous sac, to see if it communicates with the peritoneum. If the sac is closed, extirpate it. If the sac is open, divide it above the testicle by circular incision, tie it at the internal ring, and remove it. Suture the lower part above the testicle so as to form a tunica vaginalis.

If a hernia is present, Jacobson is disposed to sacrifice the testicle. The author is persuaded that this is unnecessary, because in his own case a hernia existed, and after restoring the testicle a radical cure was performed with ease.

The next steps are to loosen the testicle from adhesion and to prepare a bed for it in the scrotum by forcing the finger down into this bag. If the end seems short, make steady traction and try to bring the testicle down. If this is impossible, adopt Wood's plan: dissect the globus major from the testicle, and leave only the lowest part of the epididymis attached to the testicle. The testicle turns upside down, and we gain from one and a quarter to one and a half inches. This has been done by Wood, Morris, and the author. Invert the scrotum, pass a suture through the tissues of the inverted scrotum and through the tunica albuginea and part of the testicle, and tie. This suture should be of silk, and should not pass through the skin. A counter-opening for drainage is made through the lower and posterior part of the scrotum. Finally, the cord is sutured to the pillars of the ring (being careful not to surround the vas or the artery of the vas), the ring is narrowed, and the superficial structures are sutured. (Jacobson's "Diseases of the Male Organs of Generation.")

In a young child, soiling of the wound is certain to occur if the ordinary dressings are used, and it is well to cover the incision with iodoform collodion and then surround the parts with sterile gauze.

When inflammation occurs in a testicle retained in the inguinal region, we should treat the case as we would any other inflammation, and as soon as the symptoms subside operate.

The chief complications of a retained or ectopic testicle are:

Inflammation of epididymis or testicle.
Gangrene.
Peritonitis (in abdominal cases).
Hernia.
Atrophy.
Hydrocele.
Hæmatocele.
Sarcoma.

GONORRHŒA IN CHILDREN.

Gonorrhœa is, of course, rare in children, but not quite as unusual as might be assumed. It is more frequent in girls than in boys, and sometimes the question of diagnosis is discussed in a court of law. It is important to consider the distinction between the rare gonorrhœal urethritis and the not uncommon non-specific urethritis. In boys over thirteen gonorrhœa sometimes occurs as a result of sexual intercourse, and in much younger children as a result of attempted intercourse. The period at which a boy becomes virile varies within wide limits, the variability being influenced by race, climate, hereditary tendencies, and by unknown factors. There are many authentic instances upon record of puberty at a surprisingly early age. Ruelle reported the case of a boy but little over three years of age whose organs were full-sized and capable of seminal discharge. Dupuytren reported the case of a boy three and a half years of age who had reached puberty. Many cases will be found set forth in the "Anomalies and Curiosities of Medicine," by Gould and Pyle.

In most children infected with gonorrhœa the poison has been introduced upon towels, bed-linen, the fingers, or foreign bodies inserted into the urethra.

The author saw at the Jefferson Hospital a boy of six with gonorrhœa. He had been accustomed to sleep with his two sisters, one of whom was twelve and the other sixteen years of age. The younger girl stated that her sister had had a discharge, and that she had often pulled the little boy upon her and placed his genital organs in contact with her own.

Gonorrhœa in boys is apt to be very acute; there are much pain, considerable swelling, and some fever. Complications are usual (urethritis, conjunctivitis, balano-posthitis, posterior urethritis, cystitis, and occasionally epididymitis).

The diagnosis cannot be made with certainty clinically. The microscope must be used in every case of urethritis in a child. *Acute* gonorrhœa can be recognized with certainty, but chronic or subacute cases are often puzzling. The discharge of a non-gonorrhœal inflammation contains epithelial cells, pus-cells, and many forms of bacteria,—viz., bacilli, various cocci, and even diplococci. Diplococci, if found, are seen to be few in number and small in size. They lie on or between the cells, rarely within them. We used to say never within them, but we now know that in rare instances they will be found within the cells. When stained with a two per cent. alcoholic solution of methyl-violet, they cannot be decolorized by Gram's solution (one part of iodine, two parts of iodide of potassium, and one hundred parts of water).[1]

The discharge of a gonorrhœal urethritis contains epithelial cells and pus-cells, and many diplococci of large size and of characteristic shape and mode of grouping.

The gonococci when stained with methyl-violet are easily decolorized by Gram's solution.

Steinschneider affirms that this method of staining and attempting to remove the stain with Gram's solution gives a certain diagnosis in ninety-five per cent. of cases, and that even in the remaining five per cent. the distribution and arrangement of the cells make the conclusion certain (Taylor).

Koplik tells us not to make the diagnosis of clap because we find a few diplococci, but to make it whenever we find large numbers of diplococci *within* the pus-cells.

If there be any doubt after making a careful examination, settle the question by making cultures upon chest-serum, agar, or placental blood-serum (Heiman).

Taylor sums up most impressively the entire question of the value of these diagnostic methods. He says we must be very careful in coming to a positive conclusion. Scepticism and conservatism are warranted. In acute cases, even of several weeks' duration, the result of staining will be positive, but in chronic and subacute cases it may be uncertain. The staining method is not infallible, but is subject to error in a goodly proportion of cases, because several species of diplococci are found in the urethra which resemble gonococci in shape, and which are, after staining, decolorized by Gram's solution.

From cultures, and from cultures alone, can absolutely correct information be derived, and these cultures are made upon blood-serum or blood-serum and agar-agar.[2]

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[1] Heiman, Journal of Cutaneous and Genito-Urinary Diseases, September, 1895.

[2] Robert W. Taylor, Venereal Diseases.

STRICTURE OF URETHRA.

Pure stricture is very rare in boys, but is occasionally met with as a result of acute urethritis or of traumatism of the urethra. The urethra may have suffered from a blow, or may have been injured by a fragment of calculus. In most cases there has been an antecedent acute inflammation, and in some cases a real gonorrhœa.

L. Bolton Bangs has recently investigated this subject, and thinks that some strictures result from a backward extension of a balano-posthitis. Bangs reports two cases of stricture occurring in his own practice and collects the other reported cases (Poynter's case, Abbe's case, etc.[1]).

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[1] New York Medical Record, April 10, 1897.

ANOMALIES AND DISEASES OF THE UTERUS VAGINA, AND VULVA IN CHILDHOOD.

BY WILLIAM A. EDWARDS, M.D.

IN place of the normal development of the external sexual organs, the following malformations may occur:[1] I. If the genital fissure is not formed, the skin remains entire and the vulva is absent,—defectus vulvæ or atresia totalis. In this case neither the ends of the ureters, the seminal glands, nor the intestinal canal may communicate internally, or the intestine and the bladder may remain separate, the seminal glands opening into the latter. Instances of this malformation have been found only in non-viable, immature births; several illustrations have been given by Förster, some showing not the slightest trace of a vulva, as in the acephalus sympus; others only a small pedunculated appendage, as occurs especially in the sirenian malformation. II. If, after the formation of the cloaca, the septum between the bladder and the intestine, forming the perineum and drawing the vagina down with it, does not grow downward, the cloaca becomes permanent; then the intestine, the end of the urachus, and the seminal glands empty into a common canal. This condition has been called *atresia ani vaginalis*, but incorrectly, because the intestine does not end in the vagina, the latter beginning at a considerable distance above its lower opening. III. If the uro-genital sinus remains very long and narrow, not being shortened by the descent of the united ducts of Müller, the urethra continues short and occupies an abnormally high position. This condition is to be considered the beginning of hypospadia feminæ, a true hypospadias occurring when the bladder opens, without the vagina, into the vestibule. A case of this kind has been described by Heppner. IV. If the formation of the urethra and the closure of the anterior parts of the vulva are prevented by a delayed communication of the allantois with the external surface, or if there is a defect in the anterior wall of the bladder with a cleft of the pubic symphysis, the condition is known as epispadias. Here the clitoris and nymphæ are cleft and the anterior wall of the urethra is absent. Cases of this kind have been described by Roeser, Gosselin, Testelin, and others.

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[1] Winckel.

The causes of all such arrest in the development lie in part in the relations of the respective organs themselves,—*e.g.*, a closure of the ducts may interfere with the passage of the contents, and the consequent distention may prevent normal union, producing new separation. Again, they may be caused by neighboring organs,—*e.g.*, by the omphalo-meseraic duct, by the intestines, or by abnormalities in the larger abdominal organs, such as the liver, which, owing to its great size or unusual connections, may permanently dislocate the organs beneath it.

It is certainly incorrect to speak of a congenital vulvar atresia where there is an orifice beneath the hypertrophied clitoris leading to the uro-genital sinus, though the urino-genital secretions pass out, for here there is no complete closure and the vulva is not imperforate. The conditions which Hildebrandt represents in his work (Fig. 3, page 5) do not, therefore, show an atresia of the vulva, but hyperplasia of the clitoris, persistence of the uro-genital sinus, and an abnormal shortening of the recto-vaginal septum.

V. A peculiar malformation of the external genitals is that which has been designated hermaphroditism. Here some parts of the vulva, especially the clitoris and the labia majora, are developed to an unusual degree. The labia, uniting at a higher level, present a sort of raphe, and not infrequently contain the ovaries, increasing the resemblance to the scrotum. Other parts, on the contrary, especially the nymphæ, have been arrested in development, and hence the gland is partially exposed or the prepuce is too short. Yet usually such cases are not examples of true hermaphroditism, but of individuals of only one sex. There are, however, cases of true hermaphroditism in which testicles and ovaries may be microscopically demonstrated in the same individual, but, as a rule, the parts peculiar to one sex are much better and completely developed, while the others are rudimentary.

The following combinations are possible:

(*a*) Bilateral hermaphroditism, when a testicle and an ovary are found on each side.

(*b*) Unilateral hermaphroditism, when an ovary or a testicle is found on one side and on the other both ovary and testicle.

(*c*) Lateral hermaphroditism,—that is, a testicle on one side and an ovary on the other. These cases are said to be numerous, and have been proved by the microscopic examinations of careful observers.

Anomalies of the external genital structures in viable female children are somewhat rare. Immediately at birth we not infrequently observe simple adherence of the labia minora. In the record of the births of two hundred and fifty female children under my observation, I have noted this condition nine times, or 3.6 per cent. If the adhesion is unrelieved, it may cause retention of menses in later life. Holmes and Wright consider that the majority of cases of imperforate hymen have this pathology. The case reported by Minard[1] appears to be an illustration of the correctness of this statement. Pean[1] also describes a case of what he styles vulvar imperforation in a child aged three. The nymphæ were glued together and the vaginal orifice entirely obliterated; he denuded the septum from the urethra to the fourchette in the median line.

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[1] New York Medical Journal, 1892, lvi. 299.

While the babe is young the adhesions are easily separated by a blunt probe or an Allis dry dissector; the raw surfaces are to be kept apart by gauze smeared with oxide of zinc ointment until the parts have healed. Neglect of this simple procedure will permit the formation of an acquired atresia of the vulva, a serious complication in later life.

The natives of the temperate zone rarely present hypertrophy of the external genitalia, but it is not uncommon to read the reports of hypertrophy of the labia and clitoris in children observed by physicians whose life-work is cast in hot climates. Hypertrophy of the labia majora in the new-born is, however, occasionally seen in our latitudes, and Dumas[2] records an instance of hypertrophy of this structure in a young woman of eighteen years, and Kirchoff[3] a fibroid tumor growing from the left nymphæ in a young girl of the same age, while Schtol[4] contributes a study of congenital hypertrophy of the nymphæ and the necessary surgical procedures to correct the abnormality. Winckel has observed two instances of supernumerary nymphæ, one of which is illustrated in his atlas (Fig. 3, page 265). An anomaly that is very frequent with us, however, and one of which the literature daily records more and more instances, is adherence of the prepuce of the clitoris to the glands, with retained smegma. The symptoms resemble those seen in the male under similar conditions. Grandin[5] has recently called attention to the *rôle* of the clitoris in producing neuroses when clitoridal adhesions exist. I see several of these cases every year. Leonard[6] reports the case of a girl aged eleven years who suffered from chorea due to adherent prepuce of the clitoris; the disease had lasted a year with no period of relief. All methods had been tried. She was finally entirely cured by stripping back the prepuce, breaking up the adhesions, removing the retained secretions, and treating the parts until fully cured to prevent readhesions.

Excessive development of the clitoris is usually acquired at a later period of life than would warrant a consideration in the present article; still, it is sometimes congenital, or develops at a very early period. When it is seen early it is apt to be associated with other vices of conformation. Burke's[7] two cases of hypertrophied clitoris in babes, both twelve months old, did not present, however, any other abnormalities. A bifid clitoris has been noted, and Arnand[1] has seen a double clitoris. Sonnini[2] says that women of distinct Egyptian origin have a natural excrescence, fleshy in consistency, thick and pendulous, growing from the skin of the mons Veneris. He says that a girl of eight years presented one that was one-half inch in length. The Egyptian women have been accustomed to circumcise the clitoris in children. They remove a great part of the body of the clitoris, with the prepuce and sometimes adjacent portions of the nymphæ.

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[1] Revue Obstétricale et Gynécologique, Paris, August, 1893.

[2] N. Montpel. Méd., 1895, iv. 973–976.

[3] Centralblatt für Gynäkologie, Leipzig, Nr. 45, 1893.

[4] J. Akush, i. jensk, boliez, St. Petersburg, 1890, iv. 807–822. Index Med.

[5] Pediatrics, February 15, 1897.

[6] Sajous's Annual, 1891, H. 2.

[7] Pediatrics, vol. iii., No. 4, 1897; two illustrations.

Nævus of the labia is not infrequently seen; it may be either congenital or acquired; indeed, I have under my care at the present moment a most extensive telangiectasis of the entire external genitals and inner aspect of the right thigh in a baby aged ten months, illustrated in the accompanying photograph, in which may be seen the areas of cicatrization following electrolysis. Puncture by the actual cautery or, as I prefer, by electrolysis will, with much patience, usually remove the growth, but more or less scarring will result under all conditions and in the hands of the most experienced. A curious peculiarity in these growths is that, notwithstanding their extreme vascularity, they are prone to break down under treatment, and unless extreme care is used a large suppurative surface will result. Several times in my experience this has occurred without treatment, and the children were brought to me because an open sore existed.

Hernia of the vulva is infrequent in early childhood; it is sometimes seen in early adolescence. The most frequent variety is inguinal-labial hernia, in which the hernia passes along the round ligament and appears in the labia majora. The second variety, very rare at all ages, is that in which the hernial protrusion passes in front of the broad ligament into an opening in the pelvic fascia and levator ani and protrudes at the posterior extremity of the labia majora. The hernial protrusion may contain an ovary, as in the cases of Lockwood[3] and Owen,[4] the former occurring in a healthy babe aged six months. The swelling, three inches long and one and one-half inches wide, extended from the internal abdominal ring to the lower part of the right labium majus. Operation showed that the protrusion consisted of ovary, Fallopian tube, and broad ligament. The second case was that of an infant eleven weeks old. Upon admittance to hospital a hard, tender swelling existed in the right labium majus, which upon operation was found to be caused by an engorged ovary, a broad ligament, and the Fallopian tube. In both cases the ovary was removed.

HYDROCELE OF THE LABIUM MAJUS.

A prolongation of the peritoneum may reach below the mons Veneris through the inguinal ring, covering the round ligament. This peritoneal investment may become adherent about the ring, and a transudation of

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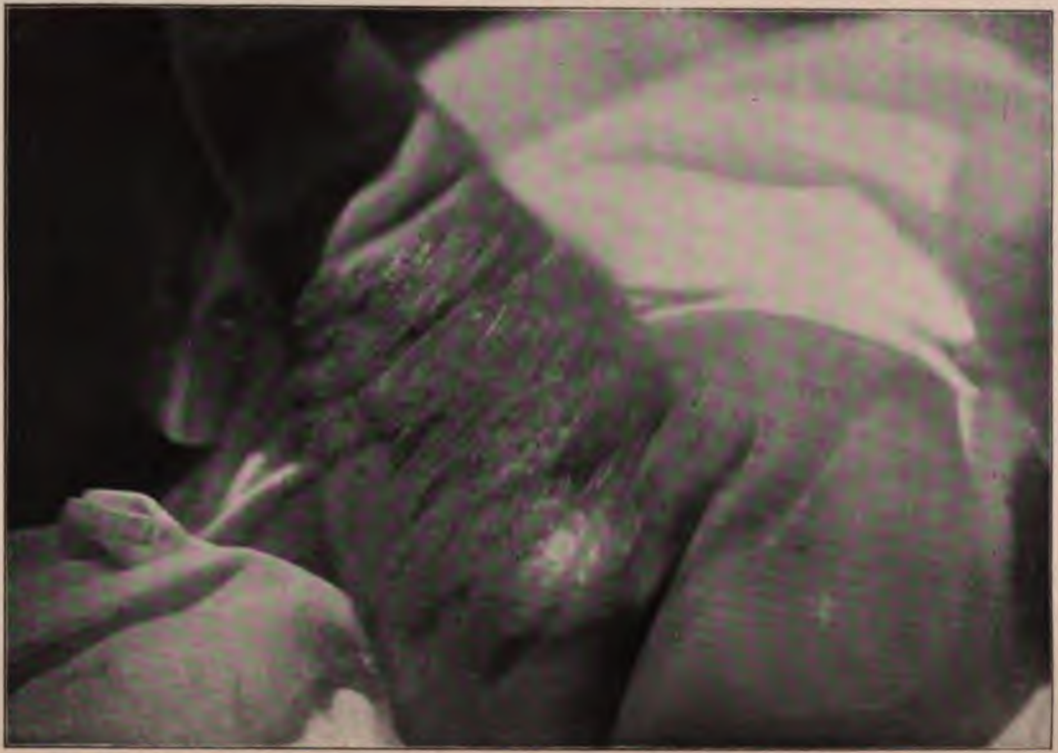
[1] Anomalies of Medicine, Gould and Pyle, p. 309.
[2] Ibid.
[3] British Medical Journal, June 13, 1896.
[4] Lancet, March 21, 1896.

PLATE I.



Telangiectasis of the external genitals and inner aspect of right thigh. Baby aged ten months.

serum occur into the cavity thus formed. The condition is then known as hydrocele of the round ligament or hydrocele of the labium majus. The labium presents a fluctuating egg-shaped tumor, more or less firm. The round or, as it is sometimes styled, the utero-inguinal ligament is a stout cord twelve and one-half centimetres in length, proportionately smaller in the child. The ligament is bilateral, and arises from or is attached to the uterus on its anterior superior surface just in front of or below the Fallopian tube. It consists of areolar tissue, dense fibrous tissue, unstriped muscular tissue, vessels, and nerves enclosed in a peritoneal covering. The unstriped fibres seem to be continuous with the superficial uterine fibres; each ligament, then, as McClellan states, "passes forward in the folds of the broad ligament to the deep abdominal opening on either side behind the epigastric artery, where it enters the inguinal tract. Here there is already provided a process of the peritoneum called the canal of Nuck, similar to the processus vaginalis, and the ligament pursues a course analogous to the spermatic cord in the male, eventually losing its character in the tissues over the pubes. In the adult there is rarely any trace of the peritoneal process or of the muscular tissues of the round ligament beyond the middle of the inguinal canal. Occasionally the canal of Nuck remains patent and may become the seat of congenital hydrocele or even of inguinal hernia." The ligament in the canal receives a small accession of striped fibres, which sometimes are attached to the pillars of the ring and to the spine of the pubis. Hydrocele of the ligament or of the labium, as you may prefer to call it, is a comparatively rare condition.

A hydrocele in the labium may be one of several varieties: 1. That just described, in which there exists a patulous canal of Nuck. The fluid is excreted from the peritoneal surfaces covering the ligament and is free to return within the general peritoneal cavity. 2. The sac may be entirely cut off from the abdominal cavity, and dropsy occur in this closed sac. Such cases have been observed and recorded by Sacchi and Fleming. 3. The cellular tissue of the labium majus consists of two layers which are prolongations of the superficial abdominal fascia. These two layers are considered the analogue of the dartos tunic. Between them a serous tumor may form. This is considered by some to be the true hydrocele in the female. 4. The substance of the round ligament itself may be the site of cyst. The gubernaculum of Hunter in the fœtus becomes the round ligament in the female. This fœtal structure is at first hollow, as demonstrated by Weber,[1] and, as Staffel[2] states, there may be a persistence of this fœtal condition which allows the formation of a cyst within the round ligament itself.

Complete absence of the labia, both major and minor, has been recorded. Scanzoni has seen it in a new-born child. Riolan has seen an infant in whom the left labia alone was absent. Congenital absence of the labia is said to be rather usual among people where the excision of these parts is a religious custom. Attention has already been called to a congenital defect in which the anus is seen immediately below the vulva, due to a complete absence of the perineum. The babe usually has so many other congenital defects that it survives but a few days or hours, or it may be a non-viable monster.

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[1] Centralblatt für Gynäkologie, 1887, Nr. 21.

[2] Ueber Cysten der Canalis Nuckii, Centralblatt für Gynäkologie, 1888, S. 273.

Premature development of the external genital organs occurs in some instances at a remarkably early age; John Lovett Morse[1] has seen a babe of nine months in whom the breasts were large, the mons Veneris prominent, and the external genitals well developed. Children of six and one-half years[2] have presented full development of these organs, and at ten not only were the external genitalia well developed, but also the mammary glands, and the menstrual epoch occurred with regularity. Olinto's[3] case was but a year and a half old, yet menstruation was established and onanism was observed. Woodruff's[4] case was one of unusually early menstruation, and in Kisel's[5] case the girl presented premature and excessive development at the age of thirteen. Collier[6] records a case of very early true menstruation. Iron's[7] case was a baby that menstruated from birth, and Howie's[8] case commenced to menstruate when three years old. Both children showed precocious development of the external organs of generation.

INFANTILE VULVAR OR VAGINAL HEMORRHAGE.

This is not to be considered a true menstrual flow. It sometimes occurs at a very early age, as in the two cases reported by McArdle,[9] in each of which the hemorrhage occurred on the fifth day after birth, lasted four days, and never returned. There was no malformation in either case. The hemorrhage is usually very slight and occurs without other complications or bleeding from other organs. It sometimes occurs when a cyst of the vagina exists; it is also seen in masturbating infants or children, and in those who have catarrh of the genital tract. Eross[10] makes a very valuable contribution to the pathology of bleeding from the genitals of the female new-born. James presented to the Obstetrical Society of London[11] the uterus and appendages of an infant with hemorrhage from the uterine mucous membrane. Comby[1] has recently given us a valuable contribution to the study of hemorrhage from the vulva and vagina in little girls, and Busey read his communication upon this subject to the Washington Obstetrical and Gynæcological Society.[2]

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[1] Archives of Pediatrics, vol. xiv., No. 4, April, 1897.

[2] Vladimiroff, Med. Obozr. Mosk., 1896, xlv. 927–929.

[3] Arch. Ital. di Pediat., Napoli, 1892, x. 262.

[4] Medical Record, New York, 1896, xlix. 338.

[5] Trudi. Obsh. dietsk. Vrach., Moskva, 1893, i. 97–106, I. pl., Index Med.

[6] Transactions of the Michigan Medical Society, Detroit, 1892, xvi. 341–347.

[7] New York Medical Journal, August, 1896.

[8] British Medical Journal, September, 1896.

[9] Medical News, Philadelphia, 1890, lviii. 399.

[10] Gyogyaszat, Budapest, 1891, xxxi. 121; also Arch. f. Kinder., Stuttgart, 1891–92, Bd. xiii. Ss. 172–174.

[11] Transactions, 1890, 1891, xxxii. 66.

ABSCESS IN THE URETHRO-VAGINAL SEPTUM.

This is a rare condition in childhood. De Barey[3] records one instance in a child one year old, and Hellier[4] reports an abscess in this region in a girl aged seventeen. This was an extensive suppurating cyst extending from the cervix to the hymen in the urethro-vaginal septum. The abscess appears as a small tumor near the vault of the vagina, and usually communicates by a small opening with the urethra, so that pus in the urine is a frequent concomitant symptom. The pus may also exude from a small opening in the vestibule just below the external meatus urinarius. The etiology of these purulent cysts is explained by the anatomy of the part. The urethro-vaginal septum may contain the remains of Gärtner's ducts. Rieder[5] has found that eight out of forty human beings possess remains of these ducts. When a portion of the duct remains until birth it will persist throughout life. Gärtner has been able to demonstrate the post-natal existence of the duct which bears his name; he has shown that it sometimes extends upward to the ovary, downward to the cervix uteri, and may open into the vagina near the urethral orifice. The duct is always in the median line, never at the lateral walls of the vagina. Dorhn has shown that the duct is lined by cylindrical epithelium loosely attached to a basement membrane; it may have two layers of cells. Cullen[6] thus formulates the possible causes of an abscess in the urethro-vaginal septum. 1. Congenital cysts, or those occurring in the new-born. The latter variety has been mentioned by Englisch,[7] who found that in new-born children small oblong cysts are occasionally present in the urethra near its orifice. He suggests that these may in after-life increase in size and give rise to the above condition. 2. A true urethral diverticulum in which all the urethral coats take part. 3. Accumulation of secretions in a urethral gland. 4. Dilatation of a lacuna of Morgagni, probably due to inflammation, closure of its orifice, and subsequent distention with secretion. 5. Dilatation and possible occlusion of Skene's tubules. 6. Arrest of calculi in the urethra, with a diverticulum forming to accommodate the same. 7. Traumatism, as a kick or injuries. Here an abrasion of the mucous membrane takes place and the urine gains access to the small pocket, decomposes, and sets up an inflammatory process. 8. A suppurating cyst situated in the urethro-vaginal septum and afterwards bursting into the urethra.

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[1] Bull. et Mém. Soc. Méd. d. Hòp. de Paris, 1896, 3 S., xiii. 719–722.

[2] Transactions, New York, 1892, vol. iii. pp. 25–32.

[3] Arch. f. Path. Anat., Bd. cvi. S. 65; Cullen, Johns Hopkins Hospital Bulletin, No. 39, April, 1894.

[4] The Scalpel, August, 1896. Year-Book, 1898. Gould.

[5] Contribution à l'étude des maladies de l'urèthre, Batuard, Thèses, Paris, 1887–88.

[6] Ibid.

[7] Wiener medizinische Presse, 1881, Bd. xxii. Ss 599–634.

The treatment is simple, and consists in removing all the cystic tissue by an elliptical incision, closure of the wound by silk stitches, and passage of the catheter sufficiently often for about four days.

Mention must be made of those curious cases of *extreme* and *complete* prolapse of the urethra in children. While they are somewhat uncommon, still they are seen sufficiently often to merit consideration in a work like this. The displacement usually consists of prolapse of the mucous membrane, although it may be complete and all the coats of the urethra suffer displacement. It is usual, however, to find that but a portion of the membrane has prolapsed. A tumor presents itself in the vestibule, more or less concealing the urinary meatus. After careful search and gentle manipulation a point will be found upon this tumor at which a sound may be made to enter the bladder. This at once makes the diagnosis perfectly clear. Blanc,[1] Bryant,[2] and Broca[3] have each recently contributed interesting papers upon these curious cases of urethral displacement in children. The treatment is simple, and consists in excision of the mucous membrane to the meatus and the introduction of cumolized catgut sutures. Winckel has seen one case in a girl of twelve, in which the prolapsed portion was nearly as large as the first phalanx of the thumb. It sloughed off and spontaneous cure resulted.

INFLAMMATION OF THE VULVA.

It is convenient to divide these inflammatory disorders into three classes,—simple, exanthemata or local, and infectious. I have already referred to telangioma.

The simple inflammations may be again subdivided into, (1) catarrhal, (2) follicular, (3) œdematous, (4) furuncular or gangrenous; the local into (1) eczema, (2) herpes, (3) prurigo; and the infectious into (1) erysipelatous, (2) diphtheritic, (3) syphilitic, (4) trachomatous, (5) tubercular.

Simple inflammations of the vulva present varied appearances, depending upon the cause and the part attacked. A simple dermatitis may involve the entire vulva; if it extends deeper and is local, it is known as a furuncle; but if this condition becomes general, we call it phlegmon, or even gangrene or noma pudendi. If the follicles alone are affected, we style it folliculitis.

Young children are particularly prone to a simple vulvitis; this form is seen very often among the lower classes, where the genitals are irritated by the urine and fæces and little attention is paid to cleanliness. Scrofula and the oxyuris vermicularis are assigned as causes; trauma, frost-bites, and diseases of other organs (vulvitis diabetica) may also cause vulvitis.

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[1] Ann. d. mal. d. org. génito-urin., Paris, 1895, xiii. 523–536.

[2] Lancet, London, 1894, i. 1189.

[3] Canadian Practitioner, July, 1896.

These children complain very much of the itching, burning, and stinging sensation; they rub and scratch the parts, and thus add to their sufferings, so that when they are presented for treatment deep excoriations and raw bleeding surfaces are often seen. The urine and fæces aggravate the condition, infections may occur, and deep abscesses form.

The disease is apt to last much longer in children than in adults. The treatment consists in cleanliness as nearly absolute as possible and the application of various drugs. Carbolic acid and camphor are serviceable; a solution of corrosive sublimate, 1 to 5000 or 8000, a one to two per cent. ointment of yellow oxide of mercury cautiously applied every second or third day, oxide of zinc, either in powder or in ointment made with vaseline, ichthyol ointment, five to ten per cent., plain or in combination with zinc ointment, are often extremely efficacious. If there are large, raw, excoriated surfaces, dusting with iodoform and boric acid (1 to 7) will often produce most happy results.

Furuncles, suppurating retention cysts (folliculitis), and phlegmon should be excised and dressed upon well-known aseptic surgical principles. It is in these instances that the aqueous solutions of oxygen are so valuable; three per cent. pyrozone will speedily cause the suppuration to cease. The special treatment for vulvitis diabetica cannot be considered here; the disease is unusual in children.

The local exanthemata of the vulva in children are identical with those in adults. Eczema may be acute or chronic, and of all its well-known varieties; it may be confined to the vulva alone, or may extend over the mons Veneris, thighs, or nates. The symptoms are those of simple inflammatory vulvitis, differing only with the degree and type of the eczema. It is usually accompanied by a purulent secretion. Its treatment is that which is appropriate to eczema and to suppurating surfaces, if these exist. Derville[1] records an instance of voluminous vegetations of the vulva in a girl thirteen years of age.

Herpes of the vulva is, in my experience, not unusual in childhood. Its appearance is the same as that of herpes of the lips. I have seen it of all degrees of severity, from a few vesicles to the formation of thick scabs and excoriations. It is usually quickly amenable to treatment, and results in a cure in a few days. Gillette[2] has observed a papilloma of the vulva occurring in a girl nine years old, in whom no evidences of syphilis could be detected.

I have seen a few instances of prurigo in children; there was a characteristic eruption of pale, discrete papules, attended with intense itching. The papules were covered with crusts of blood and serum from the constant scratching.

I have also observed the various forms of miliaria in this region, particularly the miliaria alba or miliaria crystallina, sometimes called sudamina crystallina. The miliaria rubra or miliaria strophulus is very frequently seen during the heat of summer, from too voluminous or too thick diapers and consequent sweating of the parts.

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[1] Journ. d. Sc méd. de Lille, 1893, i. 436.

[2] American Journal of Obstetrics, 1879, xii. 599.

Infectious Inflammations of the Vulva.—Syphilis will not be considered, as it is fully treated elsewhere in this work.

Erysipelas of the vulva is, or rather was until recently, a rather frequent disease of childhood, particularly in early infancy. Many years ago, in my earlier work, I saw many cases of erysipelas of the vulva as an extension of the disease which first attacked the navel during the first few days of life. Most of these cases were fatal. I also somewhat frequently saw it in the scrofulous and filthy children of the overcrowded poor districts of Philadelphia. Latterly I see it not at all.

Diphtheria of the vulva, secondary to pharyngeal diphtheria, is a comparatively common disease of childhood. I have seen one case of primary diphtheria of the vulva in a child aged two years and eight months. Winckel has seen it once in a child of from one and a half to two years of age. Coldstream's case[1] was aged twelve, and showed extensive diphtheritic membranes on both labia majora with marked constitutional symptoms. The throat was not affected. Several writers have noted it as secondary to pharyngeal diphtheria; I myself have frequently noticed it under these conditions. Jacobi[2] has seen pretty firm occlusion of the vulva and vagina as the result of diphtheritic inflammation. Hyrup-Pederson[3] reports an interesting case in a girl aged fifteen, who, during an attack of diphtheria, passed a complete cast of the vagina. Later the child developed a marked atresia of the vagina, which was cured by operation.

A disease similar to cancrum oris or noma sometimes primarily attacks the vulva, usually the labia majora. This noma of the vulva, like its analogue in the cheek, may arise as a complication or sequela of measles, scarlatina, erysipelas, typhoid and typhus fevers. In the old days it is said that one case of noma of the vulva occurred in every fifteen hundred sick children who were inmates of asylums and poorly conducted hospitals or who were subjects of epidemic measles or scarlatina. This is no longer true; the proportion is now much smaller. I have never seen a case of primary noma of the vulva.

Nicolaysen[4] believes that he has discovered a bacillus in noma that differs from the one described by Schimmelbusch.

Tuberculosis of the Vulva.—The literature contains many examples of tuberculosis of the female genitals in children. Maas[5] has collected eight cases, in some of which the tuberculous vulvitis appeared to be primary, in others it would appear that the infection was carried from the lungs to the vulva by the lymphatics, or that the child touched the vulva with fingers soiled with tubercular sputum. It is interesting to note that in some instances Maas has been able to show that a child may become infected with tuberculosis by the bacilli gaining entrance through the navel, or the vulvar tuberculosis may be secondary to a bone infection, as in the instance reported by Coudray,[1] in which there was infection of the labia majora from a tubercular osteitis of the pubis in a child of eight years. Schenk[2] has observed tuberculosis of the external genitals in a girl four and a half years of age. A small ulcer on the clitoris and enlarged inguinal glands were the first manifestations of the disease. In a month great tumefaction of the labia majora appeared. The vestibule of the vagina showed a loss of substance three centimetres by two centimetres, broad, flat, and covered by a grayish deposit. The bacteriological examination of this deposit showed tubercle bacilli present. The ulcer and inguinal glands were removed by excision. The child made a perfect recovery.

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[1] British Medical Journal, May 9, 1891.

[2] Archives of Pediatrics, February, 1891.

[3] Colonial Medical Journal; American Medico-Surgical Bulletin, April 10, 1897, p. 350.

[4] British Medical Journal, No. 1854; American Medico-Surgical Bulletin, February 25, 1897.

[5] Archiv für Gynäkologie, Bd. xii , Heft ii., 1896.

Trachoma.—This condition has been variously designated in the literature as vaginismus, vascular degeneration of the vulva and the vagina, granular vaginitis, kraurosis vulvæ. These terms do not, to me, correctly express the condition, but I prefer, as stated elsewhere,[3] the term trachoma, on account of the analogy of this condition to that seen in the eye; the pathology is identical. De Schweinitz in his book on diseases of the eye defines trachoma as an inflammation of the conjunctiva, in which the membrane loses its smooth surface owing to the formation of rounded granulations, which after absorption leave cicatricial changes. It occurs under two forms,—acute granulations and chronic granulations,—and we consider that the term trachoma is equally applicable to the disease that is about to be described. There is a curious silence in all the text-books upon this disease. Winckel remarks that perhaps its study has not been interesting enough to excite close observation. Martin[4] has described three somewhat similar cases under the title "Kraurosis Vulvæ." He also states that the disease cannot be traced to any venereal or microbian influence. It may occur in young or old, virgins or multiparæ. Sänger has also contributed a paper during the year.

With the acute granulations we have little to do, as the patients rarely come to us with this condition. All that I have seen presented the chronic forms and were of three varieties,—papillary trachoma, follicular trachoma, and mixed trachoma. I have never been able to determine the presence of a special form of micro-organism which could be looked upon as the causative agent in the production of the condition under consideration, nor am

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[1] Journ. de Clin. et de Thérap inf., Paris, 1894, ii. 283.

[2] Wiener Klin. Wochensch., 1897, x. 788. Pediatrics, June 1, 1898.

[3] Southern California Practitioner, December, 1895.

[4] Centralblatt für Gynäkologie, Leipzig, 1894.

I able at present to offer any satisfactory etiology of the disease. The usual clinical manifestations of the disease are very similar indeed to those of acute or chronic trachomatous inflammation of the eyelid. It usually begins in the region of the clitoris, extending downward over the entire vulva, and it is apt to involve the urethra and vagina. The parts present a peculiar and characteristic appearance, in places deep red, almost black, resembling a subcuticular mottling or a petechial patch and not unlike in appearance the submucous or subcutaneous hemorrhages of purpura rheumatica. The surrounding tissues are apt to be anæmic, but the papillæ stand up like sago-grains; a slight touch is apt to produce bleeding.

After the disease has existed for a time the parts become fissured and cracked more or less deeply. A thin, ichorous discharge is present, but it is always scanty; indeed, this is somewhat diagnostic.

Later in the course of the disease, like its analogue in the eye, diffuse scar-tissue results, and it is then that the disease may be called trachoma deformans. In some cases this deformity is most marked, the normal contour of the vulva is obliterated, the labia majora first adhere to the larger labia and then become obliterated by scar-tissue, the vaginal outlet contracts and the orifice is almost closed.

These patients are perhaps the greatest sufferers that we are called upon to meet, and the resources of our art do not afford them very much relief. The cases are essentially chronic in nature, and the stubbornness with which the disease persists is often most discouraging. My own plan of treatment has been to keep the parts as clean as possible either with pyrozone solutions of varying strength, saturated boric solution, or, in some cases, a twenty per cent. salicylate of sodium solution. These solutions are to be injected into the vagina after a bath speculum has been introduced, in order that all the folds and interstices of the vagina may be thoroughly cleansed. Better still, if the patient can command the services of a trained nurse, is the thorough cleansing of the genital tract like the preparation for a plastic operation. The granulations, cracks, and fissures are touched occasionally with campho-phénique or five per cent. pyrozone solution, and the parts thoroughly dusted over with compound stearate of zinc. The attendant is instructed to bathe the parts in a soda solution after each urination, and again apply thoroughly the zinc powder.

Johnstone[1] recommends an ointment of the yellow oxide of mercury, from four to ten grains to the ounce, used twice daily. My experience accords with Johnstone's in that the cases will persist from six months to two years under any and all kinds of treatment, and will usually result in great deformity.

Diseases and Deformities of the Vulvo-Vaginal Glands.—These glands may be entirely absent when the labia majora are imperfectly developed. The most frequent disease is catarrhal inflammation. Pressure will cause a tenacious mucus to exude from the distended excretory ducts. Cases of this kind occur rather frequently. In long-standing cases the mouth of the duct becomes occluded and a cyst forms which either will be globular with smooth walls if the duct alone is affected, or will be found distending the posterior external third of the labia majora if the gland itself is the site of the cystic distention. These cysts sometimes attain extraordinary size. Abscesses may arise, and, if in the duct, the perforation will be apt to occur about one centimetre below its orifice; if in the gland, the perforation always occurs on the inner surface of the labium majus. I have seen these abscesses several times in children between the ages of seven and fourteen years. Some writers say that the period of puberty is one especially liable to diseases of Bartholin's glands. The gonococcus has been found in the secretion from these abscesses.

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[1] American Gynæcological Society, May 29, 1895.

The treatment of catarrhal inflammation of the ducts and glands of Bartholin is most difficult and very unsatisfactory; the ducts are so small that it is only with great difficulty that we are able to make applications to them or to properly cleanse the tissues. On this account it is perhaps as well to excise the glands in their entirety. If a cyst of the excretory duct or gland exists it may be incised and evacuated, and if we can then restore the continuity of the duct the cyst may be permanently healed. In other cases it may be necessary to excise a wedge-shaped piece from the cyst-wall and cauterize the remainder of the surface of the cyst with trichloracetic acid, nitric acid, or iodine. In most cases it is better, however, to totally excise the cyst or abscess, much as a sebaceous cyst is removed.

THE HYMEN.

The hymen may present many anomalies of structure, form, and situation. Complete absence of the hymen is probably very rarely observed, although here, as in many other matters in our art, we find conflicting statements. Tolberg, Hartmann, Blazius, Heuermann, Lieutaud, and Rose all state that they have observed cases of this kind, whereas Devilliers, Tardieu, and Brouardel have never seen an instance of absence of the hymen in many cases examined. Winckel thinks that confusion arises in those cases in which the uro-genital sinus persists and the hymen is sought in the wrong place. A frequent anomaly is double orifice, sometimes styled duplication or biperforation of the hymen. Many cases have been observed. Winckel has seen six, and Rose, Delens, and Cornil have also recorded such anomalies. Scanzoni reports a hyperplasia of the hymen in which it projected from one to three centimetres beyond the labia majora. Winckel has repeatedly seen, and I have several times observed myself, the points of a hymen carinatus projecting from the rima of the vulva in the newborn. Boivin and Duges have had a similar experience. In a virgin of seventeen years Winckel saw the lower part of the hymen project beyond the urethra like a flap. Luschka records an anomalous hymen, the hymen fimbriatus, which presents the appearance of having been lacerated.

Congenital cysts of the hymen are occasionally met with. Winckel saw two in the Munich Clinic. Gorl[1] up to 1893 could find but six cases in the literature, all of these in infants. The cysts were situated in the median line on the outer border of the hymen near the external navicular fossa. Winckel presents two cuts of the cysts, and concludes that they were a true primary formation.

Atresia of the Hymen.—Imperforate hymen may be congenital or acquired. Atresia has been observed in very young children,—by Heilbut in one of three years and by Godefroy in a babe of two months. It causes practically no inconvenience in children before menstruation is established, and the symptoms of atresia of the hymen after menstruation do not properly find a place in this work. Godefroy's case, however, presented a fluctuating tumor at the vulva, which incision showed to contain thick, tenacious fluid, found to be mucus from the uterus. In these cases of atresia in the young child the hymen may usually be ruptured by the finger. Sometimes, however, it may be necessary to incise, make flaps, and stitch the mucous membrane to mucous membrane. It is rarely necessary to excise the entire hymen.

VAGINISMUS.

This disease is now seen in children often enough to merit consideration. It is generally conceded to be the result of a structural change in the hymen. Three varieties are described,—perineal, posterior, and vulvar. The former is due to spasm of the perineal muscles, the posterior to spasm of the levator ani muscle, and the vulvar to spasm of the constrictor cunni. It is rarely accompanied by any gross inflammatory change or lesion. The seat of the pain is apt to be either at the upper margin, near the urethra, or at the base of the hymen. The slightest touch is sufficient to cause violent muscular contractions, and in some instances excruciating pain.

Its pathology is still somewhat obscure; it is seen at all ages, and shows no special association with other diseases of the genital organs, nor does it seem to be purely neuralgic in character. Some years ago an attempt was made to show its relation to diseases of the spinal cord, but this view has not been generally accepted. Cases sometimes persist even after excision of the hymen, and in women even after multiple pregnancies. There does not at present appear to be any reasonable doubt that the seat of the disease is in the hymen itself, and that it depends upon structural alteration in the substance of the tissue, but as yet the pathology is not definite, and we are unable to say that in all cases the alterations are the same. This change usually consists of thickening, or connective-tissue hyperplasia, an increase in the number and size of the papillæ. Some of the cases studied by Winckel showed a hypertrophic fibro-papillary proliferation and a proliferation of the epithelial layer.

The treatment should be radical, and consists in total extirpation of the hymen, both posteriorly and anteriorly, if the hymen extends to and is continuous with the urethra. If any considerable portion of the hymen is allowed to remain, all the symptoms may recur. In some instances cure has been secured only by excising the urethral orifice to eradicate every vestige of the hymen. I agree with several writers who say that they have absolutely no results from any plan of treatment other than that which is radically surgical. The various ointments, suppositories, and lotions are useless. Cauterization is also valueless. Rapid or gradual dilatation has been without results in my hands.

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[1] Archiv für Gynäkologie, Sajous's Annual, 1893, H. 2, Bd. ii.

INJURIES OF THE EXTERNAL GENITALS.

With the injuries during labor we, of course, have nothing to do in the present work.

Children are apt to receive injuries to these structures by falls or blows. Many cases are reported of tears or cuts received by children falling on various objects, as chairs, sofas, or even fence-pickets. The fall may be from a height, as in the case of a child who fell from a hay-rick and was impaled on the handle of a pitchfork. The trauma may be extensive, as in Bauer's case, in which the child, while stooping, was gored by a bull, the horn penetrating the vulva, rupturing the perineum, and causing extensive laceration of the buttock. Other severe injuries in this region have extended into the rectum and a recto-vaginal fistula has resulted. Parakh[1] saw a rent in the vagina due to a fall upon the abdomen. The tear was one and one-half inches long, one-half inch deep laterally, and close to the cervix. The case is important in that it shows that rupture of the vagina may occur from an external blow which may leave no wound or other indication of its having occurred. The wounds may be of all degrees of severity. When caused by a fall, they are generally small and apt to be found in a direction parallel to the descending ramus of the ischium. Blows and falls are also apt to produce an injury of the clitoris with extension into the nymphæ. Hemorrhage is apt to be severe, in some cases alarming, and, if not speedily checked, fatal. The injuries may be self-inflicted. Masturbating children often do most serious damage to the sexual organs in the gratification of their perverted desires. I cite Bokai's case in illustration of this statement. A girl aged ten, who for a long time had practised masturbation, had for the same purpose ligated the clitoris so tightly with a thin thread that the organ swelled up to the size of an Italian hazel-nut. The thread was removed by Bokai fourteen days later and the ulcerated line of strangulation healed by proper treatment, yet the clitoris remained large, sensitive, and œdematous. The hypertrophied tissue was finally removed by the thermo-cautery.

Foreign bodies may be introduced into the vagina even in very young children, as in Smith's[2] case, in which a foreign body was inserted in the vagina of a three-year-old girl and remained until she was thirteen and a half years of age, and that of Simmovitch,[1] in a girl of eight years.

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[1] Indian Medical Rept., Calcutta; Sajous's Annual, 1894, P. G. 26.

[2] New Orleans Medical and Surgical Journal, 1883–84, N. S., xi. 592–594.

The treatment is that of all lacerated, incised, or contused wounds, and is to be governed by the well-known surgical laws for operations in this region.

THE VAGINA.

The vagina may be completely absent. Several of the older writers have recorded such cases,—Baux in 1758, Oberteuffer about 1800, and d'Azier in 1812. Bosquet describes a closed vagina and an absent vulva and uterus. Clarke's[2] case was verified by autopsy, and Cunningham also confirmed his case by post-mortem examination. Gould and Pyle in their extremely interesting book, "Anomalies and Curiosities of Medicine" (1897), say that Churchill has quoted the following observers as having mentioned the absence of the vagina: O'Ferral, Gooch, Davies, Boyd, Tyler-Smith, Hancock, Coste, Kluyskens, Debrou, Braid, and Watson. Amussat[3] saw a girl who did not have a trace of vagina, and Griffith's specimen in St. Bartholomew's Hospital Museum has neither uterus nor vagina. Ferguson describes a young girl without vagina, uterus, or ovaries. The girl had indulged in coitus through the urethra. In a girl aged sixteen, seen by Brodhurst, there was entire absence of vagina and uterus, the clitoris being abnormally developed. Many recent writers have recorded instances of complete absence of the vagina, but space forbids a further consideration of the subject. Dumitreschu[4] has contributed a study of congenital absence of the vagina from a surgical point of view.

Winckel, Maisonneuve, Lachowicz, and Otto have observed cases in which the vagina was represented by a band of connective tissue. The upper or the lower segment alone may be obliterated, the intermediate portion pervious, or fine membranous bands may obliterate the vaginal lumen; or, again, transverse septa may exist. Thus, the *atresia* may be complete or incomplete, congenital or acquired.

I have several times recorded in my notes the existence in new-born babes of a thin membranous septum or veil extending across the vagina behind the hymen. These membranous septa usually disappear during the first month of life, but I have known them to persist and require rupturing. They are apt to protrude during severe crying or straining, and in this way they come under observation. Adhesion of the mucous folds directly behind the hymen, but not connected with it, has been described as *Breisky's atresia retrohymenalis*. This has been regarded as physiologic by several writers, because it is unaccompanied by inflammatory lesions. It is usually seen in children, and resembles an imperforate hymen. It generally ruptures spontaneously, or may perhaps require surgical interference.

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[1] Yuzhno-russk. Med. Gazette, Odessa, 1893, ii. 345; Index Medicus.

[2] Lancet, London, ii. 225, 1872.

[3] Gazette Médicale de Paris, December 12, 1865.

[4] Marie Dumitreschu, Paris, 1896. G. Steinheil.

Vaginal atresia rarely presents any symptoms until the child reaches puberty, which in some girls occurs very early indeed. The symptoms then will depend upon the degree of atresia; if it is firm and complete, they are those of retention of menstrual blood and secretions. If the obstruction is thin and membranous and the accumulation is constantly added to, spontaneous rupture may occur, either through the natural channel or by some adventitious opening in the rectum, bladder, or surrounding structures. These abnormal openings are particularly apt to occur if eccentric hypertrophy of the vaginal walls has not advanced coincidently with the obstruction. Myers's[1] recent studies on two hundred and sixteen cases of vaginal atresia lead him to differ from Kussmaul's statement that ill development of the lower part of the genital tract with atresia is due to fœtal inflammation. His observations lead him to conclude that it is in infancy and childhood that inflammations occur, as vulvitis and local lesions in general infectious disorders. The vagina closes, the tissues heal and look healthy, and the atresia is not discerned until puberty, the disease being wrongly attributed to congenital origin. A further consideration of the subject would be out of place here, for the child is rapidly approaching womanhood.

Abnormal shortness or narrowness of the vagina is usually the result of a fœtal inflammatory process. Some vaginæ have been measured that are of extraordinary shortness and narrowness; Scanzoni saw one that measured but four and one-half lines.

Double Vagina.—Persistence of the septum between Müller's ducts, which has usually disappeared by the twelfth week of embryonic life, results in double vagina. Double vagina with single uterus is somewhat rare; it is more usual to find the former associated with double uterus. The literature contains many references to double vagina: Robb[2] records such an anomaly, Gould[3] cites several instances in his book, and Sänger records a supernumerary vagina which connected with the normal vagina by a fistulous opening. He did not consider this a case of patent Gärtner's duct. The halves of the double vagina may be of equal size or one may be much narrower than the other; the septum may be complete, and the deficiency, if it exist, may be either above or below.

Inversions or Prolongations of the Vaginal Mucous Membrane.—This condition I have never seen. Winckel says that the prolongations of the vaginal mucous membrane extend into the muscular layers, or even into the paravaginal connective tissues. These are partly congenital inversions of one vaginal wall or cases of unusual lengthening and widening of the mucous lacunæ situated at the sides of the columns of the vagina. They must not be mistaken for a double vagina or for the walls of a rudimentary vagina. They have thin, smooth walls, and may be 2.4 centimetres long.

Abnormal Openings of the Vagina.—Persistence of the uro-genital sinus, as already stated, is responsible for many anomalous conformations of the vagina. The vagina may open into the rectum, the so-called congenital *atresia ani vaginalis*, in which the external anus is absent, and an opening exists above the hymen into the vagina. If the anus open underneath the hymen, it is classified as *atresia ani hymenalis vestibularis*. Openings have also been seen in both the vagina and the perineum, into the bladder, urethra, and upon the abdominal parietes. These latter cases have been carefully studied by Le Fort, who has never seen the condition in a viable fœtus. Dwight[1] has reported a case of anus vulvalis, with remarks on congenital communication of the vulva and rectum. Freeman[2] makes some remarks on vulvo-vaginal anus and reports a case.

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[1] Amer. Pract. and News, March 20, 1897; Amer. Year-Book, 1898, p. 531.

[2] Johns Hopkins Hospital Reports, April, 1895. [3] Ibid.

A great deal may be done to correct these abnormal openings by careful surgical proceedings. If the opening into the vagina is not too large, it may be provided with a sphincter, and, as in the cases of Blot, Brignoles, and Jorbet, evacuation occur periodically and the patient be comparatively comfortable. Guillon's patient, aged fifteen, had an evacuation only at long intervals, and then defecation was accomplished by pressing upon the perineum and thus extruding the mass through the vaginal opening.

Radical operation offers almost certain cure in atresia ani vaginalis if the opening is not too large and not too high in the vagina, and if the case comes under surgical care early in life; provided, of course, that other anomalies with much deficiency in structure do not also exist. The operative procedures are those required to form an anus in its proper place by making an incision through the perineum and stitching the rectum to the edge of the wound. The vaginal fistula is to be closed by resorting to any of the well-known operations for such conditions which would be applicable to the case in hand. Curling reports eleven cases with ten cures.

Neoplasms of the Vagina.—Neoplastic growths are rather infrequent in the vagina, but the congenital tumors and those occurring early in childhood are comparatively frequent.

Cysts.—These are of four varieties, although as yet there is not entire unanimity of opinion in regard to their pathology. The most generally accepted are the first and the fourth. 1. Retention-cysts of the vaginal glands occur in the superficial portion of the vaginal mucous membrane, although Veit denies the existence of true glands in the vagina; he considers that the sulci between the vaginal rugæ perform the function of glands; they cause retention-cysts by union of adjacent papillæ. 2. Cysts which arise in dilated lymph-vessels are very rare. 3. Cysts due to transudation of blood or serum into the submucous tissues, usually occasioned by trauma. 4. Those that originate in the remains of Wolff's or Gärtner's canals or in the rudiments of Müller's ducts, as established by Freund and Veit, who consider them analogous to double vagina. All of these cysts are practically without symptoms other than those that are mechanical and arise from pressure. In Winckel's case, in an infant, a cyst the size of a walnut caused dysuria. The pressure may divert the stream of urine and cause it to flow into the vagina, a severe colpitis resulting. Cazin has recently contributed a paper on the pathological anatomy and the pathology of cysts of the vagina. If the cyst has thin walls, it may rupture spontaneously, but will usually refill. Resection and removal entire and intact, if possible, is the most radical course to pursue. If necessary for a complete operation, a portion of the vaginal wall also may be removed.

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[1] American Journal of the Medical Sciences, Philadelphia, 1895, N. S., cix. 433–436.

[2] Medical News, Philadelphia, 1895, lxvii. 319–321.

Polypi of the Vagina.—Vaginal polyps are occasionally met with even in very young children. Frantzl[1] has seen a polyp in these tissues in a child one year and three months old, and Wilson[2] a large vaginal polypus in a child two and one-half years old, while Marshall records a papilloma of the vagina[3] in a young child.

Tumors of the Vagina.—These comprise fibroma, fibromyomata, myomata, sarcoma, and carcinoma. All of these except sarcoma and carcinoma are rare even in women, and are very rarely seen in children, although they have occasionally been observed in girls about the age of puberty. They may be congenital or they may develop from the walls of Gärtner's canals or from an obliterated Müller's duct. Skene records a singular case of patency of a Gârtner's duct.[4] They are not particularly prone to sarcomatous or carcinomatous degeneration. Early they become pedunculated and thus escape a deal of irritation, and are usually removed before they attain any very large size. Williams[5] observed a congenital tumor of the vagina immediately after the birth of the child. The nature of the growth was not determined. The vulva was considerably redder than the surrounding tissues, and upon separating the labia almost the whole enclosed space was found to be occupied by a mass projecting from the vagina. The meatus urethræ and clitoris were forced into the anterior commissure of the labia. The tumor was paler than its surroundings and firm to the touch. There was a colorless, viscid discharge from the vagina bathing the tumor and external genitals. A dull curette passed around the tumor showed it to be about one and one-half inches long by three-quarters of an inch through. It was attached to the posterior wall of the vagina by a pedicle about three-quarters of an inch from the orifice. Pressure upon the tumor caused a passage of water from the bladder through the meatus urethræ. During the following week the child passed urine normally, as it probably had during the first twenty-four hours. A month later there was less redness of the vulva, the labia minora were in their normal position, the discharge was still present, and the vaginal orifice occupied about its proper share of territory, but projecting from it was the tumor, apparently reduced in size one-third.

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[1] Allg. Wien. Med. Ztg., 1863, viii. 238.

[2] Medical Times and Gazette, London, 1876, i. 360.

[3] British Medical Journal, London, 1889, i. 127.

[4] Medical Record, New York, 1896, xlix. 692.

[5] Boston Medical and Surgical Journal, January 14, 1897, p. 47.

Primary sarcoma of the vagina is very much more frequent. D'Arcey Power[1] records a case in a girl two years and four months old. Carpenter[2] and Hollander[3] have also observed sarcoma in very young children. The first two writers have reviewed the literature, and the table on pages 891 and 892 is taken in the main from their research.

Power concludes that primary sarcoma of the vagina in children is only a specialized form of malignant disease which may affect any or all of those connective tissues which are involved in the complicated developmental processes associated with the formation of cloaca.

The sarcoma grows in the connective tissue of the pelvic organs, and extends into the bladder, urethra, uterus, or vagina. The growth may be well circumscribed, as in Power's case, or diffuse, as in some of the others, notably Ahlfeld's. Under all conditions vaginal sarcoma in children shows a constant tendency to become pedunculated or polypoid and multiple. Its course is not apt to be rapid; it ulcerates slowly. The lymphatic glands are affected, if at all, late in the case. It disseminates very slowly. The prognosis is grave; recently, however, operative cures have been reported lasting many years, in one case for ten years without recurrence. In the main, however, it recurs quickly, and is apt to prove fatal by pressure, particularly on the bladder and rectum, causing retention of the urine and obstruction of the bowels.

There is no special trouble in the diagnosis, but unfortunately the polypi are often looked upon as benign, and the diagnosis is not made until it is too late to thoroughly remove the growth. Its early and complete removal is as effectual here as in other portions of the body.

Carcinoma of the vagina is very rare at all ages. D. Berry Hart[4] states that malignant disease of the vulva or vagina is so rare that it constitutes but about one per cent. of all malignant disease of the genital tract.

In children it is almost a curiosity, although several cases are recorded. Winckel quotes T. Smith, who found cancer of the recto-vaginal wall in a child of fourteen months. Guersant saw a carcinoma twenty centimetres long and twenty-eight centimetres in circumference growing from the introitus of the vagina in a child of three and a half years. Johannovsky discovered in the Strasburg pathological collection a carcinoma of the size of a hen's egg situated in the vaginal vault in a preparation from a girl nine years of age. B. Farquhar Curtis[5] says that there is a case on record of a cancer of the vagina in a girl only nine years of age, but he does not give the reference. Mann has reported two cases between fifteen and twenty years.

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[1] St. Bartholomew's Hospital Reports, 1896, vol. xxxi.

[2] Pediatrics, June 1, 1896.

[3] Deutsche Med. Wochenschrift, Vereins Beilage, 1896, xxii. 16; Pediatrics, vol. ii., No 10, 1896.

[4] Practitioner, London, February, 1895.

[5] The International Encyclopædia of Surgery, Ashhurst, vol. vii., 1895, p. 293.

Sarcoma of the Vagina.

AUTHOR.	AGE OF PATIENT.	REFERENCE.	SITE OF GROWTH.
Ahlfeld.	Three and a half years.	Arch. f. Heilkunde, Bd. xvi. S. 135, 1867.	Vaginal polypi; fibro-sarcoma filling pelvis; inguinal glands enlarged.
Billroth.	One and a half years.	Wiener Klin. Woch., 1889, Nr. 8, S. 159.	Warty growths in vagina and bladder; vesico-vaginal septum infiltrated; fibro-sarcoma.
Demme.	Five and a half years.	Pick (quotes in), Arch. f. Gynaek., vol. xlvi. S. 218.	Vaginal polypi; fibro-sarcoma.
Hauser.	Six months.	Virchow's Arch., Bd. lxxxviii. S. 168.	Vaginal tumors (multiple); round and spindle cells and striped muscle-fibres.
Heckford.	Two months.	Trans. Obstet. Soc., vol. x. p. 224, 1868.	Vaginal and vulval villous tumor, "medullary."
Hollander.	Nine months.	Deutsche Med. Woch., Vereins Beilage, 1896, xxii. 16; Ped. No. 10, vol. ii., 1896.	Vaginal polyp; large posterior wall, uterus also involved; spindle- and round-celled sarcoma. Operative cure by Israel.
Kolisko.	Children.	Wiener Klin. Woch., 1889, ii. 109, 130, 159, 182, 202, 222.	Vaginal polypi.
Korner.	Two years.	Pick (quotes in), Arch. f. Gynaek., Bd. xlvi. S. 220, 1894.	Vaginal polypi; vesico-vaginal septum infiltrated; fibro-sarcoma.
Marsh.	Two years.	Trans. Path. Soc., vol xxv. p. 178, 1874.	Polypi in vagina and bladder; vesico-vaginal septum infiltrated; small round-celled sarcoma.
Marshall.	Two years and seven months.	Brit. Med. Journ., vol. i. p. 127, 1889.	Vagina.
Nünz.	Children.	Frauenarzt, Berlin, 1894, ix. 297–302.	Primary sarcoma of the vagina in children and young girls.
Pick.	Two years.	Arch. f. Gynaek., Bd. xlvi. p. 192, 1894.	Vagina, infiltrating pelvic organs; spindle-celled sarcoma.
Power.	Two years and four months.	St. Barthol'mw's Hosp. Reps., vol. xxxi. pp. 121–135; Trans Path Soc., vol. xlvii.	Vaginal polypi in connective tissue right side of vagina; fibro-sarcoma and myxo-sarcoma.
Sänger.	Two years and eight months.	Arch. f. Gynaek., Bd. xvi. S. 58.	Polypoid masses in vagina, bladder, broad ligaments; deep lumbar glands infiltrated; round celled sarcoma.
Schuchardt.	Seven months.	Verhandl. der Deutsch. Gesellsch. f. Gynaek., Bd. ii. S. 239, 1888.	Vaginal tumor; mixed-celled sarcoma. Operated on by Volkman. Cure has now lasted ten years.

AUTHOR.	AGE OF PATIENT.	REFERENCE.	SITE OF GROWTH.
Schuchardt	Two and a half years.	Ibid.	Vaginal polypi; mixed-celled sarcoma.
Schustler.	Four years.	Wiener. Klin. Woch., SS. 148, 225, 1888.	Vaginal polypi; vesico-vaginal septum; myxo-sarcoma.
Smith, T. C.	Three years and eight months.	Amer. Journ. Obstet., vol. xvi. pp. 555–668, 1883; Ibid., vol. xxvii. p. 577, 1893.	Vaginal and uterine polypi; uterine subperitoneal growths; myxo-sarcoma.
Soltmann.	Two and a half years.	Jahrb. f. Kinderheil., Bd. xvi. S. 418.	Vaginal and bladder polypi; round-celled in vagina, spindle-celled in bladder.
Steinthal.	Two years.	Virchow's Arch., Bd. iii. S. 449.	Vaginal tumor; myxo-sarcoma.
Thomas.	One and a half years.	Amer. Journ. Obstet., vol. vii. p. 51.	Tumor of the left labium majus; sarcoma
Weinlechner.	One and a half years.	Wiener Klin. Woch., S. 109, 1889.	Vaginal polypi involving bladder, cervix, and urethra; myo-fibro-sarcoma.
Weinlechner.	One year.	Ibid., S. 130.	Vaginal polypi.

Rupture of the Vagina.—While a rare accident in Europe and America, this is not rare in India. Every year several cases are reported by the surgeons in the Indian service and by the medical missionaries of China and the contiguous countries. Under the system of child marriages in India, rupture of the vagina frequently occurs. Harris reports such a case in a child twelve years of age; an irregular circular rent occurred in the upper part of the left side of the vagina at its junction with the uterus. The rent extended into the peritoneal cavity. Wolfovitsch[1] has seen a traumatic lesion of the vagina in a girl of twelve years, due to coitus, and similar tears have been reported from Anglo-India in babes but three years of age, who had been married according to the customs of the country. These ruptures are almost invariably fatal.

Simple catarrhal vulvo-vaginitis is fully considered in vol. iii. p. 718, and there is nothing to add to those statements.

Infectious Vulvo-Vaginitis.—In all cases, no matter what the cause, there is a pronounced purulent discharge, which consists of epithelium and pus-cells, mucus, bacteria in large numbers, staphylococci, and streptococci. Gonococci may be absent, but their presence proves the existence of gonorrhœa. Heiman's experiments[2] have shown that it is possible in many cases to differentiate this organism from the other diplococci, although Martin,[3] in an earlier paper, seemed to think that it was not possible to do so. In chronic cases culture media alone are to be recommended for the detection of the gonococcus. Heiman also believes that a urethra may contain gonococci which lie dormant and may be innocuous for a long time, but may later become active and excite gonorrhœa in another person. Some difference of opinion still exists as to the identity of the gonococcus; for example, Sheffield's[1] views are that infectious vulvo-vaginitis in children is always of gonorrhœal nature, and that the diplococcus present in the purulent discharge is invariably identical with that of Neisser, decolorizing by Gram's method. Marx[2] has come to the conclusion that the blennorrhagic origin is the exception and not the rule. Cahen,[3] Comby,[4] Sheffield,[5] Veillon, and Halle[6] have recently studied and recorded their observations on infectious vulvo-vaginitis. In all doubtful cases and those in whom rape is suspected the secretions should be subjected to the test of culture on artificial media. In the babe or very young infant the infection is obtained from the mother either directly during labor or later by the hands, towels, wash-cloths, and the like. If it develops later,—that is, during the time the child is nursing,—it is usually due to transmission by bath or towels. The vulvo-vaginal mucous membrane in children seems to present a peculiarly acceptable soil to the gonococcus. The disease is comparatively rarely caused by criminal practices, although, of course, it does occur in this way. I quote the following from Aristides Agramonte:[7] "In view of the fact that the greatest men in the knowledge of venereal diseases are at this late date still at loggerheads regarding the specific virulence of the gonorrhœal pus, that the same state of affairs, difference of opinion, exists as to the identity of the gonococcus, and convinced that all kinds of pus are infective if not infectious, I have seldom lost time in a fruitless endeavor to find the source of infection in a given case in direct contact with a gonorrhœal male; yet, knowing that there is prevailing among the lower classes a belief to the effect that copulation or attempted coitus with a virgin child or young girl is a sure cure for all urethral inflammatory processes, I cannot but feel that the majority of cases not directly traceable to other active causes have been the victims of this infamous belief. On the other hand, we know that the healthy adult male organ, by repeated friction, may produce such an inflammation about the genitals of an otherwise healthy child as to later on superinduce a typical vulvo-vaginitis with all its accompanying symptoms; and this possibility we think ought to be borne in mind when dealing with medico-legal questions, as, for instance, when a child with severe vulvo-vaginitis justly accuses a man who on examination presents a perfectly healthy organ." The symptoms are characteristic; the urethra, vagina, and vulva are all affected. Rotch has at times noted more or less excoriation of the inner surfaces of the labia, and Agramonte a stretching or apparent tearing of the hymen, more particularly in masturbators or in those in whom an attempt at coitus has been made. A free purulent and sometimes blood-stained discharge covers the parts, which are much swollen and intensely congested, bleeding freely when handled. Urination is painful, as the urethra is early involved. The constitutional symptoms may be very slight indeed; fever may be present, although the temperature is not apt to be high. This is in strong contrast with simple catarrhal vulvo-vaginitis, in which the constitutional symptoms are most pronounced, the child being profoundly anæmic or presenting other debilitating constitutional taints; although some gonorrhœal cases present the peculiar pallor, the *anémie gonohemique* of the French, the symptoms indicating a general poisoning through absorption of the toxins of the gonococci.

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[1] Meditsina, St. Petersburg, 1893, v. 451.

[2] Medical Record, June 22, 1895.

[3] Journal of Cutaneous and Genito-Urinary Diseases, 1892.

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[1] American Medico-Surgical Bulletin, May 30, 1896.

[2] Arch. de Toc. et de Gyn., No. 10, 1895; American Gynæcological and Obstetrical Journal, February, 1896, p. 27.

[3] Verhandl. d. Versamml. d. Gesellsch. f. Kinderh. deutsch. Naturf. w. Aertze, 1891; Wiesb., 1892, ix. 212–219.

[4] Ann. d'hyg., Paris, 1892, 3d S., xxvii. 66–79; also Bull. et Mém. Soc. Méd. d. Hôp. de Paris, 1891, 3d S., viii. 395–406.

[5] American Medico-Surgical Bulletin, New York, 1896, ix. 726–731.

[6] Arch. de Méd. expér. et d'Anat. path., Paris, 1896, viii. 281–303.

[7] Medical Record, January 11, 1896, p. 46.

Most cases recover from the immediate manifestations of the disease, but there is nothing more disastrous to the female pelvic organs than an attack of gonorrhœa. I know of nothing that plays such sad havoc. It is difficult to demonstrate this in children; they pass from under our observation before the disease has sufficiently advanced to prove this statement; but such is undoubtedly the fact in women, and I see no reason to doubt it in children. Marx[1] has seen several cases in children in which suppurative salpingitis followed a purulent vulvo-vaginitis. After extensive observation this writer concludes that in nearly all purulent vaginal discharges in children inflammations occur in adjacent regions sooner or later. The effect of these inflammatory changes may not be evident until menstruation is established. Marx also states, and I fully endorse the statement, that in young girls the difficulties attending the establishment of the menstrual flow have been considered to be physiological, but that they are probably the pathological fruit from the seed sown in childhood. Pus in the tubes has been found in five cases at autopsy in girls between the ages of seven and nine years.

As in the adult, serious complications may arise; urethritis of a severe form, cystitis, and involvement of the ureters and kidneys have been observed. Again, as in the adult, the uterus, tubes, ovaries, and peritoneum may be infected. Steven[2] has observed a case of acute, rapidly fatal general peritonitis in a child, associated with vulvo-vaginal catarrh.

Gonorrhœal arthritis in children is not by any means so rare as was formerly supposed; it may occur at any age, and is perhaps more usually seen at an earlier age than rheumatic arthritis. In some cases the attack is very slight, in others most severe, and may go on to pus-formation in the joints, as in Seiffert's case in a child aged four,[1] in whom the wrists and the metacarpo-phalangeal and metatarsal joints were affected. Pus was evacuated from the wrist-joint which contained gonococci, pus-cells, and endothelial cells from the tendon-sheaths. In other cases connective-tissue inflammation occurs without joint-involvement, as in the two children reported by Cassel.[2] Lop[3] has also seen arthritis as a sequela of gonorrhœa in a child of two years. Richardiere[4] and Deutschmann[5] have observed cases, the former in a girl of seven years, and the latter in infants from twenty months to three years of age. Marfan[6] records two cases of blennorrhagic arthritis in little girls, and the literature contains many more, but sufficient have been cited to carry out the statement already made that the disease is not unusual in early childhood.

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[1] Medical Record, January 11, 1896, p. 46.

[2] Lancet, London, 1891, i. 1194.

The treatment is to-day based upon such certainty in pathological knowledge that the literature is no longer burdened with a prolix list of remedies and remedial measures. It is simply necessary to secure absolute cleanliness. My plan in children is to have them thoroughly well bathed, and then in older girls the vulva and vagina are cleaned much as they would be preparatory to a plastic operation. A douche of permanganate of potassium (1 to 2000) or bichloride of mercury (1 to 4000 or 5000 or even 10,000) is to be used once or twice a day, depending upon the severity of the attack. No special treatment of the urethra is required. In very young children it is best to use a soft catheter for the douche. During the douche the vaginal orifice is to be occasionally closed in order that all the interstices of the vagina may receive a free application of the fluid. If this treatment is thoroughly carried out the duration of the disease will be very materially shortened. My cases rarely persist over twenty days, in many but fifteen. Agramonte,[7] in a large series treated upon a somewhat similar plan, produced a cure in an average of twelve and three-fifteenths days. The constitutional dyscrasia may require general treatment, which is of course to be specifically directed to the existing condition, as, for example, cod-liver oil, the phosphates, and the phosphites will hasten recovery in a rachitic child.

The Bacteriology of the Vagina.—The normal and abnormal microbic flora of the vagina in children are practically identical with those in the adult. My own studies are in accord with those of Vahle,[8] who finds that for twelve hours after birth the vulva and vagina are sterile. From this

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[1] Jahrb. für Kinderheilkunde, 1896, xlii. 1 and 4; Ped., vol. iii. No. 6, 1897.

[2] Archiv f. Kinderheilkunde, xx. 3 and 4, 238; Ped., vol. ii. No. 7, 1896.

[3] Gaz. d. hôp., Paris, 1892, lxv. 37.

[4] British Medical Journal, November 18, 1893.

[5] Sajous's Annual, 1895.

[6] Jour. de Clin. et de Thér. inf., September 3, 1896.

[7] Medical Record, New York, January 11, 1896.

[8] Zeitschrift für Geburtshülfe und Gynäkologie, Bd. xxxii. Heft 3, 5; New York Medical Record, November 16, 1895.

germ is found in normal vaginal secretions, except a thrush fungus
capable of producing suppuration to a very limited extent in the
animal. In the yellowish or greenish, thick vaginal secretion, s
large quantities, mixed with mucus, alkaline or faintly acid, are to
the greatest number and variety of cocci and bacilli. Döderlein
scribed an anaërobic vaginal bacillus, without motion, which is fou
vaginæ, and produces an acid medium by forming lactic acid.
fungus is frequently seen in association with them; this Döderlein
to be identical with *monilia candida Bonorden*, or thrush fungus.

The vaginal bacillus of Döderlein has the power within certa
to destroy staphylococci; it is antagonistic to them under all co
This has been demonstrated by infecting the vagina of a vir
staphylococcus cultures, and within four days the staphylococci ha
appeared, and no bacteria except the vaginal bacillus was foun
vagina. This is the experience of several observers, notably Me
concludes that the germicidal power of the vaginal secretions is
to the antagonism of the normal microbic flora of the vagina and t
genic micro-organism which may be deposited there by accident;
to the product of the life-process of the vaginal bacilli; thir
acidity of the secretions; fourth, to the germicidal powers of the a
elements of the vagina; fifth, to the leucocytosis which is prov
chemotactic action either of the vaginal discharges or of the infectin
organisms invading the vagina; sixth, to the phagocytosis followi
cytosis; seventh, to the absence of free oxygen in the vagina.

Walthard has further shown that the genital canal is practicall
into two parts, one infected and the other sterile. The infected regio
vestibule, the vagina, and the lower portion of the cervical canal; t
the upper portion of the cervical canal, the uterine cavity, and t
The lower segment of the infected area contains leucocytes and bac
middle section only leucocytes, and the upper neither leucocytes nor

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[1] Monatsschrift für Geburtshülfe und Gynäkologie, Bd. ii. S. 381; Universi
Magazine, October, 1896.

[2] Das Scherdenseskret und seine Bedeutung für das Puerperal Fieber, Al
lein, Leipzig, 1892.

[3] Deutsche Medicinische Wochenschrift, October 24, 1894; Hurst, Universi
Magazine, vol. ix. No. 1, 1896.

The vaginal streptococci become infectious only when the resistance of the tissue with which they are in contact is diminished. Stroganoff considers the active germicidal powers of the cervical mucus as the cause of the sterility of the upper cervical canal and the uterine cavity. Micro-organisms are rarely seen below the external os; the cervix may contain a few, but their number is small.

THE UTERUS.

Many cases that have been reported in the living as examples of absent uterus have been incompletely studied. The uterus is rarely totally absent; it may, it is true, be represented by but a thin ribbon, like a band of muscular tissue, which may be discovered only at autopsy.

The more recent reports of absence of the uterus are those of Remfry,[1] in which the breasts were also absent; Fairfield,[2] in which no uterus or ovaries could be located; Gairabetoff,[3] complete absence of the uterus, and Jacobi's report of a case of absent uterus, with consideration of the significance of hermaphroditism;[4] Clapham's[5] report of an illustration of congenital absence of the uterus, and Temberg's[6] contribution to the knowledge of congenital deformity of the sexual organs. Phillips,[7] Dorland,[8] Spiegelberg,[9] Spruill,[10] and Wathen[11] have all recently contributed to our knowledge of congenital absence of the uterus. When the uterus is absent or merely represented by a connective-tissue or muscular band, the other sexual organs are also incomplete, the vagina is rudimentary, and the broad ligaments are simply thin transverse bands. The ovaries are ill developed, but they may contain Graafian follicles. The Fallopian tubes are usually solid, except at the fimbriated extremity, where the lumen may be patulous for a short distance. The pelvis is of the feminine type, but as the child grows the pubic hair is apt to remain absent. Sexual desire does not arise. Amenorrhœa, dilatation of the urethra, and distention of the rudimentary vagina are the usual concomitants. The literature contains many examples of this anomalous condition. The various uterine anomalies are fully treated in vol. iii. p. 743 of this Cyclopædia.

Congenital Prolapse of the Uterus.—Prolapse of the uterus in the new-born viable child is a rare condition. Ballantyne and Thomson,[12] to whose paper I am indebted for much of the matter that now appears, have recently placed on record the seventh and eighth known examples of this anomaly.

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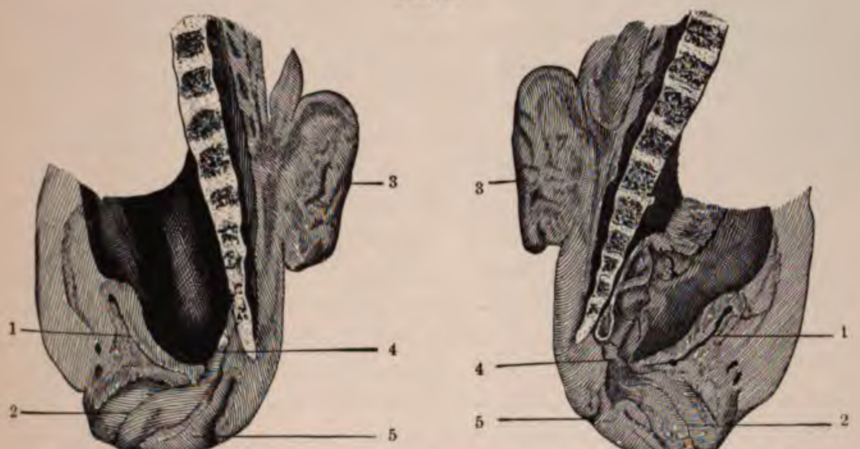
[1] Transactions of the Obstetrical Society of London (1895), 1896, xxxvii. 12.
[2] Indian Medical Journal, 1894–95, xiii. 52.
[3] Med. Obozr., Mosk., 1895, xliv. 53.
[4] American Journal of Obstetrics, 1895, xxxii.
[5] Quarterly Medical Journal, Sheffield, 1895–96, iv.
[6] Finska lak. sallsk. handl., Helsingfors, 1896, xxxviii. 805–826.
[7] King's College Hospital Reports, 1894–95, London, 1896, ii. 240–242.
[8] Philadelphia Polyclinic, 1895.
[9] Archiv f. Path. Anat., Berlin, 1895, cxliii.
[10] North Carolina Medical Journal, 1896, xxxviii. 359.
[11] Louisville Medical Monthly, 1896–97, iii. 370.
[12] American Journal of Obstetrics, 1897, ii. 35.

The first case, a full-term child, was six days old when it came under observation. The child was healthy, with the exception of a spina bifida and a double club-foot. The urinary excretion was normal and the bowels were regular. Two days after birth the spina bifida burst, and the following day the prolapse of the uterus was first observed by the medical attendant. It remained constantly down and caused continued pain and straining. The child now presented the following conditions. First, in the lumbar region was a large spina bifida which had burst; its base measured about one and a half inches in diameter. Second, protruding for about three-quarters of an inch from the vulva was a red mass closely resembling prolapsed bowel; this was, however, the prolapsed cervix uteri and vaginal wall. A sound entered the uterus one and three-quarters inches. It could be passed into the vagina at the side of the prolapse about one inch all round. The prolapse could be reduced, but readily reappeared. Third, the anus projected abnormally; its orifice was markedly dilated. Fourth, extreme bilateral talipes varus. The right patella was absent.

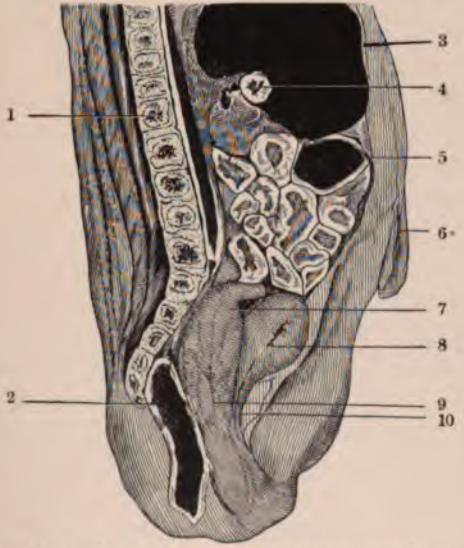
The child died seven days after birth. The post-mortem showed that the heart, lungs, liver, spleen, kidneys, stomach, and bowels were normal. Fig. 1 represents the lower end of the trunk as found at post-mortem. Fig. 2 is for comparison. The authors thus describe the condition found: "A reference to the two illustrations makes it at once evident that the uterus is really prolapsed, for in the one instance the fundus uteri lies at the level of the coccyx (in the plane of the pelvic outlet), whilst in the other it is situated 0.5 centimetre above the pelvic *brim* (its normal height at this time of life). The cervix uteri (Fig. 1) distends the vulva and protrudes slightly from it, but the degree of protrusion is much less than during life; normally (Fig. 2) the cervix is at the level of the third or fourth sacral vertebra. In the prolapse specimen the cervix and the lower part of the corpus uteri have an upward and backward direction,—that, in fact, of the normal vaginal axis,—but the upper part of the body lies nearly vertically in the pelvis. The uterine axis contrasts markedly with the normal, as shown in Fig. 2. The bladder, whose cavity has a Y-shape, is situated lower in the pelvis than is usual, and there is also a certain degree of prolapse of the vaginal walls. The rest of the pelvic cavity is occupied by the rectum and intestinal coils (not represented in the illustration). The sacrum shows no indication of a promontory, and the lower part of the spinal column is perfectly straight, except for a slight bending *backward* of the tip of the coccyx. This contrasts markedly with the normal disposition of the parts represented in Fig. 2, in which there is a lumbo-sacral and a sacro-coccygeal curve, and the tip of the coccyx is turned *forward*. The empty condition of the rectum in the prolapse case is noteworthy and contrasts with its distended state in the normal infant. The defect in the posterior wall of the spinal canal affects the last lumbar and the first two or

FIG. 1.



Vertical mesial section of pelvis of infant with congenital prolapsus uteri. Right and left sides shown.—1, bladder; 2, cervix uteri; 3, spina bifida; 4, fundus of uterus; 5, anus (Ballantyne and Thomson.)

FIG. 2.



Vertical mesial section of infant's pelvis, showing normal relations of viscera. Left side shown.—1, lumbar vertebræ; 2, rectum; 3, liver; 4, pylorus; 5, colon; 6, umbilical cord; 7, uterus; 8, bladder; 9, vagina; 10, symphysis pubis. (Ballantyne and Thomson.)

three sacral vertebræ, and the cauda equina is seen spread out over the inner surface of the spina bifida sac. A distinct perineal body of a triangular shape exists, the vaginal rugæ are well marked, and the distended vulvar orifice shows an unruptured annular hymen. Dissection of the pelvic contents revealed the ovaries and Fallopian tubes lying slightly above the level of the fundus uteri at the sides of the pelvic cavity. The broad and round ligaments were greatly stretched and thinned. The connective tissue in the pelvis seemed to be smaller in amount than normal, but the infant herself was not at all plump. The urethra was patent. Careful measurements proved conclusively that in the infant with prolapsus uteri the diameters of the false pelvis were below the normal, whilst those of the true pelvis, both at the brim and outlet, were distinctly above the average. Save for a certain but not great degree of cervical enlargement, the diameters of the uterus did not differ greatly from those in normal infants."

Congenital prolapse of the uterus is to be distinguished from congenital hypertrophic elongation of the cervix by the fact that it is apt to be accompanied by a certain amount of cervical hypertrophy. The prolapse is practically always associated with lumbo-sacral spina bifida and rectal ectropion, often with clubbed feet, and sometimes with hydrocephalus and hypertrichosis. The prolapse may be of any degree; the anomaly is usually without marked symptoms. Some of the cases have performed the functions of the rectum and the bladder in a normal manner. In addition to the frequent association of uterine prolapse with spina bifida, other causal factors have been observed, as abnormally large size of the pelvis, an enlargement of the uterine body or cervix or of the entire organ, and increased abdominal pressure.

Stepkowski[1] saw a woman aged twenty-five years with complete prolapse of the uterus and vagina, which was caused by excessive vomiting when she was thirteen years old. It had persisted for twelve years, and Karczewski[2] has seen a girl, aged thirteen, with complete procedentia developing gradually from carrying heavy burdens. The rectum prolapsed a few days before the child came to hospital.

Uterine displacement in the new-born and in young girls has recently received some attention in the literature. Spencer[3] presents a paper on retroflexion of the uterus in a new-born child, and Berti[4] one upon congenital uterine misplacement in the new-born. Penrose[5] has observed an instance of congenital absence of the uterine cervix.

Congenital Split of the Cervix Uteri with Erosion.—I have seen two instances of this condition,—one in a young, ill-developed child, and the other in a fairly well-developed girl just reaching womanhood. The disease remained unclassified in my mind until I saw the papers of Leopold,[1] Fischel,[2] and Penrose.[3]

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[1] Gaz. lek in Przegl. Chir.; La Gynic., No. I., 1897; Year-Book, 1898, Gould.

[2] Ibid.

[3] Transactions of the Obstetrical Society, London, 1892–93, xxxiv. 25–28.

[4] Spostamenti uterini congeniti in neonate, Bull. d. Sc. Med. di Bologna.

[5] University Medical Magazine, Philadelphia, 1893–94, vi. 185.

Jefferson[4] has recently reported a case in a girl approaching seventeen years of age, an undoubted virgin. The uterus had a bilateral cleft, the lips opening fully an inch, and a very extensive erosion. Fischel describes very carefully, both microscopically and macroscopically, the condition of congenital erosion and split of the cervix uteri. The following abstract is from the translation of Fischel's paper that accompanies Penrose's communication: "During the course of a series of investigations on the cervices of infants who had been still-born or who had died soon after birth, the author's attention was called to a condition which, so far as he knew, had never been described by any anatomist or gynæcologist that had undertaken the study of this part of the anatomy,—namely, the occurrence of erosions. A short time before he had discovered by accident in the Transactions of the Gesellschaft für Geburtshülfe, in Leipzig, July 15, 1872, a communication by Leopold stating that he had seen such an erosion of the cervix, and declaring it to be an anomaly in the new-born child heretofore unrecognized. In the debate that followed Ahlfeld stated that circulatory congestion in the uterus and apparently also erosions of the mucous membrane were a frequent occurrence in asphyxiated infants."[5] From this short notice Fischel judges that neither Leopold nor Ahlfeld had ever subjected these erosions to a microscopic examination. He therefore publishes the investigations he made and also the conclusions he reached as a result of them.

Of twenty-eight cervices examined, he found ten erosions of varying character and size; of these ten cervices, four were from fœtuses still-born at term, two from infants a few days old, one from an infant fourteen days old, and three from infants three, four, and five weeks old respectively. Of the eighteen cases in which no erosion of any kind was found, five were still-born at term, one was a few days old, one nine weeks, one thirteen weeks, two were eighteen months, and one was two and one-half years old. The others came from fœtuses in the sixth, seventh, eighth, and ninth months of intra-uterine life.

From these cases he found that the assertion of Ahlfeld had no value so far as cause and effect between asphyxia and erosion were concerned, since, of the five children born asphyxiated, none showed any erosion, while some did that died at varying periods after birth. The appearance which the erosion on the infantile cervix presents varies both in the extent and in the form of the change. In most cases the external os forms a rather narrow transverse opening surrounded by a reddened, velvety area from three to four millimetres wide. In other cases the erosion extends on to the lateral surfaces of the cervix higher up than on the anterior and posterior aspects. In other cases still—and these bore the nearest resemblance to erosions in the adult—the change was limited to the under surfaces of the lips of the cervix as far as they came in contact with the posterior vaginal wall. He points out that with the naked eye two forms of erosion may be distinguished: one with a satiny appearance, another of a rougher, more granular character, recognized as papillary. Microscopically the papillæ, instead of being protected by an even covering of squamous epithelium, stand out separately in a more or less irregular arrangement. Covering the uneven surface thus formed is a single layer of cylindrical cells. On the ends of the papillæ the cylindrical form is replaced by a flatter, more cubical one, but everywhere the layer is single. There is no transition form between squamous and cylindrical epithelium, but the hue of demarcation is always sharply defined, so that, for example, one side of a papilla may be lined with stratified squamous, the other with simple cylindrical epithelium. In many places compound glands, acinous in character, open into the depressions between the papillæ, and into these glands the simple columnar epithelium dips down. The mucosa itself is composed, as usual, of a connective tissue rich in nuclei.

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[1] Trans. Gesellschaft für Geburtshülfe, Leipzig, July 15, 1872.

[2] Archiv für Gynäkologie, 1880, Bd. xvi. S. 192.

[3] American Journal of the Medical Sciences, May, 1896.

[4] Medical Sentinel, vol. iv. No. 12, p. 552, 1896.

[5] Archiv für Gynäkologie, Bd. v. S. 162.

He concludes that in almost thirty-six per cent. of new-born infants the vaginal surface of the cervix from the external os towards the vaginal fornices is covered more or less extensively with a mucous membrane which, from the form of its epithelium, from its papillary character, and from its possession of mucous glands and crypts, must be regarded as a direct continuation of the cervical mucous membrane.

The dividing line between the epithelium of the vagina and that of the uterus does not lie—as we were led to believe by all previous investigators, even the latest, Klotz—at the outermost end of the cervical canal, but may be situated even on the external vaginal surface of the cervix, more or less high towards the vaginal fornices. For this condition he adopts the name congenital histological ectropium.

This dividing line between the two forms of epithelium lay, in the greater number of cases, within the cervical canal, from two to three millimetres above the external os, and coincides with the external os in only a minority of cases. He accounts for the formation of this ectropium by the theory that, after the formation of the uterus and vagina by the union of the lower segments of the ducts of Müller, which are lined by a simple columnar epithelium, a change begins at either end in the mucous membrane. In the uterus this change consists in the development of a glandular system, while in the vagina, beginning at the uro-genital sinus, the columnar epithelium gradually changes into the squamous variety. At the termination of intra-uterine life this change should have reached the external os, but owing to the varying rapidity with which it progresses in different individuals the point which it reaches varies, being sometimes

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In regard to the connection between the histological ectropium and erosions and other pathological conditions, he says: "It must be acknowledged as possible that erosions occur in adults when the individual has received a predisposition to it during the period of her development. It is furthermore to be conceded that this congenital histological ectropium of the cervix may be covered over by squamous epithelium, but still retain under its covering the glands and other attributes of a cervical mucous membrane. If, now, through some inflammatory process, the tissues become congested and infiltrated with round cells, the acquired superficial squamous epithelium will be shed and the original columnar epithelium be brought to light, presenting the complete picture of a true œdematous or papillary erosion." He admits, as he has stated in his paper on erosions, that the formation of the erosion may occur on a spot normally covered with squamous epithelium, and it becomes more and more evident to him that of women whose cervices are intact, those only will have erosions who are predisposed to them by this congenital condition.

He exhibits a photograph of the cervix of a new-born infant which presents an inferior degree of this condition. The separation of the lips does not extend all the way to the vaginal junction, but concerns only the lower two-fifths of the lateral corners. Nevertheless, the two lips, deprived of their commissures, gape open, the crest of one being nine millimetres from that of the other, exposing the surface of both lips for a distance of from five to six millimetres. This case shows that a peripheral notching of the cervix is not always a sign of a previous labor.

The condition is graphically represented in the accompanying plate. At first glance it is extraordinarily like that observed in a bilateral laceration of the cervix with ectropion, but the congenital defect is without the deep cicatricial scar or plug at the angle. The whole cervix is soft and boggy, without much interstitial change. There is usually an absence of the little retention-cysts so common in old lacerations.

The only appropriate treatment in older children or young women is amputation of the cervix, which in the few recorded instances has been entirely successful and curative.

ENDOMETRITIS.

It is not my purpose to consider the subject in all its aspects; it is so fully treated in the various standard works on gynæcology that repetition would be uninteresting. One form of this disease merits consideration in this work,—the hemorrhagic endometritis of young girls and young virgins.

Menorrhagia in young girls is often observed; it is not at all rare, and its pathology is well understood. Its treatment, however, is sadly neglected and is apt to consist of the exhibition of medicinal agents, and is, therefore, not at all curative. This is to a large extent due to the inherent modesty of the child and the physician's hesitancy in insisting upon the necessity of an ocular and digital examination to determine the proper local treatment.

Curettement is the only rational and radical measure for the relief of these cases. I see many cases every year, and the relief afforded by the curette is speedy and apt to be permanent. Blanc[1] cites the following cases, and my experience is entirely in accord with him.

A girl aged sixteen, with menorrhagia of eighteen months' standing, beginning with her second menstrual period. So much blood was lost that death was feared. Curettement was followed by relief for six months, when a second operation was necessary. There was no return of the hemorrhages after a lapse of six years. Menorrhagia of a year's standing in a girl of fifteen years of age, with pelvic pains and marked anæmia. The curette removed a quantity of pale fungosities; the patient was cured. Hemorrhages recurring at intervals for six months in a child of fourteen. Cure by curettement. Obstinate hemorrhage, which had continued for six months in spite of the administration of iron, ergot, hydrastis, and hot douches, in a patient of fifteen years. An operation was refused, and the patient eventually succumbed.

MALIGNANT DISEASE.

Carcinoma and sarcoma of the uterus in the young is still a rare condition,—not, however, as rare, I think, as it was when the original volumes of this Cyclopædia were written. The general advance in gynæcology and operative technique has caused a more careful study of the sexual organs of the young, and I am able to add several cases to those already reported. Laidley[2] has confirmed by post-mortem and microscopic studies the diagnosis of carcinoma in a child aged two and a half years. Von König's[3] case of uterine cancer occurred in a girl thirteen years old, and Johannessen[4] observed a pelvic sarcoma in an eleven-year-old girl. Goodell[5] operated upon malignant disease of the pelvis in a girl under fifteen years of age, and Coppée[6] records an observation of an enormous encephaloid cancer of rapid growth in the genital organs of a girl of eighteen years. Tschop[7] reports a carcinoma of the uterus developed at the early age of nineteen years, the uterus being removed per vaginam, followed by uninterrupted recovery. The vagina and adnexa were free from infiltration. Microscopic examination confirmed the diagnosis. Minot[1] has seen a cancer of the uterus in a patient aged twenty-one years, and Boudin[2] an epithelioma of the neck of the uterus in a woman of twenty-three years. Laver and Wilkinson[3] note a case of sarcoma of the uterus in a woman twenty-one years of age. Hollander[4] has the specimen of a total extirpation of the uterus and vagina for sarcoma in a child aged nine months. The disease had existed since the babe was seven months old. The operation was a success, and the child was discharged cured in a month.

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[1] Loire Médical, December 15, 1896.

[2] St. Louis Courier of Medicine; Sajous's Annual, 1891, F. 34, vol. ii.

[3] Allg. Wien. Med. Zeitung, 1885, xxx. 280.

[4] Jahrb. für Kinder., Leipzig, 1897, N. F., xliv. 114–122.

[5] Medical and Surgical Reporter, 1891, vi. 131–134.

[6] Bull. Soc. de Méd. de Gand, 1864, xxxi. 35–39.

[7] Centralblatt für Gynäkologie, No. 2, 1897.

Smith[5] has contributed a short communication on sarcoma of the uterus previous to puberty. Worrall[6] successfully removed an adenomatous uterus and a double ovarian tumor in a young girl, and Bradford[7] has removed by abdominal section a papillary cystoma of the uterus in a girl aged nine years. The tumor was the size of a fœtal head at full term. One ovary and tube and a large portion of the uterus were removed with the tumor. The patient made an uninterrupted recovery. Microscopic examination proved the growth to be a papillary cystoma, and some anxiety was felt lest it might return, but at the time of the report, three months after operation, there is no sign of its return, and the child is free from the cachexia of malignant neoplasm. A patient, eighteen years old, presented to the Halle clinic a tumor two centimetres in diameter growing from the posterior lip of the cervix. Braetz[8] believed it to be an endothelioma. Kaltenbach removed the uterus per vaginam. The girl died four weeks after leaving the hospital. No autopsy. The growth probably originated in the endothelium of the lymph-spaces.

Little[9] has recorded a carcinoma of the uterus in a girl aged fourteen years.

TUBERCULOSIS.

This is not a rare affection of the uterus in women; it is generally associated with or secondary to tuberculosis of the tubes. It may be secondary to a pulmonary tuberculosis or occur as a part of a general bacillary invasion. Whitridge Williams[10] has seen some cases in which it represented the only focus of tuberculosis in the body. As yet sufficient cases have not been studied in children to permit us to offer very definite statements regarding the special peculiarities of the disease in early childhood. Silcock[1] has observed a tuberculosis of the uterus in a child aged five. The cervical canal was obliterated by inflammatory changes, the uterine secretions were unable to escape, and a pyometra was formed. Chaffey[2] has also seen a case in a child aged four in which the uterus and tubes were tuberculous, the child dying of a general tuberculosis. George Carpenter,[3] of London, has given us a very interesting study upon tuberculosis of the uterus and adnexa. He finds that specimens of tuberculosis of these organs are preserved in but very few museums. In Guy's Hospital Museum there are but two, and these are very old specimens. At autopsy he has observed tubercles in the uterus of a girl aged sixteen months, and further makes the following interesting statement: "In a girl of seven years who attended my out-patients some six or seven years ago, and who looked exceedingly ill, I found an enlargement of the uterus, the right tube being the size of a lead-pencil; there was a lump, possibly tubercular, the size of a pigeon's egg, just above the top of the sacrum. Unfortunately, I soon lost sight of her, but had I possessed my present knowledge at that time, I do not think I should have rested content with the mere record of the fact and the exhibition of tonics. A girl of seven years came to the Evelina Hospital suffering from tubercular peritonitis of the ascitic variety. I found the ovaries and uterus normal, but from the top of the latter a rounded tumor arose the thickness of the index finger, of about three inches in length, which passing upward in the abdominal cavity, was there lost. Two years after this the tumor had shrunk to the size of a lead-pencil; she was in good health, and remains so to this day, and it is ten years since her illness commenced." Peron[4] adds to our knowledge in a contribution to the study of tuberculosis of the tubes and uterus in a girl twelve years of age.

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[1] Boston Medical and Surgical Journal, 1861, lxiv. 211.

[2] Lyon Médical, 1895, lxxx. 490–497.

[3] Quarterly Medical Journal, Sheffield, 1893–94, ii. 245–247, I. pl.

[4] Deutsche Medicinische Wochenschrift, 1896, xxii. 16.

[5] American Journal of Obstetrics, New York. 1893, xxvii. 577–579.

[6] Australian Medical Gazette, Sydney, 1893, xii. 308.

[7] Archives of Pediatrics, New York, 1892, ix. 508–511.

[8] Arch. f. Gynak., Bd. ii., 1896.

[9] New Orleans Medical and Surgical Journal, December, 1896.

[10] Clinical Gynæcology, Keating and Coe, 1895.

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[1] Transactions of the Pathological Society of London, 1885, vol. xxxvi. 303.

[2] Transactions of the Pathological Society of London, 1885, vol. xxxvi.

[3] Pediatrics, i., June 1, 1896.

[4] Rev. Mens. d. Mal. de l'Enf., Paris, 1894, xii. 372–379.

DISEASES OF THE OVARIES AND FALLOPIAN TUBES.[1]

BY HOWARD A. KELLY, M.D.

DISEASES of the internal genital organs, the uterus, uterine tubes, and ovaries, for the most part arise during the period of sexual activity, and the cases occurring later in life are as a rule due to the further development of diseases which have already started in middle life. While this is true in general, a careful search of the literature shows that pelvic affections during childhood are of far more frequent occurrence than is usually supposed. In some instances conditions first observed after puberty are actually due to diseases existing earlier in childhood, but not attracting attention until the organs are called into active use.

The literature relating to diseases of the female genital organs in children is for the most part confined to reports of individual cases. Professor C. Hennig's article, published in Gerhardt's Handbuch der Kinderkrankheiten in 1876, is still the most important treatise on the subject in its entirety, notwithstanding the comparatively few cases at that time placed on record. Bland Sutton has described the ovarian tumors found in infancy and childhood, especially referring to the group which he terms the "oöphoromata," as they seem peculiar to the connective tissue of the oöphoron. He has collected one hundred cases of ovariotomies performed on children under sixteen years of age. Brouardel, Talamon, Maas, and others have described cases of tuberculosis of the tubes and ovaries in children. The more recent literature also contains numerous references to inflammatory diseases of these organs in childhood, due to the extension upward of an infantile vulvo-vaginitis.

On account of the extreme importance from both a diagnostic and an operative stand-point, it would be well for future writers when reporting cases of pelvic disease in children to record carefully every detail in the clinical history, the operation, and also, what is much neglected, the later history of the patient.

Almost all forms of diseases of the tubes and ovaries found in adults have been observed during childhood, certain pathological conditions being relatively more frequent at one period than at another; thus, in children under three years principally ovarian tumors have been found, and these either dermoids or sarcomata. A. Doran[2] has described a case of double ovarian tumor in a seven months' fœtus which survived its birth only a few minutes. The tumors resembled small cystic kidneys, and the solid portion consisted of small round cells embedded in a homogeneous matrix and contained a few follicles. Doran considered the tumors to be an hypertrophy of the entire embryonic tissue of the oöphoron.

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[1] Dr. Kelly desires to acknowledge the valuable assistance of Dr. E. Hurdon in the preparation of this article.

[2] Tr. Path. Soc. London, 1888–89, xl. p. 200.

Cystic ovaries are frequently observed in infants, and have been described by de Sinéty[1] and others. According to Kissel's[2] investigations these cases of cystic follicles are rare in children after the first year, and they should not be confounded with the adeno-cystomata which make up a large proportion of the ovarian tumors of later childhood and adult life.

While ovarian tumors may be found in the earliest years of childhood, they become more frequent in girls approaching puberty. Half of the tumors at this period belong to the cystomata. A few carcinomata have been described in older children, and inflammatory disease and tuberculosis also occur.

Diagnosis.—The diagnosis of diseases of the tubes and ovaries in children may usually be made without difficulty. The diagnostic signs are the same as those in adults, with the notable exception of the differences in the topographical relations. Owing to the relative smallness of the abdominal cavity, a tumor will begin to encroach upon the vital space in a child when it has reached a size which in the adult would scarcely be noticeable. In this way an ovarian tumor the size of a child's head, lying in the abdomen, stands so high and occupies so much space that it may at first sight resemble a tumor either of the liver or of the kidney. Owing also to the smallness of the pelvic space and the straightness of the canal, the tumor is extruded earlier into the abdominal cavity than is usual in the adult.

When a tumor has been found in the abdomen which from its form, consistency, mobility, and the corona of resonance surrounding it apparently has pelvic connections, the next important step in the investigation is to make a thorough pelvic and bimanual exploration, to determine, if possible, its origin from the uterus, tube, or ovary. This part of the examination is made much easier by the fact that the examining finger is relatively longer in proportion to the child's pelvis; in other words, a finger which in the adult with difficulty reaches up through the inferior strait as far as the promontory of the sacrum, in the child may be carried well up into the abdomen. The proportion between the length of the finger and the pelvic cavity varies, of course, with the age of the child, and the advantage grows less as the age increases.

To make a satisfactory examination it is necessary to empty the bladder and the bowels thoroughly and to put the little patient under an anæsthetic. I prefer, in children, to use chloroform, as they often require but a few whiffs to produce insensibility, and do not appear to be much affected by it afterwards.

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[1] Gazette Médicale de Paris, 1875.

[2] Nouv. Arch. d'Obstét. et de Gyn., 1891.

Complications.—*Suppuration* in ovarian tumors is due to the invasion of pyogenic organisms. These gain access to the tumor through the blood- or lymph-vessels or by direct inoculation. As examples of the last mode of infection, in two cases of cysts in young girls the organisms were introduced by means of the trocar used in evacuating the fluid. The bacillus coli communis, which readily passes through the partially absorbed walls of the intestine where the tumor is adherent, is a frequent cause of suppuration. In other instances an inflamed Fallopian tube attaches itself to the ovarian cyst, with a resulting extension of the inflammatory process.

Rupture of a simple cyst is said to give temporary relief occasionally. When the cyst-contents are thicker or purulent, the peritoneum is irritated, and in consequence a more or less severe localized or general peritonitis results. There are cases where the sudden attack of acute abdominal pain, with distention, fever, vomiting and diarrhœa or constipation, and the absence of any history of pelvic trouble, have indicated an appendicitis, the true condition being unsuspected until abdominal section was performed, or discovered only at autopsy. Beale[1] observed a case in an infant of six weeks who died four days after the onset of symptoms of acute peritonitis. At autopsy purulent fluid was found in the peritoneal cavity and the pelvis was filled with pus, while attached to the right ovary were the remains of "a small ruptured cyst," evidently the cause of the attack.

Torsion of the pedicle occurs in a considerable number of ovarian tumors. Tumors of medium size and free from adhesions are especially inclined to axial rotation, which is impossible where the tumor is adherent or of large dimensions. The torsion may be acute or chronic, and Bland Sutton has pointed out that acute torsion is apt to occur in cases of medium-sized tumors, while small tumors more often rotate gradually.

When the existence of a tumor has been previously recognized, the symptoms of torsion are characteristic; otherwise the sudden attacks of severe abdominal pain, distention, vomiting, and obstinate constipation are apt to be attributed to an acute appendicitis. McBurney[2] and Bull[3] each operated on "young girls" for supposed appendicitis, and on opening the abdomen found a gangrenous ovarian tumor with twisted pedicle. A gradual rotation may occur without marked symptoms, and the tumor has been known to be slowly twisted off from its attachment, becoming parasitic.

The clinical history of axial rotation of an ovarian tumor is well illustrated by a case which I operated on at the Johns Hopkins Hospital. G. L., thirteen years old, seven months previously had been suddenly attacked with violent pain in the abdomen and with vomiting. A second attack occurred four months later, and at this time a soft, fluctuant swelling was noticed in the right ovarian region. For three or four weeks preceding operation the patient was in excellent health and had gained weight. Enucleation was easy, and the patient made an ideal recovery. The tumor, can only vaguely refer it to certain unknown conditions existing during early fœtal life. In many instances two or more members of the same family present similar defects, indicating the important influence of heredity. The failure of the infantile organs to attain the adult type is probably in most cases due to malnutrition, the result of such general diseases as rhachitis, scrofula, or syphilis.

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[1] British Medical Journal, 1891, ii. 1255.

[2] Medical Record, New York, April 3, 1897.

[3] Ibid.

Hernia.—Displacement of the ovary into the canal of Nuck, usually accompanied by the fimbriated end of the tube, is of not uncommon occurrence, and although a congenital defect may not be noticed until cystic changes in the follicles or the congestion of a menstrual period occasion attacks of pain. Ovarian as well as other herniæ are liable to become strangulated. Radical operations for the cure of ovarian hernia with torsion of the pedicle were successfully performed on young infants by Owen[1] and Lockwood.[2] Owen's patient was eleven days old. Two days before operation she was suddenly attacked with vomiting; the next day a hard, tender swelling was noticed in the right labium majus. Her general condition was good. On making an incision, the engorged ovary presented and was excised. The pedicle, which consisted of the broad ligament containing the tube and parovarium, was twisted towards the left as far as the uterine cornu. Lockwood's case was similar, but the exact age is not given. Both infants made excellent recoveries.

It is manifest that all the anomalies of form found in adult life must exist in childhood also, and only remain undiscovered until later on account of the abeyance of all functional activity. The discovery of such malformations is therefore, as a rule, the accident of the post-mortem table, if I may except from this general statement cases of imperforate hymen which mothers seem keen to observe.

OVARIAN TUMORS.

The ovarian tumors occurring in children may be classified as follows:

Cystic tumors	Adeno-cystomata. Unilocular cysts. Dermoid cysts.
Solid tumors	Sarcomata. Carcinomata.

Adeno-cystomata, or multilocular cysts, the classical ovarian tumors, form the most numerous group of ovarian growths occurring in childhood, comprising, with a few monocysts, one-half of all cases recorded. They are in all respects similar to the ovarian cysts found in adults. Probably the largest cyst observed during childhood is the case successfully operated on by W. W. Keen, of Philadelphia. The patient, fifteen years of age, for two years had noticed an increasing abdominal enlargement, accompanied by pain in the left side. She had been tapped twice, eighty-four pounds of fluid being removed. At the time of operation the abdomen was greatly distended, measuring forty-nine centimetres in circumference, and the superficial veins were prominent. Extirpation was effected without difficulty, as there were but few adhesions. The patient made a good recovery. The tumor was a multilocular ovarian cyst weighing one hundred and eleven pounds.

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[1] Lancet, 1895, vol. i. p. 765.

[2] British Medical Journal, 1895, vol. ii. p. 716.

Dermoid Cysts and Teratomata.—These tumors are for the most part of medium or small size, rarely attaining large dimensions. They are sometimes monocystic, at other times multilocular, in some instances the dermoid elements being present in only one loculus, while the remaining portion of the tumor is identical with the ordinary adeno-cystomata.

A strong confirmatory evidence of the origin of dermoids from misplaced embryonic tissue is the frequency with which they are met with in children and the comparatively early period of life at which they are observed in adults. The average age of all the patients operated on at the Johns Hopkins Hospital for the extirpation of dermoid cysts was only twenty-six years. Of the ovarian tumors found in children, one-third, a high percentage, contained dermoid elements. Tumors of this nature are in some instances congenital. In Roemer's patient, operated on when twenty months old, the abdominal distention was noticed at birth, and in the case reported by Neville the tumor was first noticed when the child was but two months old.

Dermoids are of slow growth, and often do not manifest their presence until adult life, the normal function of the ovary remaining undisturbed. This is readily understood from their histological structure, as the majority of these tumors contain developing and mature follicles, especially numerous in the vicinity of the hilum. Many of them also contain endo-, ecto-, and mesoblastic structures. Dandois successfully removed from a child of seven years a large tumor weighing seven and one-half kilogrammes, which contained cysts filled with colloid or sebaceous matter and hair, and a third of its weight consisted of bone. Other cases described as teratomata are reported by Emanuel, Neville, and Doranth.

Dermoids and teratomata are usually considered to be benign tumors, but from the history of Emanuel's case it would appear as if this were not always so.

The patient was fifteen years of age. Laparotomy was performed and a tumor of the left ovary larger than a man's head removed, and isolated pieces of peritoneum containing metastatic nodules excised. The tumor contained tissue-elements of endo-, ecto-, and mesoderm, and its stroma was sarcomatous. She made an excellent recovery from the operation, but five weeks later the abdomen was greatly distended with fluid, and in four months she died from recurrence. At autopsy a tumor of the right ovary the size of two fists was found, and nodules were scattered over the omentum and over the general parietal and visceral peritoneum; a small nodule was also present in the abdominal wall. The stump of the primary tumor was healthy. Microscopically the tumor in the pelvis and the nodules in the omentum and in the abdominal wall presented the same characteristics as the primary growth, while the richly cellular nodules studding the peritoneum consisted of small round-celled sarcomata. Cases have also been described in which a carcinomatous growth was present, apparently originating in the squamous epithelium lining the cyst. Whether the malignant process in these mixed tumors was primarily present or the result of a secondary change is a mooted point.

SOLID TUMORS.

Sarcomata.—The activity of the ovarian stroma in early life suggests an explanation of the relatively frequent occurrence of sarcoma of the ovary in children. Congenital tumors often belong to this group, and frequently their histological elements bear a striking resemblance to embryonic ovarian stroma. In their histologic structure, sarcomata occurring in infancy and childhood comprise several varieties; by far the greatest number, however, consist of small round cells. A few belong to the spindle-celled variety; one was described as a lymphangioma, another as an endothelioma, and Gage describes his case as a fibro-sarcoma. In a few cases (three out of twenty-seven) both ovaries were involved. Recurrence is frequent; several children, however, were in good health some years after operation. An interesting case is cited by Bode. A large tumor springing from the right ovary, filling the entire abdomen and growing under the sheath of the abdominal muscles, was successfully removed from a girl of thirteen. Recovery was rapid, and four years later she was in perfect health. The tumor was a round-celled sarcoma.

A similar case is reported by Gage. He operated on a girl fifteen and a half years of age, extirpating a tumor which filled the right inguinal region and was everywhere adherent. She had menstruated regularly since fourteen years old. During the previous winter there had been considerable distention, and one month before operation she first noticed a hard lump in the lower abdomen. The tumor weighed two pounds and was a small round-celled sarcoma. Five years later the patient was in excellent health and married.

A number of years ago I operated on a child twelve years old for an ovarian tumor. The abdomen had begun to swell a few weeks before, and she had been tapped several times, withdrawing dark, bloody fluid, which, however, rapidly reaccumulated. At the operation I removed a sarcoma of the left ovary the size of a man's head. The patient made a perfect recovery, but succumbed two years later to an attack of dysentery with doubtful symptoms of a recurrence.

Carcinoma.—There are on record six cases of carcinoma of the ovary occurring in children, a large number considering the rarity of carcinomata in general in early life. (See list appended.) Carcinoma occurs somewhat later than sarcoma. Redner (reported by Leopold) operated on a child of nine years for an ovarian tumor which proved to be a carcinoma. Extirpation was without difficulty, as there were no adhesions, and the child promptly recovered from the operation. One year later, however, she died from recurrence. The same writer also refers to three other cases of ovarian carcinoma in girls aged eleven, thirteen, and eighteen years. These were all operated on successfully. One patient was well four years later, another died from recurrence in three months, and of the third no further history is recorded. Leopold's patient did not come to operation. The girl, aged fourteen years, gave a history of rapidly growing abdominal tumor, associated with obstinate constipation and increasing cachexia. She died nine months after the appearance of the earliest symptoms.

Clinical History.—The clinical picture of ovarian tumors in children presents in a general way many of the symptoms found arising from the same affections in adults. Certain conditions present in childhood, however, modify the symptomatology in various important particulars. The almost constant early ascent of the tumor into the abdomen lessens the tendency to disturbance of the pelvic organs, and in only a few instances are signs of pressure on either the bladder or the rectum at all marked. On the other hand, a tumor which in the adult would be unnoticed, in a child is relatively large; thus interference with respiration and disordered digestion are more frequently present. Further, the slight physical strength, the lesser vitality, and the more delicately balanced organism of extreme youth are less fitted to withstand the effects of the tumor, which, therefore, is apt earlier to reach a fatal termination. The malignant tumors run an especially rapid course, producing such grave constitutional symptoms that unless operative interference is employed early it is useless, death occurring in from two to nine months after the onset of the first symptoms.

R. Brown[1] observed a case of rapidly fatal malignant tumor in an infant of nine months. She had become a little thinner, attributed by the mother to teething, but otherwise appeared well. Suddenly she became fretful, cried out as if in acute pain, and occasionally vomited her food. The abdomen was greatly distended and was tympanitic, with the exception of the hypogastrium, where percussion elicited a dull sound. Urine was passed freely. The child grew steadily worse, and died on the third day after the onset of the acute symptoms. At autopsy the abdomen was found to contain ascitic fluid; the intestines were distended with flatus and pushed up by a large tumor occupying the pelvis. There was no peritonitis. The tumor, which was described as an encephaloid, was of the form and consistence of the brain of an infant. Under the microscope there were "numerous oval and elongated cells, all of which were nucleated." This case is remarkable on account of the absence of any symptoms which would indicate the presence of a malignant growth. Although malignant tumors are usually the most rapidly fatal, in a few instances ovarian cysts have assumed such rapid growth that the strength of the child has been soon exhausted. A girl twelve years of age is reported by Davidson[2] to have died from a large ovarian cyst two months after its presence was first noticed. The cyst was adherent to the omentum and intestines. The solid portion weighed twenty-one pounds, and it contained two gallons of dark, syrupy fluid.

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[1] Lancet, 1858.

[2] Medical News, Philadelphia, vol. lxi.

Usually the child at first seems somewhat listless and less active while at play, sometimes complaining of abdominal pain. After a short time abdominal enlargement is observed, rapidly increasing, and accompanied by constipation, failing appetite, and gradual loss of weight and strength. Where complications exist, such as torsion of the pedicle, rupture or suppuration of the cyst, or a concomitant peritonitis, there are added the characteristic symptoms produced by these processes; in general, severe paroxysms of pain, high fever, nausea and vomiting, and constipation sometimes alternating with diarrhœa. In several cases of rapidly growing ovarian tumor the general health remained excellent. In a few instances it has been noticed that the breasts were swollen and tender and the external genitalia showed an unusual degree of development. Menstrual disturbances do not necessarily accompany ovarian tumors, and when irregularities are present they are in no way distinctive. In some instances, even where there were tumors of both ovaries, menstruation was normal; in others there was suppression of menstruation; and in still others the catamenia appeared in the eleventh or twelfth year. Prochownick[1] states that, of eight cases of precocious menstruation, four were associated with ovarian tumors. In two children, aged four[2] and seven[3] years, where a periodic bloody vaginal flow was referred to early menstruation, the tumors were sarcomata, involving one ovary.

Malignant tumors, besides producing more marked and early cachexia, are also more apt to cause ascites, an excessive accumulation of fluid increasing the distress of the patient. Furthermore, malignant tumors are more often associated with disturbances of micturition and defecation. Leopold[4] describes two cases—one a lymphangio-cystoma of the ovary in a girl eight years of age, the other a carcinoma in a girl of fourteen—which, on account of the sudden swelling of the abdomen, associated with obstinate constipation, were at first diagnosed as fecal tumors.

Routier operated on a child nine years of age for suspected foreign body in the bladder. There was a history of acute retention of the urine with abdominal distention. Catheterization resulted in the disappearance of the vesical tumor, which, however, returned the following day. Suspecting the presence of a foreign body, Routier performed a cystotomy, but found that the symptoms had been produced by a dermoid cyst of the ovary, which was impacted in the pelvis but free from adhesion. Two other cases presented marked disturbance of the urinary track,—one albuminuria and hæmaturia, the other acute retention with hæmaturia. The formation of adhesions between the surface of the tumor and the neighboring viscera is also apt to involve the bowel or ureters, producing symptoms similar to those due to direct pressure.

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[1] Archiv für Gynaekologie, Bd. xvii. S. 330.

[2] Byford. (See list.)

[3] Croom. (See list.)

[4] Archiv für Gynaekologie, Bd. vi. S. 202.

Complications.—*Suppuration* in ovarian tumors is due to the invasion of pyogenic organisms. These gain access to the tumor through the blood- or lymph-vessels or by direct inoculation. As examples of the last mode of infection, in two cases of cysts in young girls the organisms were introduced by means of the trocar used in evacuating the fluid. The bacillus coli communis, which readily passes through the partially absorbed walls of the intestine where the tumor is adherent, is a frequent cause of suppuration. In other instances an inflamed Fallopian tube attaches itself to the ovarian cyst, with a resulting extension of the inflammatory process.

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Torsion of the pedicle occurs in a considerable number of ovarian tumors. Tumors of medium size and free from adhesions are especially inclined to axial rotation, which is impossible where the tumor is adherent or of large dimensions. The torsion may be acute or chronic, and Bland Sutton has pointed out that acute torsion is apt to occur in cases of medium-sized tumors, while small tumors more often rotate gradually.

When the existence of a tumor has been previously recognized, the symptoms of torsion are characteristic; otherwise the sudden attacks of severe abdominal pain, distention, vomiting, and obstinate constipation are apt to be attributed to an acute appendicitis. McBurney[2] and Bull[3] each operated on "young girls" for supposed appendicitis, and on opening the abdomen found a gangrenous ovarian tumor with twisted pedicle. A gradual rotation may occur without marked symptoms, and the tumor has been known to be slowly twisted off from its attachment, becoming parasitic.

The clinical history of axial rotation of an ovarian tumor is well illustrated by a case which I operated on at the Johns Hopkins Hospital. G. L., thirteen years old, seven months previously had been suddenly attacked with violent pain in the abdomen and with vomiting. A second attack occurred four months later, and at this time a soft, fluctuant swelling was noticed in the right ovarian region. For three or four weeks preceding operation the patient was in excellent health and had gained weight. Enucleation was easy, and the patient made an ideal recovery. The tumor, which was a dermoid cyst the size of a four months' pregnant uterus, presented a double twist of the pedicle, but was free from adhesions.

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[1] British Medical Journal, 1891, ii 1255.

[2] Medical Record, New York, April 3, 1897.

[3] Ibid.

In the differentiation of rotation from rupture of the tumor it is noticed that in the former the tumor becomes firmer, more prominent, and sharply defined; when rupture has occurred, there is found an uneven, partially collapsed mass, the cyst-contents lying free in the abdomen.

Differential Diagnosis.—Ovarian tumors which have ascended into the abdomen, and especially when attached by a long pedicle, are readily confounded with tumors of other abdominal organs, notably those of the kidney. In children the difficulties are enhanced by the relatively close relationship of the abdominal organs to one another. McBurney has recorded a case of ovarian tumor which, on account of the position of the tumor and definite intervening space above the symphysis, was diagnosed as sarcoma of the kidney. This opinion was strengthened by the presence of albumin and a few blood-cells in the urine. On the other hand, an ovarian cyst has been suspected and at operation an enormous cystic kidney discovered. Tumors originating in the kidney preserve something of the renal form, are readily displaced into one or other lumbar region, are attached above, and have their position behind the intestine, which gives a clear percussion note.

Sacculated Peritoneal Exudate.—In cases of tuberculous peritonitis there is sometimes found a localized collection of fluid which is walled off from the general peritoneal cavity, and, forming a fluctuating tumor, dull on percussion, the note being unaltered with change of position, closely simulates an ovarian cyst. In adults such tumors have been operated on for supposed ovarian cyst. An interesting case is reported by R. Johnson where the contrary error in diagnosis was made in a child. The patient, five years of age, had been ailing for six months, during which time she had suffered from numerous attacks of abdominal pain, attended with diarrhœa and vomiting. The paroxysms increased in frequency and severity, and three weeks before operation it was noticed that the abdomen was swollen. On admission to the hospital the child looked ill and had a temperature of 101° F. The abdomen was distended and the right half occupied by a tender, elastic swelling, most prominent below and to the right of the umbilicus. The diagnosis of encysted tuberculous peritonitis was made and operation undertaken. An incision in the right linea semilunaris exposed a cyst of the right ovary, the walls of which were hemorrhagic, and the pedicle had undergone two complete rotations. Ovariotomy was performed and was followed by recovery.

A dilated bladder is recognized by the catheter. It must be remembered, however, that the condition of the bladder may be due to the presence of a tumor in the pelvis. The case recorded by Routier, above cited, demonstrates the possibility of this error.

Hepatic Tumors.—Hydatid cysts, lardaceous degeneration, or the rare congenital hypertrophies are the only conditions in the liver likely to simulate an ovarian tumor. Amyloid degeneration is always secondary to a chronic wasting or suppurative disease, and the sharp, even margin of the liver is preserved. Hydatid cysts are recognized by the position, possibly by the characteristic crepitation. Professor Jüon, of Nantes, operated on a twelve-year-old girl for an abdominal tumor supposed to be a case of hydatid of the liver. On opening the abdomen with caustics a multilocular ovarian tumor presented and was excised. The patient recovered.

Tumors of the *liver*, *spleen*, and *pancreas* have their origin in the upper abdomen, and in the case of the liver and spleen the tumor may often be observed to move synchronously with each respiration; and, further, on making a rectal examination, the pelvic organs are found intact.

In *retro-peritoneal disease*, which is not uncommon in young children, the tumor springs from the pelvis by a broad base, hence is more or less immobile, and the function of one or more organs is seriously impaired. Thus, in the case of a child of three years, in my hands, a soft sarcoma springing from the anterior face of the sacrum in position, shape, and consistency simulated an ovarian cyst, but its retro-peritoneal position was inferred from the marked deviation of the rectum and from the frequent agonizing efforts at micturition, caused by a hydro-ureter of the right side. Finally, more than once an ovarian tumor has been at first mistaken for pregnancy, the subsequent history, however, soon changing the diagnosis.

Diagnosis of the Variety of Tumor.—Monocystic tumors are recognized by their smooth, rounded outline and well-marked wave of fluctuation.

Multilocular cysts usually present a more uneven, nodulated surface, and variations in the consistency of different portions may frequently be made out.

Solid tumors, also often of irregular contour, are non-fluctuant. In general they are of more rapid growth, and, as they are for the most part of a malignant nature, are attended by more marked and early cachexia. Further, ascites is a frequent accompaniment of solid tumors.

Under four years of age the probabilities are in favor of the tumor being a dermoid cyst or a sarcoma.

Treatment.—The treatment of ovarian tumors is removal by means of abdominal section.

On account of the large percentage of malignant tumors and the consequent dangers of delay, the operation should be performed as soon as the patient can be suitably prepared for it. The disadvantages of delay greatly overbalance any apparent advantage to be secured by postponement in order to build the patient up; the best tonic is the relief afforded by the removal of the growth.

Where there is doubt as to the advisability of doing an operation on account of the possible extension of a malignant disease to other parts, the patient should have the benefit of the doubt and an exploratory operation should be made. I have seen a little girl, who appeared quite cachectic and reduced by a large tumor associated with ascites, rally and recover when recovery seemed at first almost hopeless.

The arrangements for the operation must be so carefully made that there will be no unnecessary delays, in order to limit the shock as much as possible. Chloroform is the best anæsthetic, at least at the outset; after the patient has been brought under its influence, ether may be substituted.

Every care must be taken throughout to keep the little body warm by wrapping in flannels and applying hot-water bottles around the body and by the legs and arms; the most important place to protect is the thorax.

The general conduct of the operation is the same as that in the adult. A median incision is made below the umbilicus, taking special care in this case to avoid opening the bladder, which is located in the lower abdomen.

If the tumor is cystic, it is evacuated and turned out and its pedicle clamped and the tumor removed, after which ligatures are applied in place of the clamps.

In the event of a malignant tumor it will be necessary to make the incision large enough to turn the tumor out by its small axis, so as to avoid rupturing it and producing hemorrhage and distributing its elements over the peritoneum.

As far as possible the intestines should be kept covered with warm gauze and protected from exposure.

The rule regarding the opposite ovary must always be to leave it untouched unless it is similarly diseased.

The abdominal incision will be best closed by a continuous catgut suture to the peritoneum and interrupted sutures of fine silk-worm gut embracing the fascia and the skin.

The after-care for the first week must be directed to securing perfect quiet in a darkened room and small quantities of nutritious food at frequent intervals.

In the following table of ovariotomies it is shown that such operations on children under four years of age are attended by a mortality of more than one-half of the cases; but, on the other hand, the results obtained in the case of older children are remarkably favorable. It will further be noticed that the high mortality in the former class is apparently uninfluenced by the character of the tumor, while in the latter an analysis of the cases makes it evident that the prognosis largely depends on the benignity or otherwise of the growth. Thus:

	CASES.	DEATHS.
Cysts	55	4
Dermoids	47	10
Solid tumors	24	8

The following table, containing an analysis of one hundred and twenty-six cases, is given in order to put before the reader all the available material relating to this important subject, as well as to furnish a satisfactory basis for future additions and analyses.

REFERENCE.	AGE.	OVARIAN TUMOR.	COMPLICATIONS OF OVARIOTOMY.	RESULT.	REMARKS.
D'Arcy Power, Brit. Med. Jour., p. 850, March 5, 1898.	4 months.	Cyst.	. . . .	Recovery.	. . . .
Alcock, Lancet, 1871, vol. ii.	3 years.	Mutilocular cyst.	Adhesions.	Death.	. . . .
Boldt, Proc. Path. Soc. N. Y., 1888.	4 years.	Cyst.	The cyst suppurating and everywhere adherent; treated by incision and drainage.	Death.	Complicated by acute meningitis.
Schwartz, Archiv f. Gyn., Bd. xiii. S. 475.	4 years.	Adeno-cystoma.	Omental adhesions.	Recovery.	Precocious menstruation.
Baer, N. Y., personal communication.	4 years.	Cyst.	. . . .	Death.	. . . .
Bangs, Am. Gyn. and Obst. Jour., Dec., 1896.	4¾ years.	Multilocular fibro-cystoma.	The cyst adherent above and to the left of the umbilicus.	Recovery.	. . . .
R. Johnson, Lancet, 1897, vol. i. p. 1093.	5 years.	Cyst.	Two complete rotations of the tumor on its axis.	Recovery.	. . . .
Syme, Austral. Med. Jour., vol. xv. p. 442.	5 years.	Cyst.	. . . .	Recovery.	. . . .
Lucas, Trans. Clin. Soc. London, vol. xxi. p. 224.	7 years.	Multilocular cystoma.	. . . .	Recovery.	Previous to operation there had been a hemorrhagic vaginal discharge.
Hamaker, N.Y. Med. Jour., Sept., 1889.	7 years.	Multilocular cystoma.	. . . .	Recovery.	. . . .
Chenoweth, Am. Jour. of Obst., vol. xv.	7⅔ years.	Multilocular cystoma.	. . . .	Recovery.	. . . .
Duchamp, Arch. de Tocol., 1884, p. 23.	8½ years.	Cyst.	During the operation respirations ceased and the pulse became weak and slow. Artificial respiration was employed for ten minutes.	Recovery.	. . . .
Parkes, Am. Jour. of the Med. Sci., 1890, p. 258.	9 years.	Cyst.	Universal adhesions.	Death.	The patient got up on the third day, and on the fifth died from exhaustion.
Péan, Arch. de Tocol. et de Gyn., 1893, p. 580.	10 years.	Multilocular cystoma.	Omental adhesions.	Recovery.	. . . .
Vincent, Lyon Méd., t. lxxiii.	10 years.	Cyst.	Adhesions with the colon and omentum. Peritonitis. Temperature 39.8° C.	Recovery.	. . . .
Kollock, Jour. Am. Med. Asso., Chicago, vol. xvii. p. 890.	11⅔ years.	Multilocular cystoma, double.	. . . .	Recovery.	Menstruation had been regular for a year. Ten months later her health was excellent.
Sims.	11½ years.	Cyst.	. . . .	Recovery.	. . . .

Keith, Brit. Med. Jour., 1878, vol. ii. p. 592.	12 years.	Semi-solid tumor.	Ascites.	Recovery.	. . . .
Jüon, Gaz. Heb., 1869, p. 396.	12 years	Multilocular cystoma.	Firm adhesions.	Recovery.	. . . .
Thomas, personal communication.	12 years.	Cyst.	. . . .	Recovery.	In one year a tumor equally large developed in the other ovary, and before operation could be performed it burst, causing death.
Terrillon, Bul. Gén. de Thér., t. cxv. p. 529.	13 years.	Cyst.	. . . .	Recovery.	. . . .
Homans, "Laparotomies," 1887.	13 years.	Multilocular cystoma.	. . . .	Recovery.	After the operation her health was good and she menstruated regularly.
Hume (Bland Sutton).	13 years.	Cyst.	. . . .	Recovery.	. . . .
Bell, Lancet, 1887, vol. i. p. 418.	13 years.	Unilocular cystoma.	Slight omental adhesions.	Recovery.	. . . .
Keith, Obst. Jour. Great Brit., vol. iv.	13 years.	Multilocular cystoma.	. . . .	Recovery.	. . . .
Koeberlé, Gaz. Méd. de Stras., September, 1875.	13 years.	Multilocular cystoma.	. . . .	Recovery.	. . . .
Taber Johnston, personal communication.	13 years.	Cyst.	. . . .	Recovery.	. . . .
De Sinéty, Gaz. Méd. de Paris, 1875, No. 27.	13 years.	Multilocular cystoma.	. . . .	Recovery.	. . . .
E. Cushing, Boston, personal communication.	13 years.	Monocyst.	Operation performed during an attack of typhoid fever, with the temperature 104.5° F.	Recovery.	Operation was undertaken on account of dyspnœa.
Walter, Brit. Gyn. Jour., 1895, p. 51.	13 years.	Multilocular cystoma.	One locule ruptured and abdomen filled with viscid yellow fluid. At one point the pedicle was adherent to the colon.	Recovery.	She menstruated for the first time five months later. Two years after operation was healthy.
Thornton, Brit. Med. Jour., 1887, vol. ii.	14 years.	Multilocular cystoma.	Omental adhesions.	Recovery.	. . . .
Bryant, "Surgery"	14 years.	Multilocular cystoma.	. . . .	Recovery.	. . . .
Baumgarten, Virchow's Archiv, 1884, Bd. 97.	14 years.	Multilocular cystoma	Tubercular peritonitis and the cyst-wall studded with tubercles. Adhesions and short vascular pedicle.	Recovery.	. . . .
Lazerevich (Bland Sutton).	14 years.	Cyst.	. . . .	Recovery.	. . . .
Homans, "Laparotomies," 1887.	14 years.	Multilocular cyst.	. . . .	Recovery.	Later married and gave birth to one child.
Terrier, Rev. de Chir., 1885, p. 34.	14 years.	Unilocular cyst.	. . . .	Recovery.	Well eight months later.

REFERENCE.	AGE.	OVARIAN TUMOR.	COMPLICATIONS OF OVARIOTOMY.	RESULT.	REMARKS.
Routier, Rev. Gén. de Clin. et de Thér., Paris, 1895.	9 years.	Dermoid.	Tumor impacted in the pelvis.	Death.	. . . .
Polotebnow, Central. f. Gyn., 1887, Bd. i.	9 years.	Dermoid.	. . . .	Recovery.	. . . .
Omori and Ikeda, Central. f. Gyn., Bd. xvi.	10 years.	Dermoid.	. . . .	Recovery.	. . . .
Péan, Clin. Chir., 1887–88.	11 years.	Dermoid.	Omental adhesions.	Recovery.	. . . .
Barlow and Marsh, Clin. Trans. London, vol. xi. p. 175.	12 years.	Dermoid.	. . . .	Recovery.	. . . .
Griffiths, Trans. Path. Soc. London, vol. xxviii.	12 years.	Dermoid.	. . . .	Recovery.	. . . .
Schultze, Deut. Zeit. f. Prakt. Med., 1876.	12 years.	Dermoid.	. . . .	Death.	. . . .
Péan, Clin. Chir., 1875–76.	12 years.	Dermoid.	. . . .	Recovery.	. . . .
Vincent, Lyon Méd., t. lxxiii.	12 years.	Dermoid.	. . . .	Death.	. . . .
Kelly, Johns Hopkins Hospital, unpublished.	13 years.	Dermoid.	Twisted on axis two and one-half times.	Recovery.	. . . .
Jessop, Lancet, 1871, vol. ii.	13 years.	Dermoid.	Slight adhesions anteriorly.	Death.	At the autopsy cancerous deposits were found in the liver, right supra-renal, and in the mesenteric glands.
Keith, Obst. Jour. Great Britain, vol. iv. p. 31.	13 years.	Dermoid.	. . . .	Recovery.	. . . .
Thornton, Med.-Chir. Trans., vol. lxx.	13 years.	Dermoid.	Torsion of pedicle.	Recovery.	. . . .
Haward, Lancet, 1886, vol. i. p. 920.	13 years.	Dermoid.	Peritonitis due to twisted pedicle, and tumor almost gangrenous.	Recovery.	Six months later the child was in perfect health.
Herzog, Berlin. Klin. Woch., 1897, S. 1114.	14 years.	Dermoid of both ovaries.	. . . .	Recovery.	. . . .
Mundé, Trans. Am. Gyn. Soc., 1878, vol. ii.	14 years.	Dermoid.	. . . .	Recovery.	The patient had not menstruated.
Homans, "Laparotomies," 1887.	14 years.	Dermoid.	. . . .	Recovery.	. . . .
Omori and Ikeda, Central. f. Gyn., Bd. xvi.	14 years.	Dermoid.	. . . .	Recovery.	. . . .
Bantock, Med.-Chir. Trans., vol. lxiv. p. 124.	14 years.	Dermoid.	. . . .	Recovery.	. . . .
Koeberlé, loc. cit.	14 years.	Dermoid.	. . . .	Recovery.	. . . .

Ibid.	14 years.	Dermoid.	. . . .	Recovery.	. . . .
Bantock, Med.-Chir. Trans., vol. lxiv. p. 122.	15 years.	Dermoid.	Twisted pedicle, tumor inflamed. Universal adhesions.	Recovery.	Died of obstruction the following year.
Giraldes, Gaz. Hebdom, March, 1867.	15 years.	Dermoid.	. . . .	Death.	. . . .
Von Szabo, Archiv f. Gyn., 1888, Bd. xxxii. S. 193.	15 years.	Dermoid.	. . . .	Recovery.	. . . .
Emanuel, Zeit. f. Geb. u. Gyn., Bd. xxv.	15 years.	Teratoma.	Two litres of fluid in abdomen. Metastatic nodules in omentum excised.	Recovery.	Died from recurrence four months after operation.
Omori and Ikeda, Berlin. Klin. Woch., 1890, No. 7.	15 years.	Dermoid (double).	. . . .	Recovery.	. . . .
Homans, "Laparotomies," 1887.	15 years.	Dermoid.	. . . .	Recovery.	Since is in good health and menstruates.
Hoffman, Am. Jour. of Obst., vol. xxxvi. No. 3, 1897.	33 months.	Sarcoma.	Patient in an exhausted condition, and the tumor adherent in front and around the left side.	Death.	. . . .
Cameron, Glasgow Med. Jour., 1889.	3½ years.	Sarcoma.	Marked ascites.	Death.	. . . .
Foerster, Am. Jour. of Obst., vol. xxxi.	4½ years.	Sarcoma.	. . . .	Recovery.	Died in a few months of recurrence.
Byford, Chicago Med. Rec., 1891–92, vol. ii.	4⅔ years.	Sarcoma.	Omental adhesions and half twist of pedicle.	Recovery.	Precocious menstruation.
Page, Lancet, December, 1895.	6 years.	Sarcoma.	. . . .	Recovery.	. . . .
Croom, Edin. Med. and Surg. Jour., 1893, p. 689.	7 years.	Sarcoma.	. . . .	Recovery.	There had been a continuous discharge of blood from the vagina for seven months; after operation this ceased, and two years later she was well.
Chenoweth, Am. Jour. of Obst., vol. xv.	8 years.	Sarcoma.	. . . .	Recovery.	. . . .
Gussenbauer, Wien. Med. Woch., 1894, No. 17.	8 years.	Carcinoma.	. . . .	Recovery.	Half a year later there was no evidence of recurrence.
Malins, Lancet, 1890, vol. i. p. 1174.	9 years.	Cysto-sarcoma.	Temperature, 103.2° F.; pulse, 120, weak. Omentum adherent anteriorly.	Death.	. . . .
Forbes, Austral. Med. Jour., Melbourne, 1894, vol. xvi.	9 years.	Myxo-sarcoma.	Ascites.	Recovery.	. . . .
Leopold, Central. f. Gyn., 1894, Bd. xviii.	9 years.	Carcinoma.	. . . .	Recovery.	One year later died of recurrence.

REFERENCE.	AGE.	OVARIAN TUMOR.	COMPLICATIONS OF OVARIOTOMY.	RESULT.	REMARKS.
McBurney, Ann. of Surg., Philadelphia, 1895, vol. xxi. p. 706.	10 years.	Sarcoma.	. . . .	Recovery.	. . . .
Wagner, Archiv f. Klin. Chir., Bd. xxx. S. 504.	10 years.	Sarcoma.	. . . .	Recovery.	. . . .
Croom, Obst. Trans. Edin., vol. xiv. p. 93.	11 years.	Sarcoma.	. . . .	Recovery.	. . . .
Kelly, Philadelphia, private practice.	12 years.	Sarcoma.	. . . .	Recovery.	Died two years later of dysentery.
Eckhardt, Deut. Med. Woch., 1895, S. 96.	13 years.	Sarcoma.	. . . .	Recovery.	The opposite ovary, which was cystic, was also excised. One and a half years later in good health and showing no evidence of recurrence.
Wagner, Archiv f. Klin. Chir., Bd. xxx. S. 504.	13 years.	Sarcoma.	Ascites. Adherent to the omentum at numerous points and to the cæcum.	Death.	. . . .
Leopold, Central. f. Gyn., Bd. xviii. S. 1171.	13 years.	Carcinoma.	Adherent to the sigmoid flexure. The tumor ruptured during operation, causing severe hemorrhage. All was not removed.	Recovery.	Died three months later of recurrence.
Bode, Central. f. Gyn., Bd. xviii. S. 1171.	13 years.	Sarcoma.	The tumor filled the entire abdomen, growing up under the sheath of the abdominal muscles.	Recovery.	In good health four years later.
Anderson, Bland Sutton, Dis. of Tubes and Ovaries.	13 years.	Sarcoma.	. . . .	Recovery.	Recurrence and death in a few months.
Smith, Lancet, 1874, vol. ii. p. 501.	14 years	Malignant tumors of both ovaries (carcinoma).	. . . .	Death.	. . . .
Gage, Jour. Am. Med. Asso., December, 1894.	15½ years.	Sarcoma.	Irregular tumor filling the abdomen and everywhere adherent.	Recovery.	Had menstruated for one year. Was well and married five years after operation.
Thornton, Med. Times and Gaz., 1883, vol. i. p. 211.	15 years.	Carcinoma.	. . . .	Death.	. . . .
Von Szabo, Archiv f. Gyn., Bd. xxxii. S. 193.	15 years.	Carcinoma.	Recent adhesions. Parenchymatous hemorrhage into the abdominal walls and intestines.	Death.	. . . .

CONGESTION AND HEMORRHAGE.

Congestion of the Fallopian tubes and ovaries is an occasional complication or sequela of the acute infectious diseases of childhood, such as measles, scarlet fever, and varicella; also typhoid fever, cholera, and purpura hæmorrhagica. Hennig,[1] at autopsy on a young girl dead of typhoid fever, found a hemorrhagic necrosis of the tube with rupture into the intestines.

In cases of atresia of the vagina or cervix the tubes secondary to the uterus become distended with the retained menstrual blood, and finally, unless relieved by operation, rupture occurs, the patient dying from a resulting peritonitis or directly from the hemorrhage.

OVARITIS AND SALPINGITIS.

Inflammatory disease of the tubes and ovaries is sometimes present in young children, and is usually a direct sequence of a neglected infantile vulvo-vaginitis. The frequency of this affection is indicated by the results of Marx's[2] investigations. In a series of fifty consecutive autopsies on children he found five cases of pus-tubes. The clinical picture is similar to that observed in adults. The child, apparently in good health, is suddenly attacked with high fever, pain in the abdomen radiating down the thighs, frequent painful micturition, nausea, and thirst. Palpation reveals tenderness in either inguinal region, and on examining per rectum the appendages are found swollen and tender. The symptoms may lessen or disappear, and cure seem certain; but at puberty the establishment of the catamenia is accompanied by all those distressing symptoms so often called physiological, but which in reality often indicate a grave pathological condition. Later pregnancy may light up an old inflammation; more commonly, however, sterility is the result. Sänger believes that many cases of old localized pelvic peritonitis in young girls are due to gonorrhœa in childhood.

Etiology.—Salpingo-ovaritis is in most instances consecutive to a neglected vulvo-vaginitis, and may have as the primary cause,—

1. Defective hygiene.
2. Gonococcus of Neisser.
3. Acute infectious diseases, especially measles, scarlatina, and diphtheria, and is sometimes an accompaniment of such general diseases as scrofula and rhachitis.

Marx, after carefully investigating a large number of cases, states that the cases of vulvo-vaginitis having a gonorrhœal origin are the exception, the greater number being due to other infective organisms. He points out the frequency with which every practitioner meets with instances among children of the best classes in which neither clinically nor histologically is there any evidence of venereal origin. These cases of vulvo-vaginitis of non-venereal origin are equally disposed to be followed by inflammation of the adnexa. Frequently the use of common toilet articles is the means of infection. In a ward of the Hospital of Lyons there was an epidemic of vulvo-vaginitis affecting thirty children between the ages of two and seven years, due to the common use of the thermometer,[1] and starting from a child with gonorrhœal vulvitis. Opposed to Marx's view, the majority of writers find that in a large proportion of their cases of infantile vulvo-vaginitis the gonococcus is demonstrable. The frequency with which these cases are gonorrhœal is shown in the following table:

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[1] Sänger, 1878.

[2] Rev. de Thérap. Méd.-Chir., Paris, 1895.

	Number of Cases.	Gonorrhœal.
Fisher[2]	54	50
Cassel[3]	30	24
Schultz[4]	10	9
Spaeth[5]	21	14

The onset is insidious, and the symptoms for the most part confine themselves to a more or less abundant discharge, varying from a mere oozing to a copious, thick, greenish-yellow discharge. This may disappear in a few weeks or may continue for months, the child becoming pale and weak, but with no more serious result. In other cases the child becomes the subject of an acute or chronic pelvic inflammation. Sigwish Lovén[6] relates a case occurring in a little girl five years old, belonging to a good family. She had complained in the morning of feeling ill, and vomited a few times; these symptoms were referred to errors of diet. Two days later a discharge was observed on the external genitalia and an acute vulvo-vaginitis with severe eczema discovered. For a few days she improved, but looked pale and was irritable. Suddenly symptoms of severe peritonitis with high fever developed. She quickly improved, but on the next day complained of pain in either shoulder, and the thought of gonorrhœal infection occurred. The following day a scarlatinoid rash appeared on face, neck, and chest, extending over the whole body, but there was no angina. She then complained of pain in the knees. The abdominal symptoms, which had improved, returned with great severity, and a thick, yellow discharge was found which contained gonococci. Death occurred on the twenty-second day of her illness. At autopsy there was found purulent peritonitis, the mucosa of the vagina and lower portion of the cervix swollen and red, the upper portion of the cervix less swollen. Along the sides of the uterine cavity and at either cornua the mucosa was intensely swollen nd red. Both tubes contained thick yellow pus, and in the left ovary here was an abscess. Gonococci were found in the vaginal secretion, but in the pus from the abdomen there were only bacteria resembling the streptococcus pyogenes.

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[1] Mercredi Méd., 1896, No. 46.

[2] Deutsche medicinische Wochenschrift, 1895, No. 51.

[3] Berliner klinische Wochenschrift, 1893, No. 29.

[4] Beitr. z. Path. u. Ther. der Weib. Gonorr.

[5] Huber, Archives of Pediatrics, 1889.

[6] Centralblatt für Gynaekologie, Bd. 10.

Sänger observed an instance where a whole family was infected with gonorrhœa; one of the members, a child three and a half years old, died of purulent peritonitis consecutive to the vaginal infection. An autopsy in this case was not made.

Diagnosis.—In older children the recognition of a salpingo-ovaritis hould not be especially difficult, the symptoms corresponding to those rising from a similar condition in adults, a history of an infantile vulvo-aginitis confirming the diagnosis. In infancy and early childhood, where bjective symptoms are inadequately expressed, the diagnosis is rarely ossible. F. Huber[1] reports an interesting case; the child, seven years of ge, for a short time had suffered from a vaginal discharge, with urethritis nd soreness in the pelvis. Suddenly she presented all the symptoms of ppendicitis with perforation. Laparotomy was performed on the second ay and the appendix found healthy, but the intestines were distended nd covered with lymph, and the right Fallopian tube, thickened and in-amed, was evidently the channel of infection.

TUBERCULOSIS.

While tuberculosis of the Fallopian tubes and ovaries is not uncom-on, and different observers state that from five to ten per cent. of the ases of salpingo-ovaritis operated on are of a tuberculous nature, in chil-ren it is rare, but still sufficiently frequent to be of interest from the linical as well as from a scientific stand-point.

In the literature there are records of twenty-one cases of pelvic tuberculosis in children from ten weeks to fifteen years of age. In most instances the tubes were affected, sometimes with involvement of the endometrium or the ovaries. In only one case it was stated that the ovaries alone were tubercular. An analysis of the cases reported shows that the relative frequency with which the organs are the seat of the disease is as follows: tubes, nineteen; uterus, seventeen; ovaries, seven; vagina, one; cervix, one.

Tuberculosis of the tubes and ovaries is usually secondary, and in chil-ren, at least, its presence is masked by the symptoms arising from the pulmonary or other primary affection, the attention seldom being directed o the pelvic organs. In three cases there was clear evidence of a primary affection of the genitals.

The varieties of tuberculous salpingo-ovaritis may be divided into three classes: *miliary tuberculosis*, *caseous tuberculosis*, *chronic fibroid tuberculosis*.

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[1] Archives of Pediatrics, 1889, vol. vi. p. 887.

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TUBERCULOSIS.

While tuberculosis of the Fallopian tubes and ovaries is not uncommon, and different observers state that from five to ten per cent. of the cases of salpingo-ovaritis operated on are of a tuberculous nature, in children it is rare, but still sufficiently frequent to be of interest from the clinical as well as from a scientific stand-point.

In the literature there are records of twenty-one cases of pelvic tuberculosis in children from ten weeks to fifteen years of age. In most instances the tubes were affected, sometimes with involvement of the endometrium or the ovaries. In only one case it was stated that the ovaries alone were tubercular. An analysis of the cases reported shows that the relative frequency with which the organs are the seat of the disease is as follows: tubes, nineteen; uterus, seventeen; ovaries, seven; vagina, one; cervix, one.

Tuberculosis of the tubes and ovaries is usually secondary, and in children, at least, its presence is masked by the symptoms arising from the pulmonary or other primary affection, the attention seldom being directed to the pelvic organs. In three cases there was clear evidence of a primary affection of the genitals.

The varieties of tuberculous salpingo-ovaritis may be divided into three classes: *miliary tuberculosis, caseous tuberculosis, chronic fibroid tuberculosis.*

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[1] Archives of Pediatrics, 1889, vol. vi. p. 887.

[illegible]

[illegible]

[illegible]

[illegible]

1. From areas of tuberculosis already existing, either through the blood or by extension from the peritoneum or neighboring organs.

2. By direct infection through the vagina.

In children tuberculosis of the genital organs caused by direct infection from without must be extremely rare, and careful inspection will in most instances reveal a latent focus, particularly in the lymph-glands. The most common source of the genital infection is from the lungs, but occasionally the predominating symptoms, as well as the most advanced lesions that are found at autopsy, have shown either the intestine or the mesenteric glands to be the probable seat of the primary lesion. This origin might also be suspected on account of the frequent occurrence of tuberculosis of the alimentary tract, the mesenteric and the retro-peritoneal glands in children.

The peritonitis so often associated may either be the source of the genital tuberculosis or may be secondary to it. In Talamon's case, a child eleven years of age, who gave a history of four months previously having had what was called an inflammation of the bladder, was attacked with alternating diarrhoea and constipation, accompanied by pain in the abdomen and a temperature suggestive of tuberculosis. For three weeks there was cough. Death resulted from meningitis. Autopsy showed miliary tuberculosis of the meninges, base of brain, and lungs, and ulceration of Peyer's patches. Douglas's cul-de-sac was transformed into a pus-sac and the ovaries infiltrated with caseous material in which tubercle bacilli were demonstrable. The abdominal peritoneum was healthy. In this case the origin of the tuberculosis may have been in the alimentary canal, but the healthy condition of the abdominal peritoneum and the caseous nature of the tubes and ovaries, together with the indefinite history of early pelvic disease, are somewhat suggestive of a primary genital tuberculosis. In the case reported by Edis the autopsy findings are also suggestive of a primary pelvic affection.

At the Johns Hopkins Hospital four cases of pelvic tuberculosis in children have been observed, three being autopsy cases, and in one the process was undoubtedly primary in the genital organs. The subject was a mulatto child eight years of age. Up to within one month of her death she had been in apparently perfect health. She then presented symptoms of commencing cerebral meningitis, and soon afterwards a general miliary tuberculosis developed. At the autopsy there was found to be an acute miliary tuberculosis of all the organs, while the tubes and uterus were the seat of a chronic caseous process, the ovaries and vagina also being involved.

Another child five years old was operated on by Dr. Halsted for an umbilical hernia. While releasing adherent intestines from the sac, a caseous nodule was found between the loops. Death occurred on the eighth day, and the autopsy revealed primary tuberculosis of the peritoneum and mesenteric glands, with caseous tuberculosis of the Fallopian tubes.

In the third case, a colored child of three years, there were a chronic tubercular peritonitis, caseous tuberculosis of the Fallopian tubes and endometrium, and general tuberculosis.

A young colored girl fifteen years old entered the hospital five months ago, presenting symptoms of tubercular peritonitis. Her illness had begun six weeks before with pain and swelling of the abdomen, difficult locomotion, fever, and loss of flesh and strength. Menstruation began at thirteen, and occurred regularly for one year, but since has been less frequent. There has always been pain with the menses, and she suffers from a copious whitish leucorrhœal discharge. On making an exploratory section the peritoneum was found studded with tubercles and the pelvis converted into a pus-sac containing the pelvic organs matted together into an indefinite mass. Vaginal puncture was then made and drainage inserted. She recovered from the operation, and her condition is greatly improved. Of these four cases which have come under my observation, three belonged to the colored race.

Maas cites a case of primary genital tuberculosis where he believes that the channel of infection was possibly by means of the umbilicus.

There being so few cases recorded of tuberculosis of the tubes and ovaries in children, I have thought it well to make brief abstracts of the lesions present in the cases collected from the available literature.

REFERENCE.	AGE.	
Cheatle, Lancet, 1891, vol. ii. p. 1098.	1¾ years.	Pulmonary tuberculosis, the right lung containing an abscess. Tuberculosis of the liver, right kidney, and peritoneum. *Both Fallopian tubes coiled and distended with pus,* the left opening into an abscess of the broad ligament, from which pus is exuding into the pelvic cavity. The proximal ends of the tubes, the uterus, and the vagina are healthy.
Mosler (Maas), Archiv f. Gyn., 1896.	2½ years.	Tuberculosis of the right lung; chronic fibro-tubercular peritonitis; perisalpingitis and periovaritis. *Caseous tuberculosis of both Fallopian tubes.* The uterus enlarged and containing thick fluid.
Ibid.	3 years.	Caseous pulmonary tuberculosis, tubercular pleurisy; fibro-tubercular peritonitis. Tuberculosis of the liver and spleen. *Caseous tuberculosis of the Fallopian tubes and endometrium.*
Cullen, Johns Hopkins Hospital Reports, vol. iv.	3 years.	Tuberculosis of the lymph-glands, lungs, pleura, pericardium, peritoneum, liver, spleen, kidneys, *Fallopian tubes, and endometrium.*
Frerichs (Maas).	3 years.	Tubercular peritonitis; recent tubercular pleurisy on the right side; caseous bronchial and mesenteric lymph-glands; tuberculosis of the spleen, kidneys, colon, and ileum. *Caseous tuberculosis of the Fallopian tubes and endometrium.*
Maas, Archiv f. Gyn., December, 1895.	5 years.	*Caseous tuberculosis of the Fallopian tubes and endometrium.* In the tubes the tubercles infiltrate the muscular coat, but in the uterus the mucosa only is involved; a fibrous tubercular band extends from the umbilicus to the pelvic organs. Miliary tuberculosis of the lungs, kidneys, and liver.
Williams, Johns Hopkins Hospital Reports, vol. iii.	5 years.	Primary tuberculosis of the peritoneum and mesenteric glands. *Caseous tuberculosis of the Fallopian tubes.* The uterus contains glairy fluid, but is not tuberculous.
Silcock, Trans. Path. Soc. London, vol. xxxvi. p. 303.	5 years.	*Fallopian tubes enormously distended with caseous tuberculous substance.* The uterus is also distended with caseous material. Miliary tubercles in the pia mater. Caseous substance in the tympanum. No bacilli are demonstrable in the tubes or uterus, but are found in a recent focus in the lungs.
Senn (Brouardel), Thèse de Paris, 1865.	5 years.	*The Fallopian tubes, the size of a quill and very flexuous, are distended with yellow friable material, which has infiltrated the walls almost to the peritoneal surface.* The uterine and tubal mucosa is injected and easily detached. The ovaries, apparently healthy, contain similar material. Tuberculous pneumonia and chronic enteritis.
Pillaud (Brouardel).	5 years.	*The ovaries are large and contain numerous crude and softened tubercles.* The left ovary is adherent to the rectum, into which it opens by an irregular aperture the size of a fifty-centime piece. Around the ulceration the intestinal mucosa is softened, and in this vicinity shows chronic inflammation. The lungs contain some tubercles, but the uterus and other organs are normal.

Talamon, Ann. de Gyn., 1878, t. i. 416.	6 years.	*Pelvic abscess. The ovaries enlarged and infiltrated with caseous tubercle tissue. The distal three to four centimetres of the Fallopian tubes adherent to the ovaries and tuberculous*; the proximal portions normal. The cervical canal is obliterated and the corpus uteri converted into a thin-walled sac containing greenish, viscid, non-tubercular fluid. Miliary tuberculosis of the meninges, base of brain, and lungs. Ulceration of Peyer's patches.
Beach, Obst. Trans., 1873, p. 57.	7 years.	*The Fallopian tubes and ovaries infiltrated with caseous tubercles.* The right ovary adherent to the appendix vermiformis, the left adherent to the small intestine.
Johns Hopkins Hospital Autopsy Records.	8 years.	Probable *primary tuberculosis of the genital tract,—ovaries, tubes, uterus, and vagina.* Tubercular peritonitis and meningitis; tuberculosis of the cerebrum, cerebellum, lungs, heart, liver, spleen, kidneys, and adrenals.
Cruveilhier, Traité d'Anat. Path. Gén., 1862, p. 814.	8 years.	*The Fallopian tubes, voluminous and zigzag, are distended with tubercular material which has infiltrated the walls. The uterus also voluminous and infiltrated with caseous tubercles.* Cervix normal. Tuberculosis of the lungs and bronchial glands.
Morton, Lancet, May 6, 1893, p. 1065.	8 years.	*Tuberculosis of the Fallopian tubes, uterus, and vagina.* One tube contained a tubercle the size of a walnut. (Dead of phthisis.)
Péron, Rev. Mens. de Mal. de l'Enf., Paris, 1894, t. xii. p. 372.	12 years.	Pott's disease: tuberculous pocket in the thorax. Tuberculosis of the lungs, bronchial glands, and surface of the liver. *Caseous tuberculosis of the Fallopian tubes and endometrium.* The ovaries, cervix, and vagina are healthy. Many tubercle bacilli found in the uterus.
Namais, Olshausen.	12 years.	*Tuberculosis of the ovaries*, the uterus distended to the size of a pear. Tuberculosis of the bronchial and mesenteric glands.
Edis, Obst. Trans. London, 1872, No. 99.	12 years.	The abdomen and pelvis contain greenish pus, and the intestines are matted together by the densely thickened peritoneum. The *left ovary is the size of an orange* and has thickened walls. Through *the posterior wall, which has given way*, pus is escaping into the peritoneal cavity. The *right ovary contains a small abscess. Caseous tuberculosis of the Fallopian tubes.* The uterus and abdominal organs are healthy. The lungs are not examined.
Kidd, Trans. Path. Soc. London, vol. xxxvi. p. 357.	14 years.	*Caseous tuberculosis of the Fallopian tubes and uterus.* Pulmonary tuberculosis. Disseminated tuberculosis. Tubercle bacilli are found in the uterus.
Senn (Talamon).	15 years.	*Tuberculosis of the Fallopian tubes and uterus.* (Dead of phthisis.)
Hutchinson, Path. Soc. London, 1857, vol. viii.	15 years.	*Tuberculosis of the left tube.* Miliary tuberculosis of the lungs, pleura, and dense bands of pleural false membrane.
Kelly, Johns Hopkins Hospital, unpublished.	15 years.	Tubercular peritonitis. Tuberculous pelvic abscess and the pelvic organs bound together in a dense mass. Phthisis. Curettings from the uterus show tuberculosis of the endometrium.

Clinical History.—The symptoms of tubercular disease of the Fallopian tubes and ovaries are not characteristic, and in most instances its recognition during early life is impossible. When secondary, the symptoms of the primary affection may so overshadow those of the pelvic complication that its presence is unsuspected. The presence, however, of a tubal tumor associated with phthisis or other tuberculous affection would immediately arouse suspicion as to the nature of the pelvic disease.

Primary tuberculosis limited to the tubes and ovaries may produce no symptoms whatever, or the symptoms may vary from those of a salpingitis to a severe pelvic abscess.

The diagnosis of tubercular disease of the tubes and ovaries has never, so far as I am aware, been made during life in children. The reason of this is that the disease of these organs is usually secondary to that of some other structure, notably the lungs, bronchial and mesenteric glands, or the intestines, upon which the attention is concentrated on account of the graver manifestations.

In those rare cases in which the disease is primarily pelvic, when the symptoms of pain, dysuria, difficult defecation, and a temperature curve characteristic of tuberculosis exist, no time should be lost in making a thorough investigation under anæsthesia.

Small hard masses lateral to or behind the uterus, associated with localized areas of fluctuation and varying resistance, point distinctly to tuberculosis.

Localization of the disease on one side is very rare,—in only one[1] instance out of a total of twenty-two cases.

Prognosis.—The prognosis is always grave. Primary genital tuberculosis may lead to phthisis, tuberculous peritonitis, or general miliary tuberculosis, and both primary and secondary affections may cause death through marasmus, hectic, or peritonitis produced by rupture of a pelvic abscess into the peritoneal cavity.

Treatment.—Unless contra-indicated by the general condition of the patient, tuberculous tubes and ovaries should be removed by means of abdominal section.

The apparent curative effect of abdominal section on some cases of tuberculous peritonitis indicates its use even where the peritoneum is involved with the genital affection. A coincident pulmonary tuberculosis, if in an early stage, does not contra-indicate the operation for the genital affection. Where too far advanced for excision, puncture and drainage of the pelvic abscess will relieve the distress and prolong the life of the patient.

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[1] Hutchinson's Case.

THE RÖNTGEN RAYS IN THE SURGERY OF CHILDREN.

BY W. W. KEEN, M.D.

CHILDREN are particularly favorable subjects for the use of the Röntgen rays, on account of the smaller amount of substance through which the rays have to pass than is the case with adults. The *X*-rays pass readily through the entire thickness of the body of children, so that the vertebræ and even the centres of ossification, the ribs, and to a less extent the contained viscera of the trunk, can be shown. In Plates VI. and XII. the heart is well shown. A fat or chubby child is no exception to this rule, because fat is the most transparent of all the tissues of the body to the *X*-rays. Even after the lapse of thousands of years the bones may be skiagraphed. Plate I. has a certain almost dramatic interest. It is the skiagraph of the hand of the mummy of an Egyptian princess between three and four thousand years old. The non-union of the epiphyses shows that she was about sixteen years old at the time of her death.

THE APPARATUS.

The apparatus to be used differs in no respect in children from that which is used in adults. Two different methods can be employed. First, that which has been least employed, an apparatus for generating static electricity connected with a Crookes focus-tube. My own personal experience, which is but limited, however, in the use of this form of apparatus, is that it is nearly as satisfactory as the other form of plant. The latter is now installed by a number of different firms in the United States. It consists of a storage-battery with an electro-motive force of about twelve volts and from ten to eighteen ampères. This is connected through a current-interrupter with a Ruhmkorff coil. From the coil the current is conducted through a Crookes vacuum-tube, of which different forms and kinds are now used. The best results are obtained with the form known as the "focus-tube." The Ruhmkorff coil should be of sufficient power to give a six-inch or, better, a ten-inch spark. The more powerful the apparatus the better the results and the less the dangers.

In children it is particularly important to have a powerful apparatus, because of the then shorter time necessary to obtain the skiagraph. This is of advantage in two ways: it avoids any deleterious effects from the coil, as the exposure is short; and it diminishes the probability of movement on the part of the child, and, therefore, of the blurring of the plate. Children usually become accustomed in a few minutes to the constant sparks, and are often attracted by them. I have seen more than one child fall asleep while the picture was being taken.

The sensitive plate, wrapped in two or three thicknesses of thin rubber cloth to exclude daylight, is placed as near to the part to be skiagraphed as possible. On account of the frequent movements of children, it is best that the plate should be secured to the body by bandages, which are entirely transparent to the rays. The film side of the plate should be next to the skin. That part of the body next the plate is most plainly pictured. Hence, if we wish to skiagraph the spine and get as little of the sternum as possible, the plate should be bandaged to the back; if we wish to skiagraph the sternum, the plate should be bandaged to the chest. The tube is placed on the side of the body opposite to the plate, at a distance of from one to two feet from the body.

Surface markings by which we can "orient" any deeper lesion can be shown by bismuth or by lead wire. Sinuses also, it is said, can be traced by injecting them with iodoform, as even a thin layer of iodoform is opaque to the *X*-rays. This has disappointed me in the only case in which I have had occasion to use it, but this was in the thick pelvis of a stout adult.

Arnozan and Bergonié,[1] in the case of a pleural fistula in a young girl, introduced a piece of lead wire through a hollow sound and thus revealed the exact course of the fistula.

Cannon[2] has mixed bismuth with the food administered to cats, and, as bismuth, like iodoform, is opaque to the *X*-rays, he has thus been able to study the motor phenomena of digestion by skiagraphing the movements and varying shape of the stomach and the progress of the food from the stomach into the duodenum.

It is very evident that when we are endeavoring to skiagraph a foreign body a single picture is not adequate for our purposes. For example, if a needle is in the foot and a skiagraph is taken with the plate on the sole and the tube above the dorsum of the foot, we shall obtain an exact notion of the position of the foreign body only in its transverse relation to the outer and inner borders of the foot. What its depth in the tissues is, whether it is near the dorsum of the foot or near the plantar surface, cannot be told from this one picture. If a second picture is taken with the plate sidewise to the foot and the Crookes tube on the other side, we can get not only the position of the foreign body from side to side, but also, by the second plate, its position in relation to the dorsum or sole of the foot.

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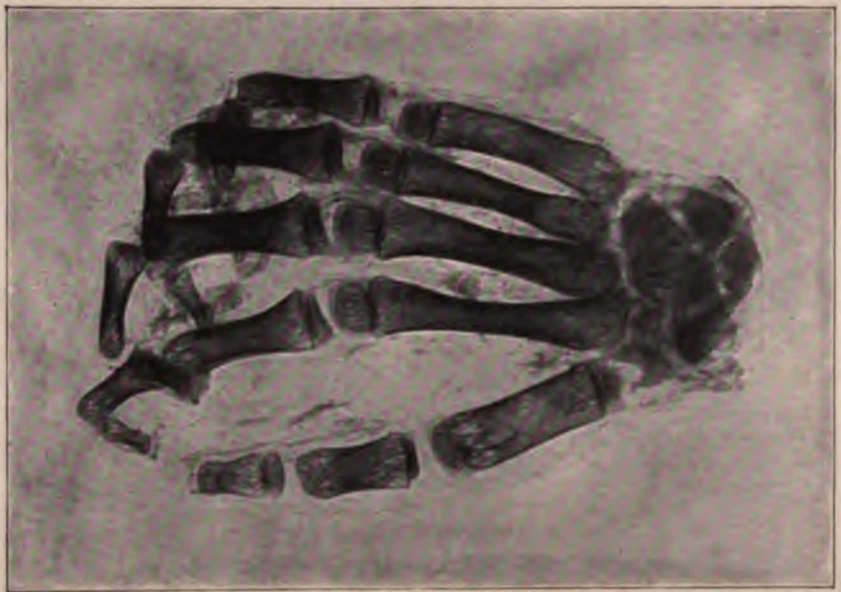
[1] Journal de Médecine de Bordeaux, November 21, 1897.

[2] American Journal of Physiology, 1898, p. 359.

PLATE I



Skiagraph of the hand of the mummy of an Egyptian princess, about sixteen years of age, between three thousand and four thousand years old. (Skiagraphed by John Carbutt, Philadelphia.)

lowing measures to obviate the danger: "It would seem advisable, first, to abandon the use of bulbs containing platinum; second, to substitute for them a properly constructed Lénard tube, containing pure aluminum only, a tube of this kind having, besides, the advantage that it is constructed with great mechanical precision, and therefore is capable of producing much sharper impressions; third, to use a protective screen of sheet aluminum, or, instead of this, a wet cloth or a layer of fluid; fourth, to make the exposures at a distance of at least fourteen inches, and preferably to expose longer at a greater distance."

Crocker[1] has given an excellent colored illustration of a severe case of this affection. Gilchrist[2] has found twenty-two reported cases of superficial affections of the skin. The skin becomes dark brown and exfoliates, and the hair falls out. At first there is no pain, but later the pain becomes very severe and of an aching, shooting, and throbbing character. Even the bones become tender, and the hand in a case he reports was useless for some weeks; sensation was impaired for a time, but was gradually recovered. Not only was the skin affected, but the skiagraph showed a distinct osteoplastic periostitis. One peculiarity about these deleterious effects is that they are not felt immediately, but, while the part exposed for any length of time to the *X*-rays at first shows no effect whatever, in the course of a few days, sometimes even so late as ten or twelve days after the exposure, the first symptoms of the dermatitis are observed in the discoloration and pain. The intensity of the irritation, as we should naturally expect, seems to be proportioned to the length of the exposure, but especially to the distance of the tube from the body; hence, as before stated, the importance of having a powerful apparatus, as it diminishes the time of the exposure very materially, and the wisdom of taking the precautions suggested by Tesla.

Oudin, Barthélemy, and Darier communicated to the Moscow Congress of 1897[3] the most elaborate paper on this subject yet published, based upon forty-four reported cases. They state that "the degree of injury varies inversely with the cube of the distance and not with the square, as is the case with light and heat," and recommend that the distance be not less than fifty centimetres (twenty inches). They have observed vomiting in two children, and in one adult the development of acute pulmonary tuberculosis as a result of the use of the *X*-rays.

The shoulder of an adult can now be well skiagraphed so as to show even the details of structure of the bones in a minute or two, and MacIntyre has taken a picture of the bones of the hand in a single flash, covering a minute portion of a second. As the sensitive skin of children is presumably more easily affected than the tougher skin of adults, the possibility and the

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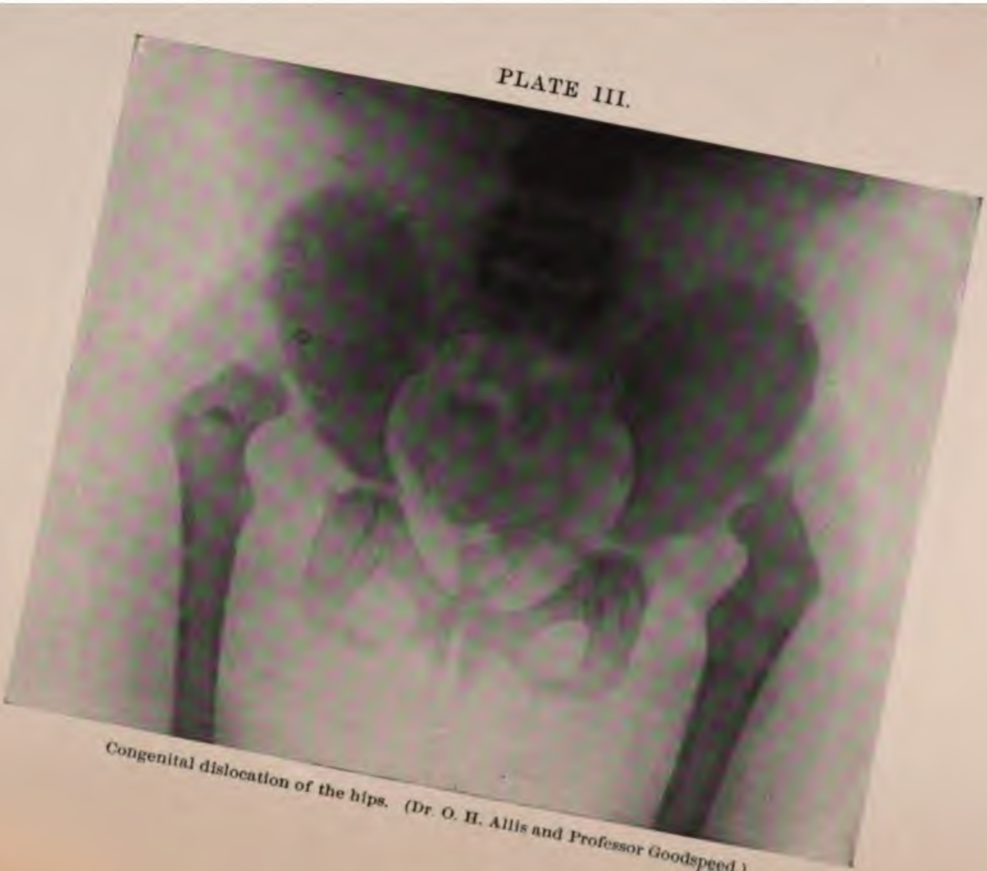
[1] British Medical Journal, 1897, vol i. p. 8.

[2] Bulletin of the Johns Hopkins Hospital, March, 1897.

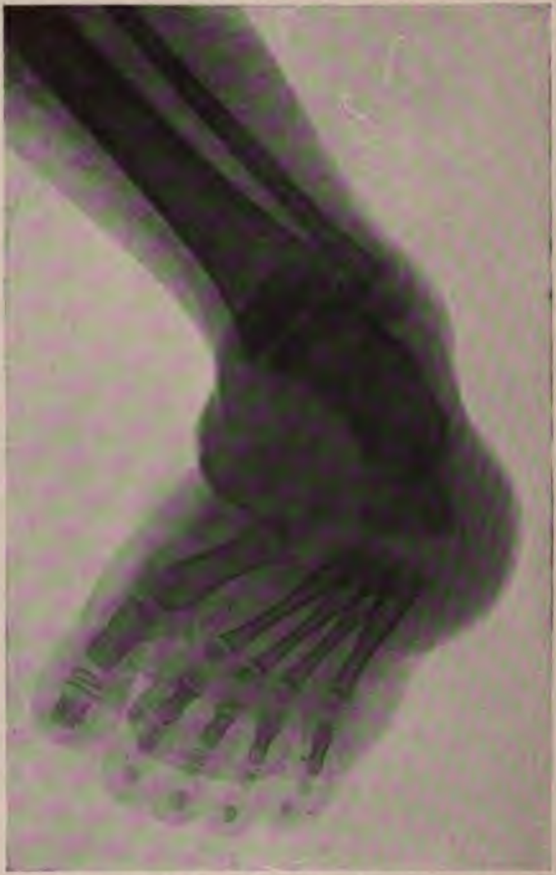
[3] Manchester Medical Chronicle, January, 1898, p. 290.

PLATE III.



Congenital dislocation of the hips. (Dr. O. H. Allis and Professor Goodspeed.)

PLATE IV.



Club-foot. (Dr. De Forest Willard and Professor Goodspeed.)

especial need of a brief exposure and a sufficient distance when skiagraphing children can be readily appreciated.

It is to be particularly noticed that not only the skin, but, as in the case reported by Gilchrist, the deeper portions of the body, as the bones, may be affected. Dr. A. W. Meisenbach,[1] of St. Louis, has described the case of an adult from whose stomach over one hundred different foreign bodies which had been swallowed were removed by operation, in which the exposure lasted for three hours. The patient afterwards suffered from an acute pneumonia of the right lung, the side on which the tube was placed. (Cf. *supra.*) Whether this and the failure of the parietal wound to heal were results of the *X*-rays is uncertain, but it seems to me very probable. In the newspapers it is said that Sorel, of Havre, has reported some cases of pneumonia thus caused. Dr. Bury, of Chicago, has informed me that three suits, for ten thousand, twenty-five thousand, and fifty thousand dollars, have been brought against one physician in three cases of ill results following the use of the *X*-rays. It is a question, therefore, whether in all cases it would not be prudent for physicians to require a written request from the patient, or, in the case of a child, from the parents or guardians, that a skiagraph be taken, and that the request should absolve the physician from any suit for damages for any deleterious effects from the use of the method.

THE USES OF THE *X*-RAYS.

(*a*) *The Anatomical and Physiological Uses.*—These will be, probably, rather limited, although it is dangerous to express an opinion as to the usefulness of the *X*-rays, so rapid have been the improvements constantly introduced in their use. Benedict[2] has especially studied the relations of the heart and the vessels. He has determined that in healthy persons the apex of the heart in systole moves away from the diaphragm. He states that this is more often true in children than in adults. The heart can easily be seen beating; the outlines of the liver and of the intestines and of the lungs can be at present indistinctly seen.

Schott[3] has clearly shown by skiagraphs, in cases of diseases of the heart, that gymnastics and hot baths have an immediate effect in reducing the capacity, and therefore the dilatation, of the heart. Vehsemeyer[4] has demonstrated a case of dexiocardia by this means. Petinelli[5] has studied from time to time the progressive reproduction of bone in a boy from whose leg the original bone had been removed. Abnormal teeth, especially in relation to the antrum, could be easily skiagraphed.

In Plate II. the joints should be especially observed. The wide intervals between the ends of the bones are due to the cartilaginous extremities, in which as yet no centres of ossification exist. It suggests the danger of suddenly jerking or lifting a young child by the arms. In the same plate the centres of ossification of the pieces of the sternum are shown (the oblique row of round disks), and also those of the lower epiphysis of the femur (not to be mistaken for the patella, which has not yet begun to ossify) and of the upper epiphysis of the humerus. No such centre of ossification has yet arisen at the upper end of the femur. Note also the separate pieces of the vertebræ and pelvis.

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[1] Journal of the American Medical Association, March 5, 1898, p. 513.

[2] Wiener medicinische Wochenschrift, 1896, S. 2035.

[3] Deutsche medicinische Wochenschrift, 1897, No. 14.

[4] Ibid., No. 12.

[5] Lancet, 1897, vol. i. p. 1244.

(*b*) As the *bones* are the least transparent to the *X*-rays, it is natural that the new discovery should be of more use in studying various lesions of the bones and of the joints than any other organs of the body.

Among the most useful applications may be mentioned, first, the study of *congenital deformities*. One of the first cases reported was by Professor Mosetig,[1] in which the great toe was deformed by a bifurcation of the last phalanx. Which of the two extremities of the phalanx was normal and which abnormal could not be determined by touch. A skiagraph of the foot instantly differentiated between the two, enabling the surgeon to remove the abnormal outgrowth. White[2] has published an excellent skiagraph of curvature of the forearm due to an arrest of development of the ulna at its distal extremity. Dowd[3] has pictured a case of cleft hand caused by fusing of the fourth and fifth metacarpals with a Y-shaped bifurcation at the distal extremity, with which the fourth and fifth fingers articulated.

In congenital dislocations of the hip the use of the *X*-rays will prove of great value to the orthopædist by determining the presence or absence of an acetabulum. If the acetabulum is present, efforts at reduction of the dislocation will be proper; if it is absent, Hoffa's or Lorenz's operation will have to be done. Plate III., from a patient of Dr. Allis, shows clearly the existence of the acetabulum below the level of the dislocated head of the femur, and that attempts at reposition rather than operation would be advisable.

The value of the *X*-rays in club-foot has been especially pointed out by Barwell,[4] with a number of illustrations. Willard[5] has also published an excellent illustration of club-foot, of which Plate IV. is a reproduction.

Spina bifida, I think, has not been shown in any skiagraphs that I have seen, but the method will probably be of use in showing positively the exact size of the opening in the bone.

Acquired deformities of the bones can also be skiagraphed to great advantage. For example, in cases of knock-knee the relative size of the two condyles of the femur, and whether the trouble is located in the femur or in the tibia, can be determined, and in bow-legs, the shape and relation of

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[1] British Medical Journal, February 8, 1896.

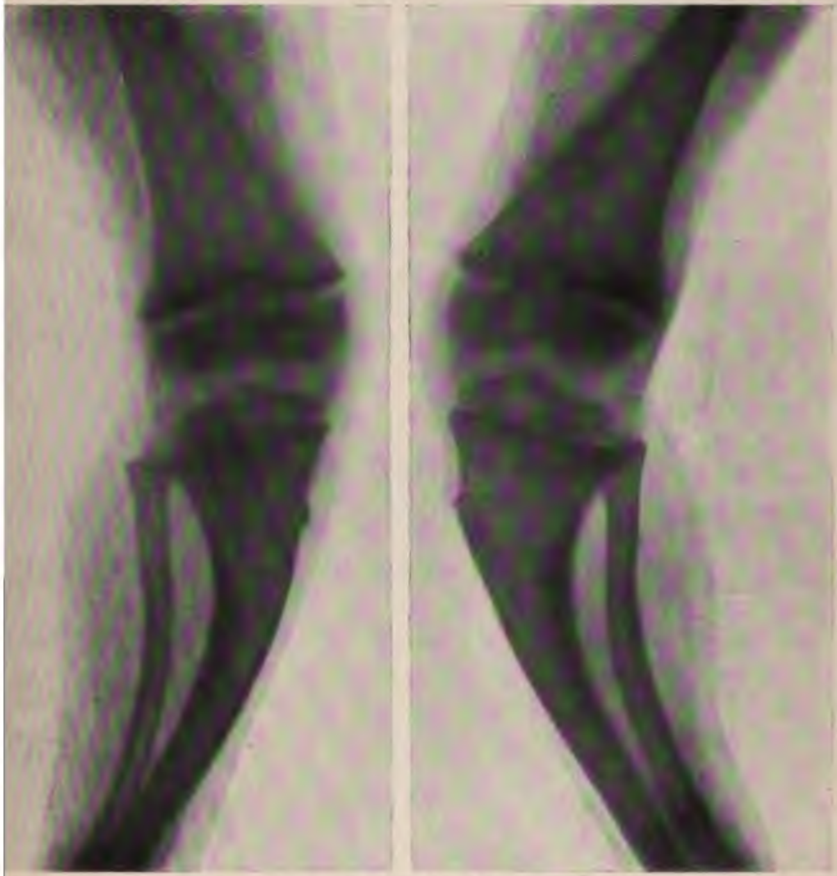
[2] American Text-Book of Surgery, Plate XXXIX.

[3] Annals of Surgery, August, 1896, p. 211.

[4] Lancet, 1896, vol. ii. p. 160.

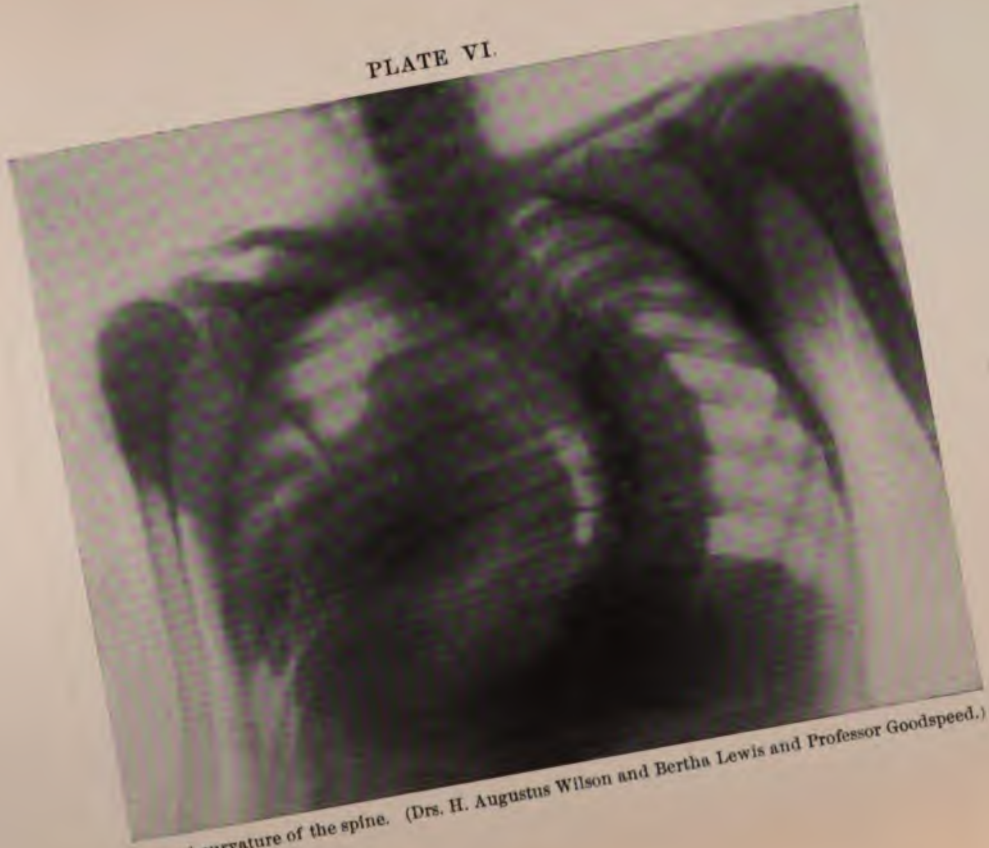
[5] Transactions of the American Surgical Association, 1896.

PLATE V.



Knock-knee. (Dr. De Forest Willard and Professor Goodspeed.)

PLATE VI.



Lateral curvature of the spine. (Drs. H. Augustus Wilson and Bertha Lewis and Professor Goodspeed.)

the bones. Plate V., from a patient of Dr. Willard, skiagraphed by Professor Goodspeed,[1] shows this very clearly. Plate VI. is an admirable illustration of a lateral curvature of the spine from a patient of Dr. H. Augustus Wilson and Dr. Bertha Lewis. This skiagraph by Professor Goodspeed is the first I have had taken of such a deformity. No better results would seem to be possible. Whether rotation has already taken place or not may be seen in a good skiagraph.

Diseases of Bone.—Mr. Noble Smith[2] has called attention to the importance of taking skiagraphs in cases of suspected caries of the spine, especially where the case is obscure, as so many of them are, particularly in the earlier stages. Should the skiagraph reveal the existence of caries, the proper treatment is indicated at once; but should it show that no caries is present, it not only frees the surgeon from anxiety upon that point, but is of especial value in freeing the patient from an irksome, long-continued, unnecessary treatment, with possible serious detriment to his health from the confinement. Plate VII., also from a patient of Drs. H. Augustus Wilson and Bertha Lewis, skiagraphed by Professor Goodspeed, shows excellently the carious disease in the upper dorsal vertebræ. Caries in other bones can also be discovered by this means and suitable early treatment be instituted.

Abscesses and other destructive lesions of the bone also can be diagnosticated by the greater transparency of the bone at the place where the osseous tissue has been destroyed. In cases of the so-called "Brodie's abscess" of bone, in which the symptoms often are obscure, especially in the earlier stages, the use of the *X*-rays will be of great value.

Bony growths, such as exostoses and true bony tumors, and especially osteosarcomata, in their earlier stages, can be diagnosticated and treated by amputation or otherwise as may be necessary.

Fractures and Dislocations.—One of the most useful applications of the *X*-rays is in determining the fact of the existence or non-existence of fractures or dislocations, in differentiating between the two, or in determining the fact of the existence of both. In injuries of the spine, from which, however, children are usually exempt, the *X*-rays will be of great use in determining the absence or existence of fracture or dislocation, or of both. Joynt[3] has called attention to a rare anatomical deformity,—the existence of a supra-condylar process on the humerus of a boy of twelve, "the pressure of which had caused a doubtful diagnosis of fracture to be added to that of dislocation of the elbow," which was cleared up by an *X*-ray skiagraph.

The method is particularly applicable to children. It will find here one of its largest fields of usefulness by reason of the fact that epiphyseal fractures are not uncommon in children, and that the surgeon often sees them late, when the swelling is so great as to obscure the diagnosis by manipulation, and thus leave him in doubt as to whether there is a dislocation or an epiphyseal fracture. The *X*-rays, especially if two or more pictures are taken in different planes, will enable us, as a rule, to determine the facts accurately and to institute the proper treatment. Plate VIII., by Dr. Goodspeed, from a patient of Dr. H. R. Wharton, shows such an epiphyseal fracture, which might readily be mistaken for a dislocation. At the elbow—a place of peculiar difficulty of diagnosis—the *X*-rays will be of especial value. It is fortunate, also, that not only bandages and cotton or other material used for padding, but even wooden (but not plaster of Paris) splints are perfectly permeable to the *X*-rays, so that a picture can be taken of a fracture or dislocation before, and again after, it has been reduced and dressed, and we can thus determine absolutely whether or not the bones are in perfect or almost perfect apposition if the case be one of fracture, or whether the dislocation has really been reduced if it is a case of the latter accident. Bryant[1] has described a case of a boy of eighteen in which the house surgeon made vain efforts to reduce a supposed dislocation of the lower end of the radius, but in which the *X*-rays showed that there was no dislocation.

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[1] Transactions of the American Surgical Association, 1896.

[2] British Medical Journal, 1896, vol. i. p. 1382.

[3] Ibid., 1897, vol. i. p. 980.

No accident, perhaps, is followed by as many suits for malpractice as fractures and dislocations; hence the value of taking skiagraphs which are permanent and indisputable records of the condition of the parts before and after the dressing. Several cases have already been reported in which the evidence of the *X*-rays has been brought into court, and it is probable that their use will be more rather than less frequent in the future. Some judges, however, have refused to admit them as evidence.

It is to be borne in mind, however, that up to the present time we have not sufficient data for absolute dogmatic opinions. It is probable that in not a few cases in which an excellent functional result is obtained the bones are not in an ideally perfect apposition. *A fracture may even exist without the X-rays showing the slightest evidence of it.* Plate IX, from a case of osteotomy, by Dr. William J. Taylor, skiagraphed through the dressings and a wooden splint the day after the operation, shows no evidence whatever of a fracture which was known to be absolutely complete and only one day old. Such a case should make us very careful in giving too positive testimony even from skiagraphs.

In *diseases of the joints*, such as tubercular disease, in which the joint surfaces have been more or less destroyed, a skiagraph may show the irregularities of the bony outline.[2] It must be remembered, however, that, since in young children the joint surfaces consist largely of cartilage, very little information may be obtained, since cartilage is almost as transparent to the *X*-rays as fat.

After resections—*e g.*, of the knee—it is often important to determine

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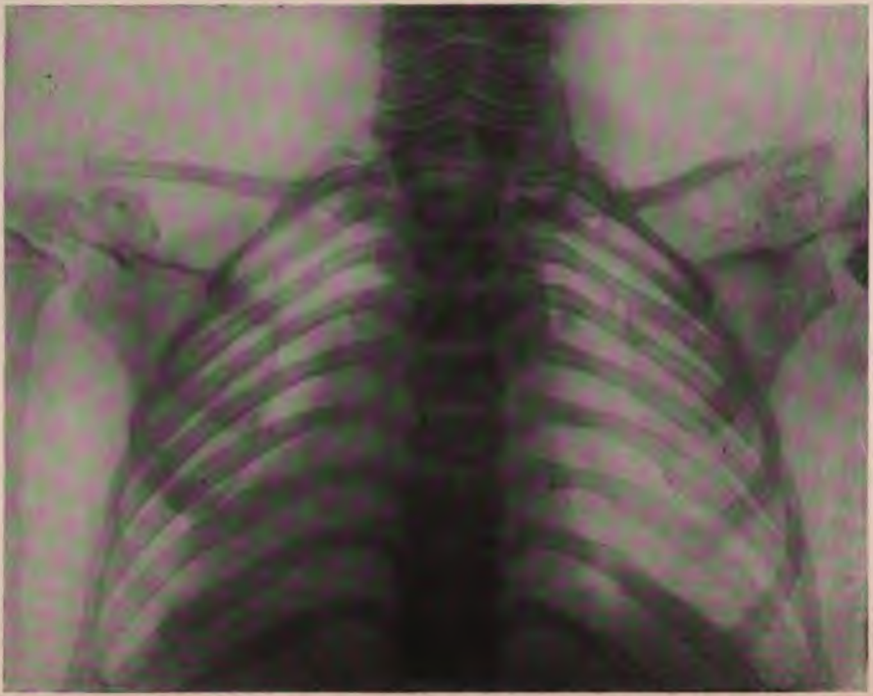
[1] New York Medical Record, April 24, 1897, p. 604.

[2] Willard, Transactions of the American Surgical Association, 1896.

PLATE VII.



Caries of the upper dorsal vertebral bodies. (Drs. H. Augustus Wilson and Bertha Lewis and Professor Goodspeed.)

when the operation has resulted in osseous union, and, therefore, when dressings or other supports may be cast aside, or whether, as at the elbow, or in cases of ankylosis, this has or has not taken place, and whether there is a possibility of establishing a movable joint. Willard[1] has shown the value of this method in determining the advisability or inadvisability of using forced movements of joints, and illustrated them with excellent skiagraphs.

(c) An equally useful application of the *X*-rays is in *the discovery of the presence or proof of the absence of foreign bodies.*

Foreign Bodies in the Tissues.—In the case of wounds by bullets, knives, needles, or glass, the *X*-rays will almost always determine their presence, or in other cases disprove their presence, in the tissues. Many a needle has been believed to be in the hand or the foot, and sought for fruitlessly, when the *X*-rays would have shown its absence. Pieces of glass, on the contrary, are not uncommonly overlooked and allowed to remain, when by the use of the *X*-rays we could determine at once their presence. The rules for the use of the apparatus and the need of pictures in two or more planes apply particularly in these instances. It should not be forgotten, especially in the case of needles, that if the interval between taking the skiagraph and doing the operation is of any great length, such a foreign body may have changed its place. The operation, therefore, if any is to be done, should follow the taking of the skiagraph at a very short interval.

In not a few cases the presence of the foreign body may entice the surgeon to do an operation which would be better left undone. Plate X., by Mr. H. B. Shallenberger, shows a fragment of a needle which had been harmless in the hand of a woman of twenty-four for twenty years. In such a case the temptation to operate should be strenuously resisted, as it is evident that the surgeon would do more harm than the needle.

In the eye this method is sometimes absolutely indispensable. As is pointed out by Lewkowitsch, the ophthalmoscope may give no help when the foreign body is very small, when it is embedded in the non-transparent tissues of the eyeball,—*e.g.*, the sclerotic coat,—when a large extravasation of blood obscures the view of the fundus of the eye, or when the cornea has become impermeable to light. Foreign bodies in the eyeball can be detected very certainly in a large number of these cases by means of the *X*-rays. Cases have been reported by de Schweinitz, Hansell, and others in which they have been found, and, having been found, their removal was easy.

In the brain, based upon earlier observations, the opinion was expressed by myself and others that the presence or absence of foreign bodies, especially of bullets, could not be determined by means of the *X*-rays, on account of the bony skull being an impenetrable obstacle. But Eulenberg[2] relates two cases, the one a young man of eighteen, the other an adult of thirty-three, in whose brains the bullets were thus successfully located; and Brissaud and Londe[1] report a third case in an adult in which the bullet was similarly located. Bunce[2] reports a fourth case in a boy of sixteen. Henschen and Lennander[3] have reported a fifth in a man of thirty-three, which both pathologically and surgically is of the greatest interest and importance. If this can be done in adults, in whom the skull is thick, it is very clear that through the thin skull of a child a bullet or other similar foreign body can be much more easily seen. Dr. Edward P. Davis[4] (Plate XI.) has shown by an excellent skiagraph the presence of two buckshot placed inside the skull of a child.

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[1] Transactions of the American Surgical Association, 1896.

[2] Deutsche medicinische Wochenschrift, August 17, 1896.

Foreign bodies are frequently introduced into the cavities of the body, especially the nose, by children, and the *X*-rays will enable us to detect them, provided they are opaque to these rays. Beans, peas, and other such vegetable substances cannot thus be determined, but glass beads or similar opaque substances can be easily skiagraphed. MacIntyre[5] has written an excellent article on the use of the *X*-rays in diseases of the nose, throat, and thorax. He has described two methods of taking pictures of the nose and throat. First he places in the mouth a screen in the form of a tongue-depressor, covered with a thin aluminum plate, as the fluorescent salts are poisonous. If it is desired to take a picture of the nose, upper jaw, and roof of the mouth, the screen is placed next the roof of the mouth, and the Crookes tube above and in front of the face. If, on the contrary, it be desired to get a picture of the larynx, tongue, and lower jaw, the screen is placed on the tongue and the focus-tube in front of the throat below the screen.

Substances that have been swallowed or that have passed into the Air-Passages.—Not uncommonly children are thought to have swallowed metal whistles, safety-pins, coins, jackstones, and other foreign bodies, but a great deal of doubt exists as to whether the foreign body has been absolutely swallowed or not. The *X*-rays are invaluable in these cases. White[6] and Wood[7] and myself[8] (Plate XII.) have all published cases of jackstones in the œsophagus which were located by the *X*-rays, and, in accordance with their position, were operated upon by different methods. In my own case, palpation did not give any evidence of the presence of the foreign body, nor did the laryngoscope show it. The jackstone lay opposite to the fourth and fifth cervical vertebræ, and was evidently more accessible by œsophagotomy than by any other method. In White's and Wood's cases the jackstone lay in the thoracic portion of the œsophagus,

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[1] Semaine Médicale, June 24, 1896.

[2] New York Medical Record, April 17, 1897, p. 536.

[3] Nordiskt medicin. Arkiv, 1897, No. 3.

[4] American Journal of the Medical Sciences, March, 1896.

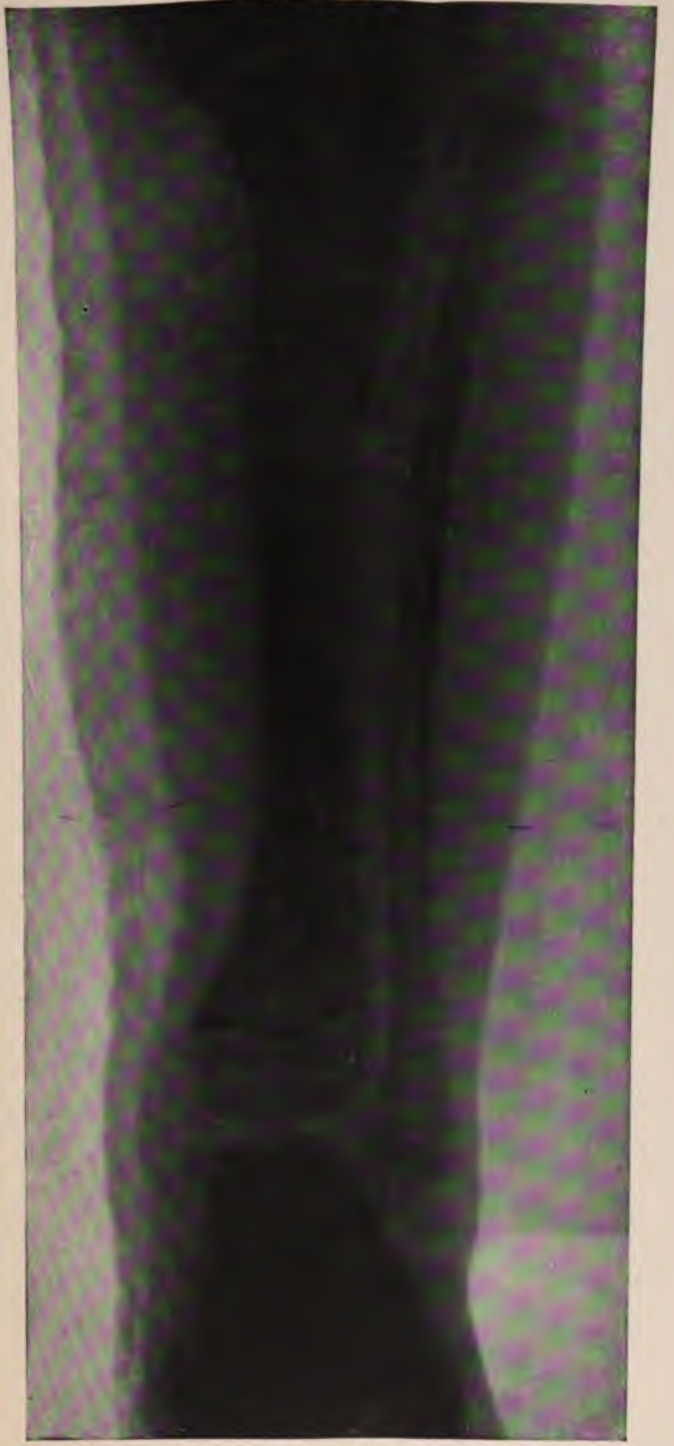
[5] Practitioner, January 18, 1897, vol. lviii. p. 42.

[6] University Medical Magazine, June, 1896, p. 710.

[7] Ibid., October, 1896, p. 29.

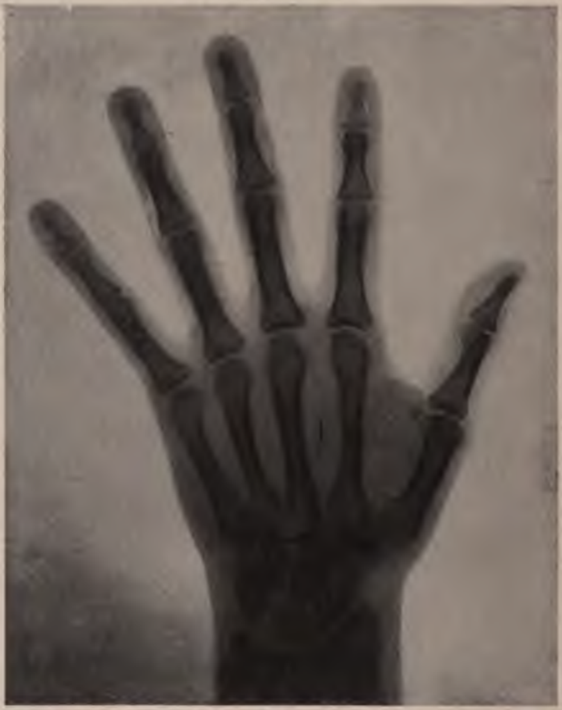
[8] Therapeutic Gazette, April 15, 1898, p. 233.

PLATE IX.



Osteotomy (fracture) of tibia and fibula. Though skiagraphed the day after the operation, no evidence of the fracture is seen. (Dr. W. J. Taylor and Professor Goodspeed.)

PLATE X.



Fragment of needle in the hand skiagraphed after twenty years. The eye of the needle is well shown. (Mr. H. B. Shallenberger.)

and, in the short neck of the child, was not accessible by œsophagotomy. In each of these cases a very ingenious method was adopted which proved successful. A gastrotomy was performed, and a string was then introduced through the œsophagus into the stomach by means of a small rubber probang, which passed between the spurs of the jackstone. Pieces of gauze of suitable size were then attached to the string, very much like the "bob" of a kite. In one case the jackstone was drawn down into the stomach, in the other upward into the mouth and easily removed.

Péan[1] and Rau[2] have similarly discovered and removed coins that had been swallowed. In Péan's case the œsophagus was exposed but not opened, and the coin gradually "coaxed" upward by pressure until it could be extracted through the mouth, thus avoiding the necessity and the dangers of œsophagotomy. In a recent case, in which a child of sixteen months had swallowed a twenty-five-cent piece, I was able to verify its presence in the right iliac fossa, and later to determine the fact that it had disappeared, though it had not been seen in the stools.

In case the foreign body has passed into the air-passages, its presence can be almost equally well ascertained. In children the ribs, vertebræ, and sternum, it is true, are bone, and may interfere to some extent with its location, but if the foreign substance is of metal, and therefore more opaque to these rays, the shadow cast by it will be so much deeper than the shadows cast by the bones that the foreign body can be readily located even through the bones.

Foreign Bodies formed within the Organism.—The most important of these are vesical calculi. Stone in the bladder cannot be as successfully skiagraphed as would be the case if it were not surrounded by the bony tissues of the pelvis. This obscures to some extent the shadow thrown by the stone. The most successful skiagraphs I have seen published are those by Laurie and Leon,[3] in the case of a boy of eight, and they state that they obtained equally good pictures in another case of a boy of fifteen. The original pictures are, perhaps, much better than those published in the journal. Recently Dr. A. H. Cordier has shown me admirably clear skiagraphs of stone in the bladder, far better than any previous one, taken by placing the plates in the vagina or rectum, the Crookes tube being placed above the pubes. The rectum could be utilized in children, but not the vagina. It is most likely that in a relatively short time the improvements in the methods which are following one another so rapidly will enable us to determine the presence or absence of stone in the bladder with much greater certainty than at present.

(*d*) *Diseases of the Viscera.*—As a rule, but few diseases of the viscera have as yet been skiagraphed with any satisfaction. Bouchard,[4] it is stated, has been able to show pleural effusion by means of the *X*-rays, and also pulmonary tuberculosis and pneumonia, especially in the stage of hepatization in adults. In children these diseases would probably show more distinctly than in adults. As yet, however, the field for the *X*-rays in internal medicine has been but little exploited. The future will probably enable us to improve upon the present methods and obtain much better results. The results obtained by Schott (cf. *supra*) point in this direction.

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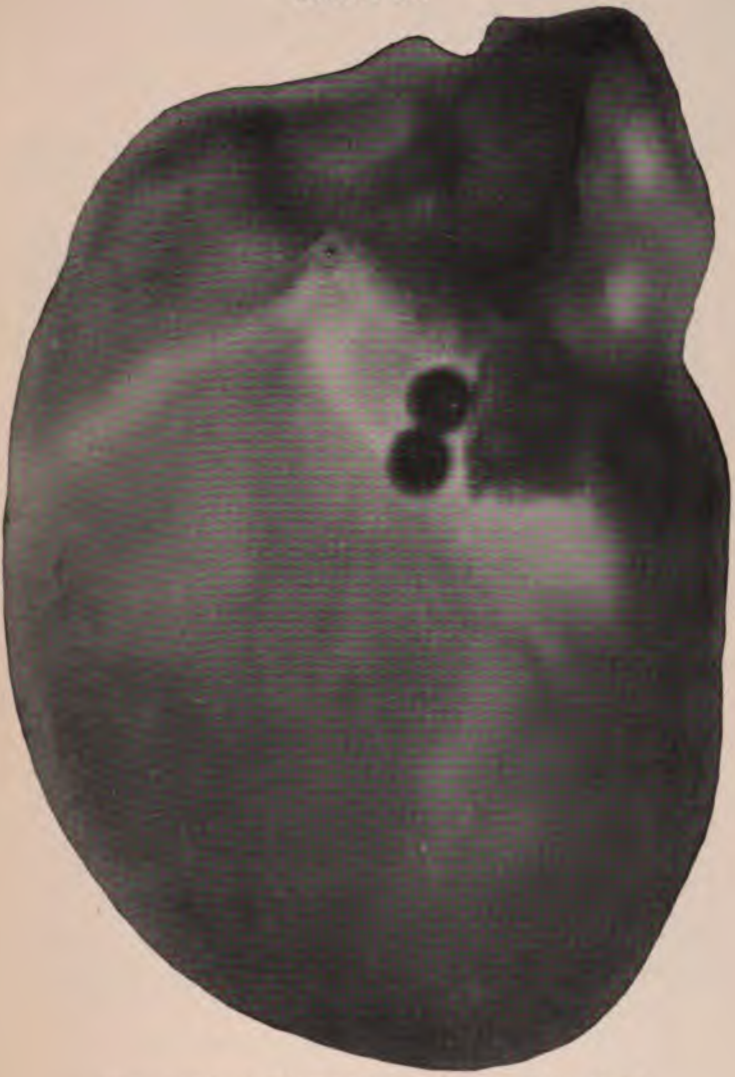
[1] Semaine Médicale, 1896, p. 494.

[2] British Medical Journal, 1896, vol. ii. p. 1768.

[3] Lancet, 1897, vol. i. p. 169.

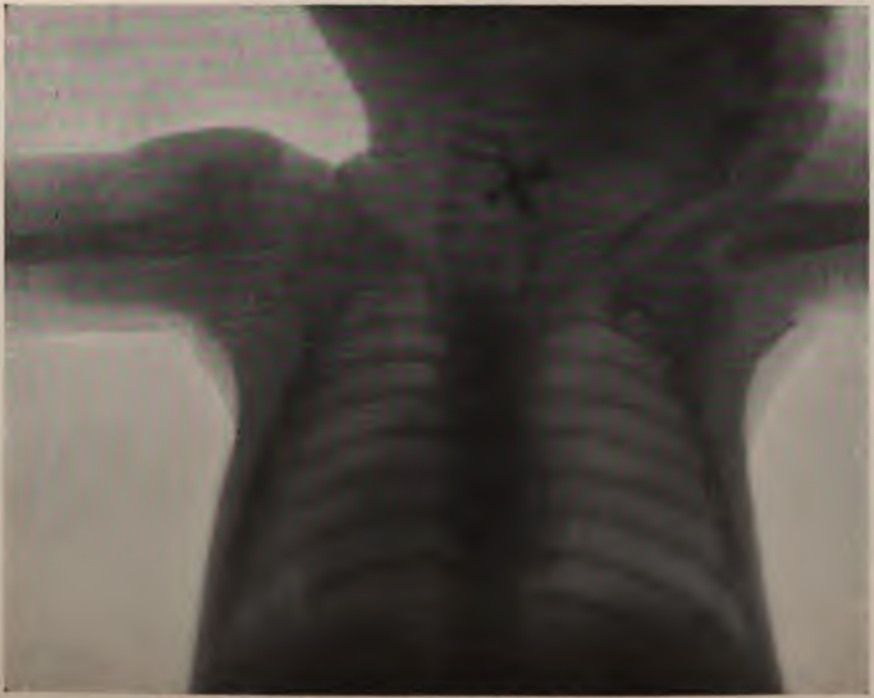
[4] New York Medical Record, January 29, 1897, p. 70.

PLATE XI.



Skiagraph of fetal skull in which two buckshot had been placed.
(Dr. E. P. Davis.)

PLATE XII.



Jackstone in the œsophagus (Drs. W. W. Keen and W. M. L. Coplin.)

PLASTIC SURGERY.

By THOMAS S. K. MORTON, M.D.

Plastic surgery (*plastikos*, formative) includes all procedures for the artificial formation of congenitally defective or absent parts, the substitution of healthy for unhealthy tissues, the restoration of function in parts disabled by disease or injury, and the retrenchment of abnormal hypertrophies.

The suffix "plasty" is generally employed in connection with the name of the part operated upon by reparative or formative measures: thus, uranoplasty, cheiloplasty, rhinoplasty, osteoplasty, neuroplasty, thoracoplasty.

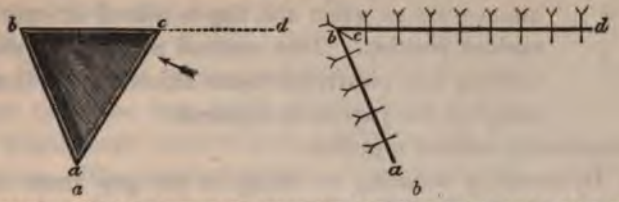
Autoplastic operations consist of restoration of defects with tissues taken from the same individual; when a second person or an animal supplies the material they are termed *heteroplastic*.

The *indications* for plastic operations upon children do not differ essentially from those governing such undertakings in adults, save that the parts usually dealt with in these procedures—namely, the skin and the subcutaneous tissues—do not in young persons bear handling, and especially stretching or other form of tension, nearly so well as in older individuals. Death of flaps from these latter causes is common even in the healthiest children. After the seventh year success is more uniform, and from the twelfth to the twentieth year probably the maximum resisting power is present.

The special indications for plastic operations may be divided into *cosmetic* and *functional*, although both frequently coexist in the same defect. In the former, operation will be purely a matter of election upon the part of the parent, but in the latter the surgeon will generally make the decision as to the necessity and advisability of interference.

As failure will almost necessarily involve more loss of tissue in very young than in older children, the question of selecting the proper *time for interfering* is a serious one. In general, it may be stated that the earlier in life that a successful operation can be performed the better will be the ultimate result. But success is more uniform as age increases and the structures employed have become larger and less delicate. Each case must be judged by its requirements and conditions. The at times unreasonable demand by parents that immediate operation be undertaken to remedy defects that are obnoxious to them in a child must be met by firm refusal by the surgeon if in his judgment it is not at that particular time to the

FIG. 3.



Incisions prolonged from one corner of a triangular wound: *a*, before, and *b*, after inserting the sutures. (Tillmans.)

The methods employed in plastic surgery are admirably summarized in the following table of J. B. Roberts,[1] somewhat modified.

METHODS USED IN SURGERY.

DISPLACEMENT.—Stretching or sliding of tissues.

- I. *Simple approximation after freshening of edges,* as in harelip, vesicovaginal fistula, and notches caused by tearing out of ear-rings.
- II. *Sliding into position after transferring tension to adjoining localities,* as in V-shaped incision for ectropion and cicatricial contraction of joints after burns, and in linear incisions to allow stretching of skin to cover large wounds and to relax contracted parts.

INTERPOLATION.—Borrowing material with a pedicle from adjacent regions, from another person, from an opposite limb, or from an animal.

- I. *Transferring a flap with a pedicle.*
  - *A.* Putting in place at once.
    - 1. By rotating flap on the pedicle in its own plane through one-fourth or one-half a circle, as in making upper eyelid or nose from forehead.
    - 2. By twisting flap on its pedicle, as in making side of nose from lip or cheek.
    - 3. By everting flap entirely so that the raw surface is uppermost, as in covering bladder-defects by a scrotal or an abdominal flap.
    - 4. Superimposing one flap upon another which has been everted, as where a thick wall is desired, as in closing the front of an exstrophy of the bladder or in building up a nose from brow- and cheek-flaps.
    - 5. By jumping, or carrying a flap across a bridge of skin and fixing only its end or side to the parts to be repaired. When the flap has become attached the pedicle is divided; or a distant part, as a hand or forearm, may be slipped under a strip of skin cut free except at one or both ends upon abdomen, chest, or thigh. This is probably impracticable in children.

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[1] Modern Surgery.

B. Putting in place gradually by successive migrations by same manœuvres as when the flap is placed at once in its permanent position. This method may prove valuable when nothing but cicatricial tissue exists in the immediate vicinity of the part to be repaired.

II. *Transplanting without a pedicle.*

a. By carefully suturing or fixing in the gap areas of tissue recently dissected from distant regions, or taken from an amputated or excised part, or from a second person or an animal, such as replacing bone-chips or the button after trephining, inserting portions of nerve-trunks, filling operative or other bone-defects with decalcified bone-fragments, and so on.

b. By skin-grafting with small pieces or large shavings of skin. This is the measure of this class that has been followed by greatest success and has eliminated many plastic measures from the surgery of children. As it limits cicatricial contraction, it may be used at times advantageously in plastic operations that necessarily leave surfaces to heal by granulation.

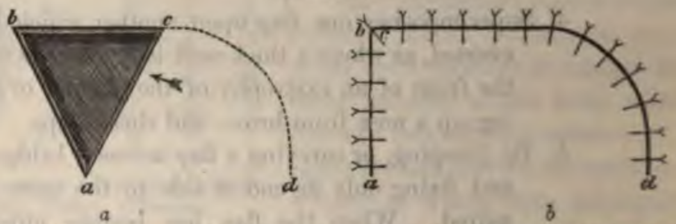
RETRENCHMENT.—Removal of superficial material and causing cicatricial contraction.

I. By cutting out elliptical or semi-elliptical pieces of tissue, as in ptosis, cystocele, and prolapse of the rectum.

II. By cutting out wedge-shaped or triangular portions of tissue, as in decreasing the size of a lip, an ear, or a nose, and in separating webbed fingers.

The operator should think out a definite plan of procedure, and the various steps should follow each other in an orderly sequence. It is well also to map out the outlines of flaps in nitrate of silver or other staining solution prior to making the first incision; frequently it will be found of advantage in intricate operations to experiment with paper patterns, subsequently to employ their dimensions and contour in laying out the of intended flaps. All flaps must be cut at least from one-sixth to one- larger than the space to be filled, so as to provide for immediate and

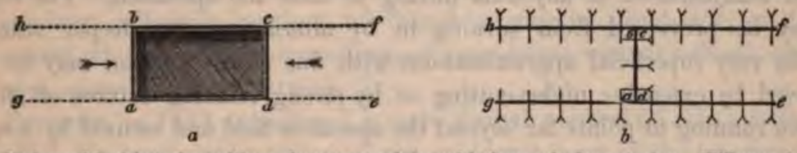
FIG. 4.



Curved incision from one corner of a triangular wound: *a*, before; *b*, after introduction of sutur
(Tillmans.)

quent shrinkage, which continues for weeks after union has taken place. Wounds from which skin has been derived, from a similar cause, become one-third larger. Flaps should have as broad and thick pedicles as possible. The base should be directed in the direction of the greatest arterial supply; but upon the face, where anastomosis is so free, this rule may be ignored. When large blood-vessels can be carried along with the flaps, it should be done. The whole thickness of the integument, including a little subcutaneous fat, should compose the flap; its edges should be as broad as possible. Hairy surfaces are to be avoided unless the transfer is being made to form an eyebrow or other naturally hairy region. The pedicle of flaps may often be cut in a curved direction so as to cause less tension when they are rotated or twisted.

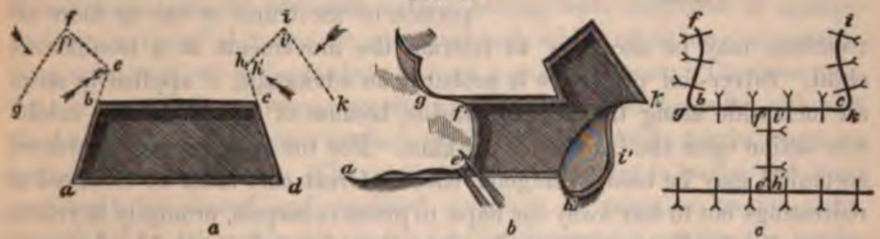
FIG. 5.



Prolonged incisions for uniting a four-cornered wound before and after suturing. (Tillmans.)

The surfaces to which tissues are to be transferred must always be prepared before flaps or grafts are cut. Cicatricial tissue is cut freely away, edges are freshened and broadened, and all hemorrhage is thoroughly controlled by sponge-pressure, torsion, or ligature. If ligatures are employed, which should be very seldom, the material should be catgut of small size. Hot water or chemical styptics must not be used to check bleeding, as they interfere with the subsequent nourishment of the graft from beneath. Placing a piece of rubber tissue or protective upon the prepared surface and then temporarily binding it up tightly with a pad of gauze on top is an effective and harmless method of stopping the troublesome ooze of blood that may float up and thus destroy grafts or flaps. If for any reason flaps are cut before the receiving surface is prepared, it is necessary that measures be taken to preserve their warmth and moisture. Masses of gauze wrung out of hot normal salt solution (six-tenths of one per cent.) are best for this purpose. If the parts are entirely severed, they should be immersed in the

FIG. 6.



Formation of two lateral flaps of skin. (Tillmans.)

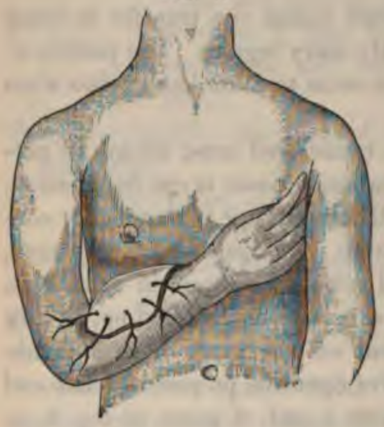
saline solution, of a temperature of 100° F., until wanted. The very sharpest instruments are required. All unnecessary handling of tissue is to be avoided, especially by forceps and scissors. Cutting by scissors unnecessarily bruises the edges of flaps; hæmostats should never be applied to a flap or upon the surface to which it is to be attached.

There must be no tension upon the wound-margins or upon the transplanted tissues. The edges are retained in contact by numerous sutures. These should, as a rule, be of very fine silkworm gut, tied gently in simply the first twist of a surgeon's knot. This permits of adjustment and readjustment at any time during or after the operation. The edges may be prevented from turning in by alternating the deeper stitches with very superficial approximations with fine silk. Tension may be relieved by extensive under-cutting or by strong relaxing sutures of silver wire running to points far beyond the operative field and secured by a split shot resting upon a metal plate. Or relaxing slings of catgut may be run from the deeper structures across beneath the flaps. Owing to local damage and secondary bleeding, the Esmarch bandage is not generally employed in plastic work.

The defects caused by removal of flaps are closed by simple suture, by a secondary plastic procedure, or may be at once or at a later time grafted by the Thiersch method (*vide infra*).

A sufficient and firm aseptic dressing is finally adjusted so as to insure drainage and complete rest of the parts involved. A starch or plaster of Paris roller over all, and perhaps including a portion of the trunk or one or more extremities, may be necessary to restrain the movements of a troublesome child. Silver-foil protective is probably an advantage, if applied in strips an inch wide along the wound-margins, because of its well-known inhibitory action upon the bacteria of the skin. For the same purpose powdered acetanilid may be used with good effect. Great care must be observed in redressings not to tear away the flaps, to preserve asepsis, promptly to relieve tension by dividing sutures, and to let out pockets of serum, blood, or pus should they form. Peroxide of hydrogen and weak antiseptics are best for irrigation.

FIG. 7.



Pediculated flap. Chest to forearm. (Tillmans.)

Not infrequently a series of operations is required to close large defects. Here as much as is thought prudent is accomplished at each sitting, and an interval of longer or shorter duration is permitted to elapse before again resorting to operation. This interval must be sufficient to permit thorough vascularization, shrinkage, and adaptation of the parts already in position.

At subsequent sittings great care must be taken to disturb as little as possible the work already done.

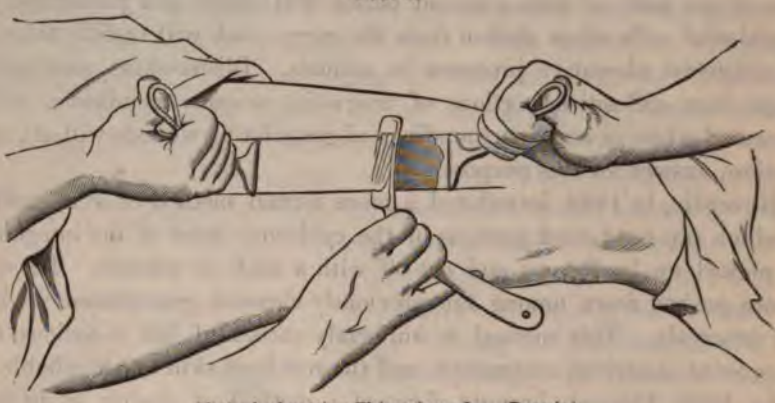
SKIN-GRAFTING.

Upon granulating surfaces that have been made clean by peroxide of hydrogen and subsequent flushing with normal salt solution (six-tenths of one per cent.) small portions of dried epithelium from neighboring or distant parts of the body or from a second person will adhere and proliferate, just as epithelial cells when shaken from the curry-comb will rapidly take root and obliterate ulcerative processes in animals. Blister-skins, shavings, or filings from callosities, portions of frog-skin, mucous membrane, or the macerated white or scaly surroundings of granulating wounds will all, upon occasion, answer for this purpose.

Reverdin, in 1869, introduced a more formal method of skin-grafting by which pin-head-sized portions of the epidermic layer of the integument are picked up by forceps and cut off with a knife or scissors. These are at once pushed down among the previously cleansed granulations at half-inch intervals. This method is uniformly successful, but is tedious, does not prevent cicatricial contraction, and the resultant skin is not reliable.

In 1886 Thiersch brought forward a method of closing, by primary union with true skin, even very extensive wound-areas by the transplantation of large strips of epidermis including a portion of the rete mucosum and connective tissue from the tips of the papillæ. This method has proved so successful both in children and in adults as to displace many of the plastic procedures involving flaps with pedicles. If the closure is made as a primary measure, the grafts are simply spread out upon the raw surfaces of the fresh wound in the manner to be described below. If the surface be a granulating one, however, it should first be prepared by thorough curettement, because if the granulations remain, a thin stratum of cicatricial tissue ultimately forms beneath the new skin and decreases its vitality. Hemorrhage from the prepared surfaces having been controlled by sponge-pressure, strips of skin are cut, about an inch wide and as long as possible, from some portion of the patient or from a second person. The outer portions of the thighs and arms are favorite sources for such grafts, although the chest, abdomen, or other surfaces are likewise available, but in many respects not so convenient. The parts whence grafts are to be derived must previously be shaved and rendered aseptic. A razor or section-knife, ground flat on the under side, is the best implement for cutting the grafts. The "safety" razor has also been strongly recommended for this purpose. It, as well as the part to supply the graft, is well lubricated with sterile olive oil. The skin should be put upon the stretch by a hand of the operator above and one of an assistant below the operative field. Some prefer to use skin-stretchers of special construction (Fig. 8). Sharp-pointed, broad retractors will often answer the purpose effectually. Then, holding the razor parallel with the skin-surface and making at the same time a little pressure downward and advancing it with a seesaw motion, the strips of skin are cut and heap themselves upon the blade, whence they may at once be transferred to the area to be grafted,—which is the method recommended,—or they may be temporarily stored in warm normal salt solution. The shavings should be about the thickness of tissue-paper. If fatty tissue is exposed, the graft has been cut much too thick, and is not likely to adhere or grow.

FIG. 8.



Method of cutting Thiersch grafts. (Dennis.)

The grafts, when enough have been cut, are very carefully laid upon the surface to receive them in strips from side to side, overlapping each other, as well as the wound-margins, at least an eighth of an inch in all directions. If this precaution is not taken, granulations are prone to spring up in the interspaces between the grafts and cause partial or complete destruction of the epithelial substance, and, in any case, much retard healing. Great care must be exercised to prevent curling under of the edges of the grafts, for only the raw surfaces will "take." When the whole wound has thus been covered in, it is gone over with a spatula to force out any drops of blood or air-bubbles that may separate the grafts from the base of the wound. Then pieces of Lister protective or gutta-percha tissue, oiled with sterile olive oil, are laid on so as to cover in the whole field. A large, evenly applied, firm dressing of gauze, saturated with salt solution, is applied over all, and the part placed at rest in moderate elevation. The dressings should be kept wet with salt solution until the first dressing is made, at the end of from two to four days, according to the amount of sanious ooze into the gauze. The subsequent dressings are to be made in a precisely similar manner, after douching with peroxide of hydrogen and salt solution, but need not be moist. No irritating antiseptics are employed until the grafts are firmly taken. When this has occurred the protective may be substituted by a boracic acid ointment. The epidermic layer usually comes away from the grafts about the fourth day, and may give rise to the erroneous impression that they have failed; but, in absence of motion, sepsis, and hemorrhage, failure is unusual and primary union the rule.

The procedure is particularly successful in children, although in them, as in older persons, the grafted region occasionally undergoes keloidal hypertrophy and contraction even where complete primary union has been secured.

The surfaces from which grafts have been derived should be dressed with a protective and gauze, or may simply be dusted thickly with formaldehyde-gelatin powder and permitted to scab over. These wounds, being exceedingly superficial, heal over in less than two weeks, and are ready to yield grafts again at the end of four additional weeks. Growth of hair is not interfered with, as the hair-bulbs lie beneath the razor-track.

Thiersch grafts after an interval of a few months are reddish and somewhat glistening in appearance; they are upon the same level as the surrounding skin, are freely movable upon their bed, and usually possess both tactile and thermal sensibility. After a longer interval they become natural in color, and can be lifted up in folds from the underlying tissues. As they possess no oil-glands, it is well to keep them anointed slightly with lanolin for an indefinite period, to prevent excoriation.

WOUNDS.

By ROBERT G. LE CONTE, M.D.

DURING the years that have elapsed since the previous article on Wounds appeared, so ably written by Dr. McCann, few changes have occurred. The description of wounds, their classification, symptoms, complications, treatment, etc., have changed but little. However, the recent researches on the pathology of the repair of wounds have materially changed our pre-existing views on this subject, and have also in some degree modified our treatment. To set forth these changes will be the object of this supplementary article.

HEALING OF WOUNDS.

It was at one time thought that an immediate union of wound-surfaces could take place without the interposition of granulation tissue,—that is, by the adhesion of the microscopical structures of the part without any reparative effort. It is now known that such a union is merely the temporary adhesion of fibres by means of fibrin, and is preliminary only to final union by the formation of new tissue.

About thirty years ago Virchow announced that the large number of new cells seen during the process of repair were formed by a proliferation of the pre-existing cells of the part. A little later Cohnheim advanced the theory that the leucocytes supplied all the material for the new tissue; but a better knowledge of the histology and the physiological action of cells has restored the view of Virchow, and the fixed cell of the tissue has regained its former prominent position. The part taken by the leucocyte is now reduced to the removal of necrosed or foreign tissue from the site of the wound, and also to supply, in part, food to the fixed tissue or regenerating cells.

The present theory is that the fixed tissue and parenchyma cells are the active agents in the work of regeneration, and that the wandering leucocytes are doomed to destruction either by absorption or by expulsion. Repair, then, is an active process in which the cells of the part are enabled to replace tissues which have been destroyed.

When new tissue is formed in the body, it is accomplished by a division and multiplication of cells. This cell-division takes place either directly or indirectly. Direct cell-division is a segmentation of the nucleus, followed by a division of the whole cell, during which process no increase in the amount and no change in the arrangement of the nuclear chromatin or threads are observed. In indirect cell-division or karyokinesis the nuclear threads and nucleoli become more distinct and increase in size. In some the chromatin assumes a granular appearance, in others it arranges itself in contorted filaments. Gradually the contour of the nucleus is lost and the nucleoli disappear. The chromatin is now seen arranging itself in separate crescentic or V-shaped filaments in the equatorial zone of the cell, with their angles converging towards the centre. At the same time delicate threads become visible at the poles of the nucleus, arranged in the shape of a spindle. The filaments in the centre of the cell now divide and arrange themselves in such a manner as to form two similar groups. These groups gradually separate, moving towards each pole, changing at the same time their formation, so that the angles of the filaments look towards the poles instead of towards the centre of the cell. As they move towards the poles, the groups assume more and more the appearance of the original chromatin net-work, and are gradually surrounded by a new nuclear membrane. During this last stage of karyokinesis the protoplasm of the cell commences to constrict at the equator. This becomes more and more pronounced, until, finally, it ends in complete separation and two individual cells have been formed. The above description is a type of the process of division, but variations in this may take place. The division of the nucleus may be multipolar instead of bipolar. In this way many nuclei are formed, and if the corresponding division of the protoplasm is delayed or does not take place, large giant cells are developed. The immediate result of this cell-division is the formation of a mass of variously shaped cells, called formative cells, embryonal cells, or plasma cells. Some of them strongly suggest epithelium in appearance, owing to the large round nucleus, which readily takes a stain; hence they are also called epithelioid cells; and, again, they are called fibroblasts, owing to their power of forming connective tissue. With these cells are found a greater or lesser number of leucocytes, and the whole tissue is called granulation or embryonic tissue. The formative cells enlarge and become spindle- or club-shaped, or develop one or more processes. These processes anastomose with one another, forming intricate interlacings. The fibrous portion of the scar is developed partly from the protoplasm of the cells and partly from a homogeneous intercellular substance produced by the cells. The fibrillation may begin in any part of the cell,—the ends, the sides, or in one or more of the processes,—and as it forms it fuses with the fibrils of adjoining cells. The nuclei, with some of the protoplasm, remain as the fixed connective tissue cells.

At the beginning of the reparative process the cells receive their nourishment from the plasma in the plasma canals, and also to some extent from the infiltrating leucocytes, but later new vessels are formed. The first step is an accumulation of granular protoplasm, which shows itself on the side of a capillary vessel. This gradually forms a solid, nucleated filament, which may be single or branched, and which fuses either directly with the same or with another capillary vessel or with a bud from a neighboring loop. These filaments are reinforced by the young connective tissue cells and shortly liquefy in the centre, forming a continuous lumen with the parent vessel. At first they transmit the liquid portions of the blood only, but soon they enlarge, are lined with an endothelial membrane, and perform all the functions of true capillaries. Most of these new vessels are later obliterated by the contractions of the fibrous tissue, as shown in the scar changing color from red to white.

When primary union of the skin and subcutaneous tissue is taking place, the edges of the wound are glued together for the first two or three days by the coagulated fibrin. During this time the granulation tissue is forming, and gradually pushes its way across the wound to join the opposite side. As the cells of this embryonic tissue return to a quiescent state, connective tissue or scar tissue remains to bind firmly together the wounded surfaces.

In healing by granulation or secondary union the process of repair is the same histologically as in primary union, and differs rather in the amount of new tissue to be formed than in the manner of forming it. In the so-called healing by organization of blood-clot, as in subcutaneous wounds, the coagulum serves simply as a scaffolding, and is at first infiltrated by leucocytes and then by the germinal cells. The latter subsist upon the leucocytes until the cell-mass becomes vascularized, when it is converted into the usual scar tissue.

When a wound becomes infected, the uniting coagulum or adhesive fibrin is destroyed. According to Nancrede, peptonizing ferments are formed which dissolve the intercellular cement and separate the new-formed cells from their source of nourishment, while the bacterial products directly attack the vitality of the cells. The infiltrated edges of the wound are attacked by the same agencies and more or less destroyed, and may form sloughs of varying size. The result is, the granulation cells increase far in excess of the needs for repair. Many of these cells die from lack of food, many are thrown off in the discharges, but far too many survive to form dense cicatricial scars. In the end, suppurating wounds heal in the same manner as in aseptic union, only the cicatricial tissue is much in excess.

From the previous remarks it is seen that the essential of repair is the return of the tissues at the seat of injury to their embryonic state, with the formation of new embryonic tissue, and that the new embryonic tissue, as it proceeds in its development, forms the permanent bond of union. Union cannot take place without this new tissue, but the amount of new material required will decrease proportionately with the perfection of apposition in the divided tissues. In children the tissues more readily return to the embryonic state, hence their wounds heal more quickly.

REPAIR OF SPECIAL TISSUES.

The formative cells of the different tissues are so alike in appearance during the early stage of their development that it is impossible to tell into what permanent tissue they will ultimately form; but it is probable that they only produce cells to which they are embryologically related. As connective tissue is so widely distributed through the body, almost all wounds will destroy some of this tissue through their violence; therefore, when regeneration takes place, connective tissue almost always takes some part in the process, if not the largest part.

Epithelium.—Repair of epithelial surfaces takes place more or less perfectly, provided the loss of tissue is not great and healing is prompt. In skin the epithelial cells at the border of the wound undergo the karyokinetic changes already described, and push their way out towards the centre of the wound, gradually covering all the granulating surface. If the denuded surface is large, the underlying fibrous tissue, by contracting, tends to bring the epithelial surfaces nearer together. Regeneration of the fibrous portion of the skin is complete, but the bundles are more irregular. Lymphatics, sebaceous and sweat glands, hair, and the true rete Malpighii are not reformed. The epithelium of the intestinal tract and other glandular epithelia, as in the liver or kidney, will regenerate almost completely under favorable conditions, but when there is a large loss of substance in these organs, or the wounded surface, through irritation, heals slowly, cicatricial tissue will be developed.

Muscle.—Regeneration of muscle-fibres takes place only in a very limited degree in man, and such wounds heal by scar tissue, forming a tendinous union. However, in slight injuries some muscular tissue will develop. The living muscle-cells proliferate at the ends and sides of the fibres at the seat of injury, forming club-shaped or fusiform bundles called muscle-buds. These split up into a number of new fibres, which interlace and gradually increase in bulk, and finally join together the ends of the divided muscle. Many of the fibres fail to develop, and disappear by fatty degeneration. Transverse striation appears about the third week.

Tendons and Fascia.—In these tissues regeneration is practically complete. When a tendon is cut the proximal end retracts in its sheath and the space left is generally filled with blood-clot. At the end of two or three days the cells of the sheath rapidly proliferate and push their way into the clot. The cut ends of the tendon at first do not appear to take any part, but later they proliferate to a slight extent and aid in the formation of the new granulation tissue. The exudate extends some distance above and below the divided extremities. By the fifth or sixth day a spindle-shaped mass fills the gap, the cells arranging themselves mainly in a direction parallel to the long axis of the tendon. As repair proceeds the cells diminish in number and form fibrous tissue, and the clot and excess of material are absorbed. The new tissue cannot be differentiated from normal tendon, except that the sheath is adherent at the point of section.

Cartilage and Bone.—The regenerative power of cartilage is very small indeed, and loss of substance in it is generally repaired by the interposition of fibrous tissue. In bone, however, we have a true regeneration, which at times is so perfect that it is difficult to detect the original seat of fracture. In fracture of a bone a considerable amount of blood will flow from the ruptured medullary and Haversian vessels, as well as from the periosteum and lacerated soft parts in the neighborhood. This coagulates, and is soon infiltrated with leucocytes, the whole mass extending well above and below the seat of fracture. The deeper cells of the periosteum and medullary tissues and also the connective tissue cells proliferate freely and invade the blood-clot, and the ends of the broken bone are embedded in a fusiform mass of firm cellular exudate. This provisional callus is divided into the external callus, formed from the periosteum, the internal callus, formed from the medullary sheath, and the intermediate callus, probably formed from both of these sources, and lying directly beneath the end of the bone. It is the latter which is finally developed into the permanent bone tissue. The osteoblasts derived from the above cells are now seen embedded in a finely striated intercellular substance, surrounded by a halo, presenting the appearance of cartilage cells. As they develop, their protoplasm is changed into fine fibrillæ which interlace and form trabeculæ. A deposit of lime salts now takes place, beginning generally at the angles formed between the separated periosteum and the bone, which gradually extends through the mass of provisional callus. This newly ossified callus consists of a coarse-meshed, spongy bone, containing a vascular granulation tissue surrounded by a row of cells. It is evident that these cells are actively concerned in the formation of new bone, as the deposition of lime salts is seen taking place between the cells. (Warren.) In the medullary canal the granulation tissue is changed into embryonic marrow, and ossification starts in the part nearest the cortical layer of the bone and proceeds until the whole cavity is filled with the same porous bony tissue. Gradually the ends of the broken bone soften and become more porous and the vascular spaces enlarge. We now have a mass of spongy bone uniting and shading into the original bony structure. When union has taken place the external and the internal callus undergo absorption, beginning first in the medullary spaces. In these spaces are found numerous giant cells called osteoclasts, which are said to form carbon dioxide and thus dissolve the lime salts. As the provisional callus is removed the permanent callus becomes more dense, until finally it assumes the appearance of normal cortical bone. When overlapping occurs, the open ends of the medullary canal are closed by bone, the cortical portions which are in contact soften and are changed into cancellous bone, and gradually a more or less complete medullary canal is restored.

In open fractures or in necrosis, when the sequestrum has been removed, healing takes place by granulation (second intention) or by organization of blood-clot, but the process histologically is essentially the same.

Arteries.—When a ligature is placed around a large artery in continuity, the intima and a portion of the media are ruptured, the adventitia is gathered into a dense sheath about the constriction, and the blood-current is permanently stopped. Thrombi are formed in the lumen of the vessel, which vary considerably in size, but the distal thrombus is usually smaller than the proximal. In aseptic operations they are frequently extremely small, some writers claiming that they may be entirely absent; but Warren, in all cases examined, has never failed to find a thrombus. His description of the process of repair is as follows. Granulation tissue forms for some distance above and below the point of ligature on the outside of the vessel; presently the adventitia is invaded by leucocytes about the seat of the ligature, the infiltration reaching sufficiently far to penetrate the thrombi. The bundles of fibres surrounded by the ligature gradually soften and disintegrate, and the two ends of the vessel separate from each other, leaving the knot embedded in the centre of the callus. The ends of the vessel now expand and the granulations penetrate freely into the thrombi, accompanied by new blood-vessels which spring from the vessels surrounding the ligature. These granulations form irregular masses with spaces between them, which become blood-spaces communicating with the lumen of the vessel as the clot is absorbed. These spaces later communicate with capillaries in the granulation tissue. As the provisional structures are gradually absorbed, it is seen that a growth has taken place from the intima, many of the wandering cells found in the clot coming from this layer. With the disappearance of the internal callus a permanent cicatrix is formed, closing the ends of the vessel. This cicatrix varies in shape according to the presence or absence of large arterial branches. It is usually crescent-shaped, the horns projecting symmetrically along the inner walls of the vessel. If the branch is given off on the side, one horn will reach only to the point of junction, while the other on the opposite side extends much farther into the vessel, narrowing its lumen and allowing it to taper gradually towards the mouth of the collateral branch. The permanent cicatrix, when fully formed (from two to six months), consists of a reproduction of the three walls of the vessel: the inner layer, endothelium, formed from the intima; the middle layer, muscular, formed from the cells of the media; and the outside layer, fibrous, formed from the adventitia. A fibrous cord unites the two ends of the vessel. The new vessels have also disappeared, and usually a small central vessel is seen penetrating the cicatrix from the lumen and anastomosing with the capillaries surrounding the end of the arterial stump.

After ligature of an artery in an amputation the process of repair is the same, but the lumen of the artery is greatly diminished by contraction, and also by the cicatricial tissue extending a long distance into the interior of the vessel, sometimes even through its whole length. The collateral branches increase in size, and in the end we have a small vessel which terminates in a number of branches distributed in various directions. In small wounds of an artery a thrombus may form, extending through the gap in the wall and into the interior of the vessel, but not entirely occluding the lumen. Granulation tissue springs up and invades the portion of the thrombus between the wounded surfaces, which in turn is replaced by a connective tissue scar. The pressure of the blood-current may cause this scar to yield gradually, and then an aneurism is formed.

Nerves.—Regeneration of the fibres of the central nervous system does not seem to occur, or, if it does, only in the very slightest degree. Wounds of the brain are only partially closed by the development of new tissue, and it is probable that this is due to the proliferation of the cells of the neuroglia. While karyokinetic changes have been seen by some observers to take place in the nuclei of the ganglia cells, the process seems to be arrested, and does not go on to the complete formation of new cells.

Repair of the spinal nerves, however, is complete under favorable circumstances. The new nerve-tissue is reproduced from the pre-existing nerve-tissue, and not from the connective tissue structures which form part of the nerve-trunk. When a nerve is cut generally the whole of the distal portion and part of the proximal degenerates, and the new embryonic nerve-fibrils spring chiefly from the axis-cylinders of the proximal end. Howel and Huber, Warren, and others describe the process as follows. The space between the divided ends of the nerve fills up with granulation tissue. On the fourth day the myelin sheath breaks up into a number of segments, accompanied by a breaking of the axis-cylinders. By the seventh day an active proliferation of the nuclei of the neurilemma is seen. These nuclei migrate, and several are frequently found in one internodal space. By the fourteenth day all the myelin has been absorbed, together with the fragments of the axis-cylinders. Protoplasm now begins to accumulate around the new nuclei until a continuous fibre is formed within the old sheath. It is supposed that a new sheath is formed from the peripheral layers of these protoplasmic fibres, while the old sheath becomes part of the endoneural connective tissue. These fibres eventually change into complete nerve-fibres, having myelin sheaths and axis-cylinders, but experiment has shown that while in the embryonic state they are capable of transmitting nerve-impulses. In the dog, return of function begins at the end of three weeks and is complete in three months. The best results are obtained in immediate nerve-suture, as regeneration takes place more rapidly, although complete degeneration of the peripheral end apparently takes place. Aseptic union also hastens recovery, owing to the small amount of granulation tissue present, while suppuration, by increasing the amount of granulation tissue, makes it more difficult for the embryonic nerve-fibres to penetrate through it. Again, the length of time for recovery will increase proportionately with the amount of nerve-tissue destroyed and the separation of the ends. If the separation of the ends is too great to be bridged over by the new nerve, regeneration will cease at the embryonic stage, and a bulbous enlargement will form at the end of the nerve, composed chiefly of fibrous tissue. The longer the distal portion of the nerve the more time will be required for a cure. Clinical experience would seem to teach that immediate union of a nerve with restoration of function is possible, but pathology has proved this impossible, owing to the degeneration which always takes place of the distal portion of the nerve. In such cases the apparent return of motion or sensation is explained either by the anastomosis of the peripheral branches of the divided nerve with other nerves which have not been cut or by the occurrence of the so-called supplementary or vicarious sensibility.

TREATMENT OF WOUNDS.[1]

When wounded surfaces are free from germs, regeneration of the tissues will take place quickly with the least development of scar tissue. It is also true that healthy tissues will destroy or remove a certain number of pathogenic organisms, and recovery will take place without disturbance or sign of inflammation. The more vascular tissues possess this immunity in the greatest degree. Again, the scarcity or absence of a suitable food for the germs will so inhibit their growth as to render them almost incapable of mischief. Therefore, to enable germs to produce their effect, they must either be present in a wound in large numbers or, the vitality and resistance of the tissues being lowered, a small number may rapidly multiply into an overwhelming force, or, an abundance of suitable food being present, separating the germs from the resistant action of the tissues, they may flourish and increase to enormous numbers. With these facts in view, wound-treatment will be directed (1) against the admission of germs to the site of a wound, (2) to keep the vitality of the tissues at the highest point attainable, avoiding all unnecessary damage or manipulation, (3) to the removal of all fluids and material that can serve as a germ food.

Whatever may be the nature of the wound, whether it is incised, contused, lacerated, or punctured, whether it is surgically clean or infected, our first care will be to prevent primary or further infection. A few years ago antiseptics were relied upon to a large extent to accomplish this purpose, but the increasing knowledge of the action of these drugs, and of how readily they are decomposed and rendered inert in the presence of wound-fluids and living tissues, has curtailed their use very materially. As far as possible, all articles that are to come in contact with or near a wound, as instruments, ligatures, sponges, dressings, or towels, are sterilized with heat, either by boiling or by steam under pressure, while antiseptics are used for the hands of the operator and his assistants and for the disinfection of the skin adjacent to the wound.

The most important factor in wound-treatment is the avoidance of contamination by direct contact, so it will not be amiss to run over the different procedures of an aseptic technique.

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[1] It is almost impossible in a short article to give a detailed view of the many modern theories of wound-treatment; therefore the more important points only will be considered here.

Disinfection of Hands.—The nails are first carefully cleaned and cut; next the hands are thoroughly scrubbed with soap, warm water, and a stiff nail-brush—the water being repeatedly changed—for five or preferably ten minutes. They are then rinsed in cold water, washed in alcohol to remove the remains of the soap and fatty materials of the skin, and plunged in a 1 to 1000 bichloride solution for three or four minutes. The mercury is removed by rinsing in sterile water. If the hands have been recently contaminated with pus, fecal discharges, or any septic material, it is best, after the soap and water scrubbing, to rub them well with ground mustard and water for a couple of minutes, and then proceed with the alcohol and bichloride solution.

Disinfection of the Field of Operation.—Whenever possible, the child is given a warm bath previous to operation. The seat of operation is carefully shaved, not so much to remove the hairs as the superficial layers of the epidermis. The skin is now gently scrubbed with warm water, soap, and a brush, especial attention being given to all creases and folds. The soap is washed away with clean water, and the part rubbed with ether, alcohol, or turpentine to remove the fats. Next a moist antiseptic dressing (1 to 2000 bichloride) is applied, and this is to be removed only on the operating-table. Here, if the provisional dressing has been on for twenty-four hours, it is well to rub the skin again with alcohol or ether, to remove the products that have been secreted overnight, and then douche with a mercuric solution. In young children with delicate skins it is best to dilute the bichloride solution to 1 to 4000 or 1 to 5000, lest a dermatitis be started, and for the same reason a soft-soap poultice, which softens dirt so admirably in a tough skin, is to be used with caution only.

Sterilization of Instruments.—All instruments must first be thoroughly cleaned with soap, water, and a brush. Then they are sterilized by boiling for five minutes in a one per cent. soda solution, after which they may be placed in trays and covered with sterile water or simply laid on a sterile towel. The soda in the solution prevents rusting. As the sharp edge of a knife is dulled by prolonged boiling, it is best to wipe its surface with alcohol, and not drop it in the pan with the other instruments until the last minute of boiling.

Sterilization of Dressings, Gauze-Sponges, Bandages, and Towels.—Dressings are usually made of loosely woven cheese-cloth. They should be cut to the required size and shape, loosely folded, and covered with a piece of muslin. The sponges are made of several thicknesses of the same material, the frayed edges turned in and basted together with cotton thread; or some may prefer little balls of cotton covered with gauze. Absorbent cotton is torn or cut into various-sized pieces and wrapped in a piece of muslin. Towels, if new, are to be boiled in a five per cent. soda solution for fifteen minutes to remove fatty materials, then rinsed in cold water and dried. They are also loosely folded and covered with a towel or piece of muslin. All these materials are sterilized with steam under pressure for an hour. Care must be taken that the different materials are not tightly wrapped or folded, as the steam must have ready access to all parts of the dressings. After sterilization they are cooled for a few minutes in the air and stored in sterile glass jars.

Sterilization of Sutures and Ligatures.—Silver wire, silkworm gut, silk, and horse-hair should be boiled in soda solution with the instruments just before operation. Silk, which is so frequently covered with germs, should be previously sterilized with the dressings in steam for an hour, and silkworm gut, owing to its method of manufacture, is safer when it has been soaked overnight in a 1 to 1000 mercuric solution. Catgut is rendered safe by fractional sterilization. The gut is soaked in oil of juniper for a week, then in ether for two days, and finally in alcohol. While in the alcohol it is boiled for half an hour on three successive days. It is then kept in alcohol until used. Water rots the gut, and alcoholic solutions of antiseptics are so feeble as to be valueless for disinfection purposes.

Disinfection of Sea-Sponges.—These, as they are bought in commerce, are first pounded with a wooden paddle until quite soft, to remove the sand and bits of coral, and washed in cold water. They are then soaked for twenty-four hours in a three per cent. hydrochloric acid solution, to remove any finer particles of sand that may have been left, and again washed in water. Next, to remove any fat or albuminous material, they are placed for twenty-four hours in a three per cent. carbonate of sodium solution, and then washed in water until entirely free from the soapy feeling. To bleach them they are placed for a day in a seven per cent. sulphurous acid solution, after which they are again washed in water and stored in alcohol or in a three per cent. carbolic solution. Previous to operation they are squeezed dry and rinsed in sterile water, when they are ready for use. Heat spoils the elasticity of a sponge, and mercury generally stains it black.

Glass and rubber drainage-tubes are prepared in the same manner as the instruments. The large dishes, trays, and bowls used in an operation can be sterilized by boiling, but, as they are so cumbersome, it is best to disinfect them mechanically with soap, hot water, and a brush, and then fill them to the brim with a 1 to 500 bichloride solution. Let them stand for an hour and then rinse out with sterile water. Water is rendered sterile simply by boiling, but when it is to come in contact with wounds one per cent. of sodium chloride is added to render it more bland to the tissues.

With some system of disinfection and sterilization like the above mentioned the dangers of infecting a wound by contact are reduced to a minimum, but the price to be paid is constant vigilance and absolute attention to detail. If your hand, a ligature, an instrument, touch for a second an unsterilized object, and is then brought in contact with a wound, the most elaborate aseptic technique is absolutely destroyed, and it is a matter of pure luck if a sterile wound results.

To maintain the Vitality of the Tissues.—All chemical antiseptics are irritating to wound-surfaces, and generally the more powerful the antiseptic the greater the irritation. A three per cent. carbolic or a 1 to 1000 bichloride solution will produce cell-necrosis with the formation of minute sloughs,—*i.e.*, one of the best foods for pathogenic germs. If the solution is not strong enough to produce this cauterizing effect, it will at least irritate the parts and lower the vitality of the tissues. There is no doubt that antiseptics will completely inhibit or absolutely destroy germs when they are grown in the laboratory on prepared foods, but the conditions that are present in living tissues are absolutely different. Here the solutions used do not reach the germs in a sufficiently concentrated form to kill them, and those germs that lie in the deeper tissues may not be reached at all. The tissues soon decompose, precipitate and dilute the antiseptic, so that its inhibitory power is destroyed, and then the bacteria remain free to propagate, with minute sloughs for food, in the presence of a tissue whose resisting power has been lowered. Hence antiseptic irrigations are giving way in favor of sterile salt solution, as the latter will flush the germs out mechanically as well as an antiseptic, and being bland to the tissues, will not lower their vitality. Some surgeons go so far as not to allow a drop of fluid to come in contact with an operative wound, preferring to wipe the tissues free from fluids and blood-clot with dry sterile gauze. Even in fresh wounds which have been contaminated by various kinds of dirt antiseptic irrigations may be dispensed with, and the wound cleansed mechanically by cutting away the severely contused and lacerated tissue, scraping with a curette, and wiping dry with sterile gauze or flushing with a sterile solution. Infectious material that cannot be removed in this manner is not likely to be destroyed by antiseptic irrigations. For uninfected wounds dressings containing antiseptics have been replaced by sterile gauze. A sublimate gauze is not only dangerous on account of its frequently irritating a delicate skin, but also, on account of its method of preparation, it is often contaminated with germs. The unavoidable handling necessitated in the course of its drying, cutting, folding, and storing makes it extremely unreliable, especially as the sterilization of the material is the first step in the process and not the last. Again, the idea that antiseptics must be present in a dressing to keep the discharges sweet and clean has been disproved by Neuber, who demonstrated that the dryness of a dressing was its best safeguard from infection. He showed that a sterile dressing saturated with the discharges of a fresh wound would remain sweet for an indefinite period if rapid evaporation were allowed to take place. In this way a wound is practically converted into a subcutaneous injury, the dried dressing acting as a scab. For the same reason the various drying and antiseptic powders, of which iodoform is a type, are no longer necessary, as they not only have a tendency to cake about the wound and prevent the discharges from being absorbed by the dressing, but also are occasionally the carriers of germs, unless they have been carefully disinfected just previous to use. For suppurating wounds, where the secretions are thick and often contain masses of decomposing material, a dry dressing is not advisable, as the evaporation favors a crusting of the discharges, and as a consequence a penning in of the irritating material. Here a moist dressing is best, as it more readily absorbs the thick discharges. The parts may be lightly dusted with iodoform, and sterile gauze wrung out in an antiseptic solution or in sterile water applied, the whole being covered with waxed paper to prevent evaporation. Such a dressing should be frequently changed. In wounds communicating with the digestive tract, as of the mouth, anus, or rectum, iodoform is very useful in preventing the septic processes which are likely to follow.

The question of drainage depends largely upon the judgment of the surgeon, as no hard and fast rules can be laid down. In aseptic wounds, where the discharge is bloody serum, no drainage material will be required, even though the wound is large and deep. If complete approximation of all the wounded tissues cannot be made, and some dead spaces remain, it is generally sufficient to leave one or both of the angles of the wound slightly patulous, or, by placing the sutures farther apart, leave spaces from which the serous ooze can escape. Should the surgeon, however, believe drainage necessary, a strip of gauze or a small rubber tube is the best material to use. The practice of laying in a wound strands of catgut for a drain is distinctly bad. Catgut has very small capillary powers, besides which it swells greatly in the presence of moisture, frequently forming a plug, preventing the escape of serum instead of aiding it. Healthy tissues will readily reabsorb a quantity of serous discharge. In infected wounds, and also in those in which the vitality of the tissues has been much lowered by bruising and tearing, free drainage will be required. The free evacuation of pus is the best safeguard against septicæmia. Here rubber or glass tubes must be used, as gauze strips will not drain pus well. In large lacerated and contused wounds drainage is used to prevent distention and pressure on tissues whose spark of life needs every encouragement.

In conclusion, we may say that the danger of sepsis is proportionately increased with the number of articles brought in contact with a wound. If one assistant is sufficient for an operation, why have two or three? as every pair of hands, no matter how carefully prepared, is an additional menace. If a few simple instruments will suffice, do not have a number of complicated ones, as complicated machinery is difficult to clean. If a simple sterile gauze dressing answers every purpose, do not add as an extra precaution drying and antiseptic powders or silver leaf or any other material, as you defeat your object. At best, such materials are useless on a sterile wound, and they may be a dangerous source of infection.

ANÆSTHETICS AND ANÆSTHESIA.

BY GWILYM G. DAVIS, M.D., M.R.C.S. (ENG.).

THE choice of anæsthetics for the production of general anæsthesia limits itself practically to ether and chloroform. While many other agents have been used to a greater or less extent, they have not succeeded in displacing or in showing themselves more desirable than those named. The rivalry of these two anæsthetics still continues, but, apparently, ether has established itself more firmly in the estimation of the profession, while in many cases the use of chloroform has been abandoned. This is particularly the case on the Continent. The relative mortality of chloroform to ether, as given from a very large number of statistics collected by Dr. George M. Gould, is nearly five to one,—that is, chloroform is five times more dangerous than ether. Sansom[1] states that "The mortality from chloroform administration does not diminish." Public opinion in most parts of America is decidedly against the use of chloroform, and the loss of a patient from it is apt to do the physician considerable professional harm.

The advantages possessed by chloroform, to my mind, fail to overcome these objections except in rare instances. Surgeons are apt to prefer and use those agents to which they are most accustomed. In his article on this subject in vol. iii., Dr. Allis commends chloroform, but he does so solely on the condition that it shall be properly administered. This is doubtless the position taken by most of those who use it, but it has been in use now fifty years, and if it is true, as no doubt it is, as Sansom says, that deaths from chloroform continue as numerous as ever, it shows that as yet we have no means of safely administering it, and any one who examines the subject will be convinced that the search for a safe method has been exceedingly diligent. That care in administration will avoid some fatalities is undoubtedly true, but it is equally true that, in spite of the most careful administration, deaths are liable to occur. The drug in its very essence possesses a deadly quality that is apt at any moment to show itself in spite of the utmost care and watchfulness.

Chloroform has been claimed to be peculiarly suitable for children, both on account of its supposed greater safety in the young and on account of its pleasantness and ease of administration. That it is slightly safer in children than in adults is possibly true, but only because children, from a physical stand-point, are apt to be healthier than adults. Damaged kidneys and weakened hearts and depressed vitality generally are less common in children. The recuperative powers of childhood surpass those of the adult. This advantage, however, is not sufficient to justify the selection of the more dangerous drug, for it does not exist to anything like the extent popularly supposed. Dr. Allis says, "The supposed immunity enjoyed by children is not borne out by statistics or clinical practice. Deaths have occurred in early life, and would be more frequent were operations as common in childhood as in adult life." Woodhouse Braine[1] says that, while it has been advised not to give ether under five or over sixty years of age, such has been contrary to his experience. George H. Bailey[2] says he would sooner use ether for the extremes of life, and that "most certainly the young die under chloroform, and the stimulation is good for the old." Rudolph Kundrat,[3] out of twenty deaths from chloroform, gives eight as being fifteen years of age or younger. This would seem to indicate that the young are more likely to be killed than adults. Kuykendall[4] gives a typical case of death in a boy aged six years, the heart ceasing before the respiration. Children are more susceptible to the action of anæsthetics, as well as other agents, than are adults. They come more readily under their influence and recover more quickly from their effects.

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[1] British Medical Journal, 1894, vol. ii. p. 1397.

It being, then, admitted that the supposed immunity in childhood is not enough to justify the use of chloroform, let us see what other reasons there may be for its preference. The unpleasant odor and pungency of ether constitute a serious objection to its use in childhood. Children almost universally object to the administration of ether. Its pungency at once arouses their opposition. Ordinarily this is not a serious thing. They come under its effects so soon that the period of struggling is short. In some cases, however, particularly in private practice, it is desirable to avoid arousing the opposition of a child, and then it is necessary to use chloroform. When such is the case, it is my practice to begin the anæsthesia with chloroform and, as soon as the child is under its influence, change to ether. The danger is thus reduced to a minimum.

In affections of the respiratory passages chloroform is preferable. Thus, in tracheotomy, if any anæsthetic at all is to be given, chloroform is probably the better: ether, even in healthy subjects, produces an abundant flow of tenacious mucus. In healthy children, however, this is not liable to produce any bad effects on the lungs. Woodhouse Braine states that he has never seen bronchitis in a baby due to the administration of ether. When, however, the trachea is compressed or œdema of the air-passages threatens, the use of ether is to be avoided and chloroform to be preferred. In operations on the mouth, where it is impossible to keep the patient under ether except by interrupting too much the operative procedures, a stronger anæsthetic, as chloroform, either pure or with ether, is desirable. In operating for cleft palate this is particularly the case. It should not be forgotten, however, that chloroform mixtures are almost or quite as dangerous as pure chloroform. The A. C. E. (alcohol, one part; chloroform, two parts; ether, three parts) mixture has not demonstrated that it is to any great degree safer than chloroform alone, and, although it has been before the profession for fifteen or more years, it has not succeeded in establishing its value as a better anæsthetic than ether or chloroform alone. Gurlt in 1894,[1] from a large number of cases, gave one death from chloroform in two thousand six hundred and forty-seven, and with ether and chloroform mixed, one in eight thousand and fourteen. Joseph White[2] states that the A. C. E. mixture is certainly less depressing than chloroform, and apparently not more irritating to the pulmonary mucous membrane. In an administration of fourteen hundred and thirty-three cases he states that he has never seen any but favorable results. The use of alcohol added to anæsthetic mixtures is objectionable on account of its less volatility and slower elimination. The patient does not so readily recover from its effects as when either ether or chloroform is used.

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[1] Practitioner, October, 1896, p. 365.

[2] Ibid., p. 371.

[3] Wiener klinische Wochenschrift, 1895, Bd. viii. Ss. 1, 26, 44, 64.

[4] Medical Sentinel, vol. iii., 1895, p. 21.

The object of adding ether to chloroform is to stimulate the circulatory system. That it does so primarily there is no doubt, but later, according to H. C. Wood,[3] the pressure falls, so that it then becomes useless as an agent wherewith to counteract the depressing effects of chloroform. The *Lancet*[4] gives the following as a typical example of the usual onset of dangerous symptoms under the A. C. E. mixture. "Three minutes after the anæsthetic had been stopped the pulse was observed to grow weak, and in a few seconds to stop. The respiration continued for fully five minutes after this; no loss of color was seen, although just before the cessation of the pulse the lips became congested." It was evidently a case of failure of the circulation. Dr. Mears, in operating for cleft palate, uses one part of chloroform to three or four parts of ether, and, should a more powerful anæsthetic than ether be desired, I would be inclined to follow his example. Should an operation be necessary in a child suffering from albuminuria, my preference would likewise be for chloroform. There is no doubt that anæsthetics have considerable effect on the kidneys, often causing a temporary albuminuria when none has been detected prior to the operation, and increasing the amount of albumin when previously present. Chaput, Angelesco, and Le Noble,[5] in a series of one hundred and sixteen cases, found less renal disturbance after ether than chloroform. Eisendrath,[1] however, in one hundred and thirty cases, seventy chloroform and sixty ether, found that the amount of albumin was greater after ether than chloroform in the proportion of thirty-two to twenty-five; also that the changes are less marked in children than in adults. When the question of renal disturbance is a prominent factor, as little of the anæsthetic as possible should be given; this can be favored both by shortening the time of operating and by the use of chloroform.

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[1] Gesellschaft fur Chirurgie, Berlin, 1894.

[2] Transactions of the Medical Society of London, vol. xvii., 1894, pp. 205–226.

[3] Dennis, System of Surgery.

[4] Vol. i., 1895, p. 237.

[5] Bulletins et Mém. de la Société de Chirurgie, 1895, t. xxi. pp. 368–383.

Nausea and vomiting in children are usually neither so persistent nor so depressing as in adults. They are to be avoided by seeing that the patient's stomach is empty at the time of operation. It is preferable to operate in the morning. The stomach thereby has an opportunity during the night of emptying itself of any solid food that has been ingested during the previous day. On the day of operation a small amount of liquid food may be allowed, but should be taken, if possible, three hours previous to the administration of the anæsthetic. Vomiting in children is usually a much milder process than in adults; in children, however, the weakening effect of lack of food is more marked. For these reasons it is not desirable to enforce abstinence from food for so long before operation in children as it is in older people. Beef tea or a clear soup is preferable to milk, which is liable to be vomited in the form of clots. Minor operations requiring the use of an anæsthetic are more apt to be undertaken in children who have recently eaten solid food than in adults. Care should be taken that this does not occur. Children are inclined to bolt their food instead of properly chewing it, and their stomachs are liable to contain pieces of meat of considerable size which may readily produce serious trouble when vomiting takes place. I once nearly lost a case of circumcision from this cause.

The effect exerted by anæsthetics on the vitality of the patient is an important one. Anæsthesia is a distinctly abnormal condition. It cannot be kept up indefinitely or it will cause death. Therefore, other things being equal, the period of anæsthetization should be shortened as much as possible. Unduly prolonged anæsthesia weakens the patient and tends to lessen the chances of recovery. The best criterion we have of judging as to the strength or condition of a patient is probably the pulse. Shock and collapse show very markedly in the weakening of the pulse. Of course there are other signs, such as a blanching of the face, shallow respiration, and absence of reflexes, but if the pulse continues regular and of good strength there is usually no cause for alarm.

The constitutional effects of ether and those of chloroform are very different. There has been much discussion of recent years as to whether chloroform kills by paralyzing the heart or respiration. The practical clinician need be little concerned about it. He should know, however, that it has been positively demonstrated and universally accepted that chloroform has a very distinct depressant action on the circulation. Whether this is due to a direct action on the heart itself, as maintained by Professor Wood, or on the vaso-motor system, as held by Professor Hare, makes but little difference: it is still the circulatory system that is affected. The Hyderabad commission, while admitting that chloroform depressed the circulation, held that the respiratory function was first paralyzed, and that death came through this and not through cardiac paralysis.

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[1] Archiv für Kinderheilkunde, 1896.

The effect of the statements of the Hyderabad commission has been to cause the profession to magnify the importance of respiratory symptoms and minimize the importance of circulatory ones. While there has been this dispute about the pathology of chloroform deaths, there has been no dispute as to the clinical features. Ever since the introduction of chloroform cases have been occurring in which the circulation suddenly ceases, the patient collapses, and death ensues. Joseph White,[1] speaking from a personal experience of over seven thousand anæsthesias, says of chloroform that "warning of approaching danger may be gathered from the rapid enfeeblement and flickering of pulse which (as far as my own experience has gone) has almost invariably ushered in the crisis of danger." Hewitt, in the discussion of White's paper, said that the Hyderabad commission had attempted to lead the profession astray. Professor Hare in his last paper read before the Philadelphia College of Physicians, January 6, 1897,[2] stated that chloroform produced its effects on the circulation by paralysis of the vaso-motor system, and that the symptoms were those of hemorrhage, the patient being bled into his own vessels. The committee of the British Medical Association, in 1880, were probably very near the truth when they stated that, while chloroform always produced lessened blood-pressure, it had at times a most capricious effect on the heart's action, the pressure being reduced with sudden rapidity almost to *nil*, whilst the pulsations were greatly retarded or even stopped.

The effect of ether on the circulation has not been so elaborately studied. It is a well-known fact, however, that it is not nearly so depressing as is chloroform, and it practically never causes sudden death as does that drug. H. C. Wood, one of our best authorities,[3] states that during etherization there is usually a pronounced rise in the arterial pressure, which is commonly maintained even through a prolonged narcosis, and may continue after manifest failure of the respiration. Sooner or later, if the inhalation be continued, the rise of arterial pressure is followed by a fall, which may progressively increase. Again, in exceptional cases the stimulant influence of ether upon the circulation is very slight and transient.

Both ether and chloroform are liable to produce serious alterations in the respiration. The respirations should be sufficiently full and free to draw the air well into the lungs. If they are shallow it is a bad sign. Hewitt[1] pertinently remarks on this point that he was astonished to find that physiologists regarded respiration as proceeding so long as certain feeble and fitful contractions about the chest and abdomen persisted,—contractions which were, and for some time had been, utterly unable to cause the slightest ingress or egress of air. Practical surgeons should be careful that they do not fall into the same error. Cyanosis is no proof of lack of respiration, but it is of want of oxygenation of the blood. The case published by Stratton[2] is an example of this. The patient was a man aged thirty-four years. The operation was finished when he became cyanosed, and the pulse was lost at the wrist. Cyanosis quickly increased in intensity, but respiration was rapid, free, and full, gradually failing until voluntary respiratory efforts ceased. Death evidently occurred from heart-failure.

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[1] Transactions of the Medical Society of London, vol. xvii., 1894.

[2] Therapeutic Gazette, February 15, 1897.

[3] Dennis, System of Surgery, vol. i. p. 650.

ADMINISTRATION OF ANÆSTHETICS.

Before beginning the administration of an anæsthetic the anæsthetizer should be informed of the condition of the kidneys and lungs. If albuminuria is present, he should be aware of it beforehand. If the patient has bronchitis, advanced lung disease, or empyema, this likewise should be known to him. He should listen to the heart, so as to be informed as to the existence of any advanced organic disease thereof. Any obstruction to breathing through the nostril should be noticed, and the mouth examined to be assured that it contains no false teeth, tobacco, chewing-gum, or other foreign substance. As the patient is liable to vomit, a basin should be at hand, also a towel. A couple of small sponges on forceps will be of service to clear the throat of mucus. It may become necessary to open the tightly clinched jaws. For this purpose a wooden wedge three inches long by one inch thick is one of the best. A gag should also be at hand, the parts which touch the teeth being covered with rubber drainage-tube, as thereby the liability of breaking the teeth is lessened. This precaution is not infrequently overlooked. Some instrument should be provided wherewith to pull out the tongue. One of the many tongue-forceps on the market will do. The use of a hæmostatic forceps for this purpose is not to be encouraged. A tenaculum is efficacious if introduced well back in the tongue. I value aqua ammoniæ highly as a means of stimulating the respiration and restoring the patient. It should not be too old or it will have largely lost its strength. In using it the bottle should never be presented to the nose of the patient. The cork may be held in front of the nose, or, better, a small amount may be poured on the fingers of the anæsthetizer and held to the nose and mouth of the patient. It is surprising how quickly shallow respiration responds to this drug and becomes free and full. A hypodermic syringe, in working order should be handy, together with such stimulant medicines as may be desired. Personally my preference is for strychnine, one-thirtieth of a grain, and tincture of digitalis, five drops, administered twice or thrice, or more times during and soon after an operation if shock is at all pronounced. To these may be added one-hundredth of a grain of atropine if desired. The anæsthetizer should also be prepared to assist in measures for combating chloroform collapse. The head of the patient should be lowered and the body raised. A pad should be bound firmly on the abdomen. Artificial respiration may be performed, preferably by the method of Silvester. Flex the forearms on the arms, and grasp an elbow in each hand. Cross the patient's arms on the chest and press firmly on it, thus expelling the air; then draw the elbows down and out away from the body and up directly above the head, thus expanding the chest and drawing the air in. These motions should be made at the rate of from fifteen to twenty per minute.

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[1] Practitioner, October, 1896.

[2] Occidental Medical Times, Sacramento, 1895, vol. ix. pp. 8–13.

The method of rhythmical traction of the tongue (of Laborde) has lately been used in France with favorable results. It consists in grasping the tongue and drawing it forcibly forward at intervals of a few seconds. It is of too recent introduction to decide positively as yet as to its value. König's method of intermittent compression over the region of the heart has also been recommended, but has not found general acceptance as yet. Electricity, although before the profession a long time, has failed to show itself of any real service.

Disturbance of the respiration to a more or less serious degree occurs constantly. When slight, it is often caused by the tongue and epiglottis falling back or by an accumulation of mucus. This latter can be removed by sponging. The obstruction arising from the tongue and epiglottis can usually be removed, as advised by Howard, by forcibly extending the head. This should be accomplished by pressing the chin from below upward and backward to an extreme degree. In pulling the tongue forward by means of a forceps or tenaculum it should not be pulled down over the lower teeth, but upward and forward as advised by Hare and Martin. They also advise extending the patient's head and bringing it forward in the position held in running.

When, in performing artificial respiration, no air is observed entering and leaving the chest, Alexander Maclennan advises tracheotomy.[1] He gives three cases in which this was successfully done. Five causes of obstruction are given,—viz., 1, tenacious secretion in the larynx or trachea; 2, spasm of the cords; 3, œdema of the larynx; 4, vomited matter; 5, a combination of two or more of the above conditions. In addition to these, it seems to me that at times the epiglottis is held so firmly down on the larynx by spasm or attempted inspiration as to frustrate all efforts to dislodge it by altering the position of the head or by traction on the tongue.

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[1] British Medical Journal, November 21, 1896.

In this case tracheotomy will allow free ingress of air and may save the patient.

The attempts to obviate the dangers incident to chloroform have led to the invention of numerous inhalers. Only the most simple of these have ever been much used. The dangers of chloroformization are best guarded against by exceeding caution and care in administration. Attention paid to or any reliance placed on special forms of inhalers detracts from the watchfulness that should be bestowed on the patient, and is liable to engender a false sense of security which may end in disaster. Chloroform, I believe, is best administered by dropping from a drop-bottle on one or more thicknesses of flannel, lint, or gauze stretched over a wire frame. This is Skinner's apparatus. Esmarch's apparatus is only a little more simplified form of Skinner's. Having the flannel stretched on a frame instead of using it alone is better because it keeps the material a short distance away from the face and therefore does not prevent free access of air, and also because it is more readily handled. The use of chloroform in any closed ether inhaler, as those of Clover and Ormsby, is decidedly more dangerous. In Junker's inhaler air is pumped through a bottle of chloroform to either a face-piece or a tube introduced into one nostril. This is not so safe as the wire frame described above. In operations on the mouth the inhaler of Junker is convenient, but I believe it safer, even in such cases, to administer the anæsthetic either by dropping it on the corner of a folded piece of lint held over the nostrils, and at times the mouth, or by dropping it on a small pad of absorbent cotton held by means of a forceps. The administration of oxygen with chloroform, as suggested by Neudörfer, of Vienna, and others ten years ago, has recently again been tried in Philadelphia and Brooklyn. That it diminishes the dangers of chloroform administration is probably true, but that it does away with them has not been proved. For anæsthesia in children the combination has little to recommend it, and will probably never come into general use. The administration of ether satisfactorily is an art which requires a considerable amount both of study and practice. The ignorance and carelessness attending anæsthetization by means of it are simply appalling. Patients are terrorized and made to suffer unnecessarily, and the operator is worried and harassed by the necessity of superintending the anæsthesia as well as performing the operation. In giving ether the following points should be observed:

1. Begin the administration very gradually. Never attempt to give it in a concentrated form at the start. The vapor of ether is exceedingly pungent. If concentrated, and the respiratory passages have not had their sensibility already partially deadened, it is almost irrespirable. It is so irritant that spasm of the glottis is at once provoked, the patient feels suffocated, and struggles to the utmost to get a breath of fresh air. This fault in giving the drug is exceedingly common, and is unpardonable, for it is the result either of ignorance or of carelessness. It is to be avoided in

one of three ways. If an inhaler is used in which the amount of vapor can be regulated, as in the Clover, the air can be turned off gradually and the ether vapor admitted very slowly. If the ether is being given on some porous substance on which it is dropped, it should be given at first a single drop at a time, and this increased as its effects become more manifest. If a cone is used after the ether has been poured into the interior, it should be held at quite a distance from the face and approached very gradually until the nose and mouth are covered.

2. When the patient begins to show signs of excitement it is evidence that the stage of irritation has passed and consciousness is disturbed or lost; the anæsthetic should then be given in its most concentrated form, pushed to its utmost. This is to shorten the period of excitement. In many patients, if adults, fewer if children, this stage of excitement seems to last almost indefinitely if any air is admitted not saturated with the ether vapor. This can be avoided by keeping the inhaler so closely applied to the face that no air can come in except that which is loaded with the ether vapor. If a face-piece is used, it must be pressed firmly on the face. If a towel or cone is used, it should be held closely around the nose and mouth, so that no air can enter beneath the edges. Without this precaution the use of ether is often very unsatisfactory and exceedingly troublesome.

3. Avoid cyanosis. This is to be accomplished by seeing that all obstructions to the free ingress of air are removed and by temporarily removing the ether. Cyanosis is due to a variety of causes. Usually the patient is not breathing properly, or perhaps not at all. In this case remove the ether at once and free the air-passages by extending the head to its utmost or by drawing the tongue up and out or pulling it up with a tenaculum inserted in its base. It may be that mucus or vomited matter is lying in the pharynx; if so, the mouth should be pried open with the wooden wedge or mouth-gag, and the pharynx wiped out with a sponge held in either a forceps or a sponge-holder. In all difficulties with respiration, particularly from mucus, the patient should be turned on the side and the face turned downward; then the mucus will run out of its own accord, and often even the tongue will fall away and cease to obstruct the larynx. If the patient is on his back the slightest collection in the mouth will tend to fall back and obstruct the opening of the larynx. Cyanosis is almost always due either to the patient holding his breath more or less voluntarily or to mucus or vomited matter. It is, however, not always so. It may occur even while breathing is going on. A marked example of this is the case of Stratton, already quoted. If the anæsthetizer is inexperienced he is apt to cause cyanosis by excluding the access of air by means of the inhaler used. This is particularly so if etherization is induced by pouring the drug on gauze or a towel laid directly on the face of the patient. When gauze gets soaked the fibres mat together and prevent the passage of air, and in etherization by that method I have frequently

seen the gauze so saturated that the ether was running in streams from its edges. Under these circumstances the liquid ether is also liable to be drawn directly into the air-passages.

4. The anæsthetic should not be kept continuously applied. After the patient has once been brought under its influence it requires a much smaller amount of the drug. Therefore, at intervals of a few minutes or less, according to the form of inhaler used, it should be removed and the patient allowed to breathe fresh air. It requires a certain expenditure of physical force to breathe through gauze or inhalers, and when anæsthesia is sufficiently profound the inhaler should be removed entirely. It is a habit of some anæsthetizers to allow the inhaler or gauze to remain in place, and simply because no fresh ether is added to think that no harm is done. This is all wrong. When it is not desired to give additional ether, everything should be removed and the patient allowed to respire freely.

5. An unnecessary amount of the anæsthetic should not be administered. In children the effect of profound anæsthesia is marked, and the pushing of the anæsthetic after the patient is once under its influence is not to be tolerated. It should be the aim of the anæsthetizer to perform his duties with the expenditure of the least possible amount of the drug. Ether-"soaked" patients do not recover readily from shock, and have more trouble from after-vomiting, nausea, and general disturbance than they otherwise would have.

6. Vomiting during the anæsthesia is due to the patient coming from under its influence. It is to be avoided by so carefully watching the patient as to detect the first signs of approaching consciousness and "push" the ether before vomiting can take place.

The question must be decided as to what form of inhaler shall be used. It should be borne in mind that skill in administration is the main element in successful etherization. A careful and skilful man can safely etherize a patient with any form of inhaler. Inhalers are mainly intended to aid the unskilful and to protect the patient. They are of two forms, the one closed and the other open.

Closed inhalers are those in which the patient breathes to and fro into a rubber bag, thus respiring the same air over again. The Clover is the best-known type.

Open inhalers are those which do not exclude air, such as gauze, towels, the Allis inhaler, and many others. In these the air passes directly through the inhaler.

The induction of anæsthesia is oftentimes a troublesome, tedious, and difficult procedure.

The difference between open and closed inhalers is marked. With closed inhalers my experience leads me to believe that anæsthetization can be more easily and quickly accomplished, with less discomfort to the patient and the anæsthetizer, and the expenditure of a less amount of the drug. The ether vapor also is not disseminated so much throughout the apartment.

It is a much more powerful agent, however, than open etherization. In the hands of the unskilful it may become dangerous. The concentrated ether vapor plus the carbonic acid poisoning due to the repeatedly respired air is a method that has to be carefully watched, particularly if used exclusively. It is not so objectionable if the administrator is careful and skilful, or if it is used simply to bring the patient under the influence of the anæsthetic and then an open inhaler substituted. H. C. Wood condemns closed inhalers as being dangerous. That they are so under certain circumstances is probably true, but their employment for the induction of anæsthesia, and not its maintenance, unless in careful and skilful hands, would, in my judgment, rob etherization of many of its terrors and save the patients a large amount of suffering. In children, however, the closed method is hardly ever desirable. If young, they go under the influence of the anæsthetic so readily that etherization with the open method ordinarily is not attended with difficulties.

In the open method the ether is given either by dropping it on folded gauze or a porous towel, which may or may not be folded in the form of a cone, or by means of some special inhaler. The inhaler most used is that of Dr. O. H. Allis, described and figured in a previous volume (iii. p. 916). It consists of a frame with many folds of a bandage strung from side to side with the edges up. The ether being poured on the edges of the bandage evaporates from its sides while the air passes freely up and down between the folds. This inhaler, particularly in children, acts satisfactorily; a smaller size, however, is more desirable than that used for adults. I have recently used an inhaler made of an oval frame, covered, which contains a hole through which the ether is poured on gauze, which is held in place by means of a wire frame. The inhaler is made to fit the face by having a cover of rubber sheeting which projects an inch beyond its lower edge.

The use of gauze likewise requires skill. Twenty-four thicknesses of a wide-meshed gauze should be employed. The tendency is to have too few layers and too fine a mesh, both of which are objectionable. I think it much less desirable than one of the forms of inhalers described above. The use of oxygen in connection with anæsthesia is attracting considerable attention. It will, however, not be as important in children as in adults. It is largely to Drs. C. S. Cole and F. H. Markoe[1] that we owe its introduction. It is not satisfactory to use it by passing the gas through the ether, as done with chloroform, because it is too difficult to give the ether in a sufficiently concentrated form, but it should be given pure alternately with the ether vapor. Etherization should first be induced by the usual methods and then the ether inhaler removed and oxygen given. The ether inhaler may then be replaced. At the close of the anæsthetization oxygen should be given alone. The method of giving the oxygen is to allow the gas to escape from the cylinder into a rubber bag. From this it passes through a wash-bottle containing water and is conducted by means of a rubber tube to a paper cone. This paper cone should be large enough at its base to cover the nose and mouth of the patient, and the tube should be stuck into its end or apex. I have found this method of administering the oxygen of marked service in adults, but have not considered its use so desirable in children.

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[1] New York Medical Record, October 12, 1895.

Likewise the use of nitrous oxide gas to precede the administration of the ether, while of great service in adults, particularly if followed by the use of a closed inhaler like those of Clover, Ormsby, or Wanscher, is hardly necessary in children.

TUMOR-GROWTH IN CHILDHOOD.

BY DOUGLASS W. MONTGOMERY, M.D.

THE true tumors are the result of error of growth in one or more of the four great groups of tissues of which the body is composed: of epithelial tissue, of connective tissue, of tissue of muscle, of tissue of nerve. In childhood the tumors resulting from an error of growth of the connective tissues are by far the most frequent. Many of them are congenital, and many more of them, not demonstrably present at birth, show by their early appearance or by their structure that their foundation was laid in early fœtal life, before the tissues of the body had definitely arranged themselves.

Marc, in searching the records of the Prince Peter of Oldenburg Hospital of St. Petersburg, has found[1] that among the five thousand and twenty children treated in the surgical clinic during fourteen years there were one hundred and twenty-one cases of neoplasm. Seventy-three, or 60 per cent., of these cases were females, and forty-eight, or 40 per cent., were males. In forty-five of these cases, or 37 per cent., the tumor was observed in the first year of life, and in four of these the affection was congenital. From the first to the sixth year there were thirty-five cases, from the seventh to the twelfth year there were thirty-six, and there were five cases in children older than twelve years. Forty-six, or 38 per cent., of the tumors were angiomas; thirteen, or 11 per cent., were sarcomas; ten, or 8.3 per cent., were cystomas; nine, or 7.4 per cent., were papillomas; eight, or 7 per cent., were malignant lymphomas; five, or 4.1 per cent., were polypi; five were hygromas; five were atheromas; three, or 2.5 per cent., were lipomas; three were epulis; two, or 1.6 per cent., were chondromas; two were lymphangiomas; two were granulomas; two were strumas; two were ranulas; one, or 0.8 per cent., was a fibroma; one was a glioma; one was an adenoma; one was a keloid.

It will be seen that in childhood more females are afflicted with tumor-formation than males. Farther on, however, it will be shown that although tumor-formation is more frequent in female than in male children, yet malignant disease is far more common in boys than in girls. This is due to the fact that sarcoma, which is almost the only malignant disease of childhood, is in childhood, and indeed throughout life, by far more common in males than in females. But later in life, although sarcoma becomes more frequent than in childhood, and more sarcomas continue to develop in males than in females, yet females become more afflicted with malignant disease than males. This increase of malignant disease in females is due to the great susceptibility of the womb and female mammary glands to cancer. This is an excellent illustration of the inadvisability of classifying diseases under a purely clinical attribute, such as malignancy. In fact, the term malignant disease is simply the expression of an opinion as to how a given neoplasm will act, and is not a diagnosis, as any one of the neoplasms may become malignant.

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[1] Ein Fall von Leiomyoma subcutaneum congenitum, von Dr. Serg. Marc, Virchow's Archiv, Bd. cxxv. S. 543.

SARCOMA.

Although sarcoma cannot be said to be rare in childhood, it is much less frequent at this time of life than in adults. However, it constitutes practically the only malignant tumor of children, as carcinoma is rarely met with at such an early age.

According to D'Espine and Picot,[1] in four hundred and twenty-seven cases of malignant neoplasm in childhood, seventy-seven occurred in the first year, thirty-nine from one to two years, fifty from two to three years, and forty-eight from three to four years; later they occurred more rarely. The kidneys and the testicles were the organs most frequently attacked. The kidneys were the seat of the disease one hundred and thirty-six times in the four hundred and twenty-seven cases, and most of them occurred under four years of age. In point of frequency, malignant disease of the eye and the orbit came next after that of the kidney and testicle; then the bones and periosteum.

In children, as in adults, there is frequently a history of sarcoma quickly following a blow or other injury.

In early life malignant disease is particularly apt to recur, and Emanuel says[2] the reason for this lies, in a great measure, in the fact that the diagnosis of malignancy is made too late. While the tumor is still small, the surgeon cannot realize the malignant character of the disease, and therefore hesitates to subject such a young person to an operation that always has its dangers. When the patient is seen at an early stage of the disease, other signs of malignancy, such as loss of flesh, pain, and, if the sarcoma is in the abdominal cavity, ascites, are generally not present, and one is only too inclined not to take such a grave view of the case. A second examination, which possibly is made only a short time after the first, may be in the nature of an unqualified surprise. The tumor may not alone have grown very rapidly, which of itself is a suspicious circumstance, but other conditions may have so changed for the worse that no doubt exists of the nature of the trouble. An operation is now undertaken with the hope of relieving the patient of pain and discomfort, but with little outlook for the cure of the disease.

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[1] Quoted by Wincouroff, Arch. f. Kinderheilkunde, Bd xxi. S. 203, 1896.

[2] Ueber Teratoma Ovarii, von R. Emanuel, Zeitsch. f. Geburtsh. u. Gynäk., Bd. xxv. S. 206.

Sarcoma of the Mediastinum.—W. A. Edwards[1] has tabulated sixteen cases of sarcoma of this region in childhood. The ages varied from five to eighteen years. All the cases were fatal; the duration varied from three weeks to ten months. The following varieties were recorded: lymphosarcoma, ten; round-cell sarcoma, three; not stated, three.

Sarcoma of the Kidney.—Sarcoma of the kidney has received a good deal of attention recently,[2] and deservedly so, as Döderlein,[3] after summing up all the reported cases of tumor of this organ at whatever age, has found that thirty-eight per cent. of them occur in childhood. Most of the tumors of the kidney in early life are sarcomas, and not alone this, but sarcoma is by far the most common abdominal tumor of childhood.

Sarcoma of the kidney usually manifests itself as a painless, solid, smooth, rounded tumor, slightly movable but not displaceable, growing rapidly forward from the loin, in the space between the crest of the ilium and the arch of the ribs, and pushing the colon in front of it. The tumor most frequently appears during the first four or five years of life. There may be blood in the urine appearing either as casts of the pelvis of the kidney or of the ureters, or as smoky urine, or as a fairly constant small number of red blood-corpuscles discoverable only with the microscope.

Most of the sarcomas of the kidney are small, round-celled growths; others are spindle-celled. Still others contain a great variety of connective tissue, muscle tissue, both striped and smooth, and epithelium, but retain nevertheless the general characteristics of sarcomas. The results of operation are better now than they used to be, but of course they are bad, and must, from the desperate nature of the affection, remain so. Lewi has recently found[4] the mortality due to operation to be about twenty-eight per cent., and fifty-three and one-third per cent. of his list of cases died from recurrence of the disease. The subsequent history of quite a number of Lewi's cases was not ascertainable, but only three out of sixty, or five per cent., were known to have passed the three-year limit without recurrence.

The average duration before a fatal termination in cases not operated on is about one year.

Sarcoma of the Testicle.—As mentioned above, next to the kidney the testicle is the organ most frequently attacked by sarcoma in children. Many of these tumors, like those of the kidney, are highly complicated growths, containing not alone connective tissue, but also epithelial elements. It is such tumors, occurring in any region of the body, that justify Thoma in his wide definition of sarcomas as a group of tumors in which epithelial structures either are completely absent or occur merely as accidental or unimportant structures.[1] It would probably be better to classify these mixed tumors under malignant teratomata, with the understanding that they act like and are very similar to the more simply formed sarcomas.

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[1] Archives of Pediatrics, July, 1889.

[2] *Vide* articles by Wentworth, Fruitnight, and Lewi in Archives of Pediatrics, 1895–96, and cases by Osler in The Diagnosis of Abdominal Tumors, New York Medical Journal, July 28, 1894.

[3] Quoted by Lewi, *vide supra.*

[4] Archives of Pediatrics, February, 1896.

Sarcoma of the testicle in children usually occurs early; the neighboring lymphatics are quickly involved, and an operation is rarely performed soon enough to avert a fatal issue.

Primary Sarcoma of the Bladder in Children.—F. Betzold[2] has been able to gather only thirty-two cases, including one of his own, of this rare affection. In thirty of these, where the sex was mentioned, twenty were males and ten were females, so that the bladder in children follows the general rule of sarcomas that males are more liable to be affected than females.

As in sarcoma of the kidney and testicle, the great mass of the cases occurred during the first five years of life. In twenty-nine cases where the age was given, twenty-three were during the first five years of life, including one new-born infant, and six cases fell between the fifth and thirteenth years.

Hæmaturia, which usually appears very early in the adult, is seldom present in the child. The first symptoms are usually those of difficult urination, strangury, anuria, pain in the bladder and in the glans penis, causing many of the patients to pull continually at the organ.

The prognosis is, as a rule, fatal.

Sarcoma of the Liver.—A few cases of primary sarcoma of the liver in children have been reported,—one each by West,[3] Roberts,[4] Lendruf,[5] and Axtell.[6] The difficulties of diagnosis seem to have been uniformly insurmountable, and the issue, of course, fatal.

Primary Sarcoma of the Pancreas.—The diagnosis of sarcoma of the pancreas seems to be as obscure as that of the liver. Wincouroff has reported a case[7] in a boy of twelve years, where there was no glycosuria,

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[1] Thoma's Pathology. Translation by Bruce, p. 580.

[2] Der Kinderarzt, 1896, Bd. vii. S. 80. Abstract by T. M. Rotch and A. H. Wentworth, Boston Medical and Surgical Journal, January 14, 1897.

[3] West's case was in a child of eight months. West, Diseases of Children, 1884, p. 764; quoted by Leith, Lancet, January 16, 1897.

[4] A Case of Fungus Hæmatodes of the Liver. Lancet, January 19, 1867, p. 77; quoted by Leith, Ibid., January 16, 1897.

[5] Universal Medical Journal, Philadelphia, 1893; quoted by Byrom Bramwell, Lancet, January 16, 1897, p. 170.

[6] Primary Sarcoma of the Liver in a Male Child of Three and One-Half Years of Age, by E. R. Axtell, of Denver, Colorado. New York Medical Journal, March 3, 1894.

[7] Zur Lehre von der Sarcomatose, Wincouroff, Arch. f. Kinderheilkunde, Bd. xxi. S. 203, 1896.

[illegible] of appetite [illegible] lead one to suppose that the pancreas was affected. In Litten's case, where a diagnosis of neoplasm of the kidney was made, there were no digestive disturbances, nor glycosuria [illegible]. And in [illegible]'s case[2] neither glycosuria nor [illegible] was present.

The course of sarcoma of the pancreas seems to be a rapid one, as, for instance, Winocouroff's patient was only five weeks in the hospital and Litten's case lasted only a few weeks.

MYXOMA.

Pure myxoma is a rare tumor, as mucous tissue is most frequently a minor constituent of some of the complex connective-tissue or teratomatous neoplasms. The uncomplicated myxomas usually appear in youth, principally in children, and sometimes are congenital.

A myxoma sometimes appears at the navel, and is called fungus umbilici or sarcomphalus. It is a fungus-like growth, which is either covered with skin or ulcerated, and usually dries up and disappears with the shrivelling of the umbilical vessels.

It appears that even some of the pure myxomas have been found malignant, and the myxo-sarcomas are so, of course.

KELOID.

Keloid rarely occurs before puberty, but may appear in childhood, or even be congenital.[3] Vidal mentions a cicatricial keloid that appeared after vaccination at the age of three months, and at four years each of the tumors had reached the size of a hazel-nut.[4]

Negroes are particularly liable to keloid, and the predisposition to this form of growth may even be hereditary with them. A. L. Hogdon has reported a negro family in which a father, his daughter, and two daughters of this daughter were all afflicted with keloid.[5]

Keloid of the lobe of the ear, consequent usually on puncture, most frequently occurs in early life, but this obviously depends upon the fact that the female of our species enters on the ear-piercing stage of her existence towards the last years of her childhood.[6]

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[1] Read before the Wiener med. Gesellschaft, October 16, 1880; quoted by Winocouroff, loc. cit.

[2] Mentioned by Winocouroff, loc. cit.

[3] H. Radcliffe Crocker (Diseases of the Skin, 1893, p. 624) cites a case of congenital keloid reported by Bryant, and A. von Winiwarter (Die Chirurgischen Krankheiten der Haut, S. 501), on Volkmann's authority, also says that it may occur congenitally.

[4] De la Cheloïde, par H. Leloir et E. Vidal. Annales de Dermatologie et de Syphiligraphie, 1890, p. 193.

[5] Hereditary Predisposition to Keloid in a Colored Family, by A. L. Hogdon. Maryland Medical Journal, 1896, vol. xxxiv., No. 21; abstract in Archives of Pediatrics, September, 1896, p. 720.

[6] For references on this subject see article by William Scheppegrell, New York Medical Journal, October 17, 1896, p. 510.

FIBROMA.

Many of the fibromata, both solitary and multiple, commence in childhood, or even in infancy. The multiple ones may exist in hundreds, either scattered along nerve-trunks or as small, movable, painless nodules, situated in the skin or subcutaneous tissue.

Fibroma molluscum is sometimes present at birth or develops during the first years of life.

LIPOMA.

Lipoma occurs very rarely in childhood. It usually appears later in life, when more fat is formed.

ENCHONDROMA.

By a simple application of the law of chances it follows that the more actively a tissue increases the more subject it will be to errors of growth. Tissue of bone and tissue of cartilage are particularly inactive structures, and therefore are not frequently subject to errors of growth leading to tumor-formation. They are, however, during childhood comparatively much more active than in adult life, and, corresponding to this, it is during childhood that most of the chondromas and osteomas appear.

Usually chondromas enlarge very slowly, but their rate of growth is singularly uncertain. Paget relates the case[1] of a little girl with a cartilaginous tumor surrounding the upper two-thirds of the tibia that grew to a circumference of two feet in eighteen months. Quickness of growth in tumors frequently indicates malignancy, but chondromas may increase with startling rapidity and yet be of perfectly benign nature. Paget refers to the above case and some others as instances where rapid growth in tumors was not associated with malignancy. The cartilaginous tumors that grow in connection with the cranial bones are particularly apt to progress quickly, and they have also an ugly habit of running along between anatomical planes of tissues and insinuating themselves into fissures and foramina, making it particularly hard for an operator to eradicate them. This difficulty of extirpation, together with their activity, makes such tumors appear malignant when they really are not so.

The growth of cartilaginous tumors in the phalanges sometimes twists the fingers into the most gnarled and fantastic shapes. These digital enchondromas begin almost always early in life, even before birth, enlarge slowly and painlessly, and may involve one, several, or all of the fingers of one or both hands.

OSTEOMA.

As has been mentioned in considering enchondromas, osteomas occur most frequently in childhood. They are usually found growing from the skeleton and in the neighborhood of the junction of the diaphysis with he epiphysis. This is exactly what one might expect, for it is the point f greatest activity in the growth of a bone, and is therefore most exposed to those errors of growth that are called tumors. Another fact that connects them very closely with the growth of bones is that they usually cease to increase in volume when the patient has attained his full stature. When osteomas are multiple, and they often are so, they are commonly associated with a lack of growth of the affected bones. As a rule, osteomas are of slow growth, and they are not malignant. They are among the few tumors that show any hereditary tendencies, and the heredity is sometimes very marked, as corresponding bones or parts of bones are often similarly affected in different members or generations of the same family.

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[1] Paget's Pathology, p. 490.

The frequency of the occurrence of osteomas at the site of the epiphyseal cartilage, and their appearance in childhood when this cartilage is most active, their heredity and symmetry, as well as their cessation of growth on the attainment of adult life, all point them out as the best example of a tumor-growth due to some inherent quality in the tissue itself.

GLIOMA.

Gliomata of the brain and spinal cord frequently occur in early childhood, and, as a rule, they grow slowly. Holt and Herter have, however, reported a glioma in a child twelve months old, in whom, although the disease had lasted for only three months, the gliomatous tissue occupied a good deal of the medulla and almost the entire extent of the cord.[1]

MYOMA.

Striped muscle-tissue occurring as a tumor is rare, and forms usually only one constituent of those complex tumors that most frequently develop in the kidney or testicle. They are also found in other parts of the body; for instance, Prudden has reported such a tumor of the parotid region occurring in a boy of seven years,[2] and recently a complex neoplasm of the pelvis, containing striped muscle-fibres,[3] has been described in a male infant of eleven months. Thoma says that a mass of striated muscle-fibres forms one of the varieties of congenital macroglossia.[4]

Tumors formed from smooth muscle-fibre almost always occur in adult life, some of them very late, as the leiomyomata of the prostate. Myoma of the uterus is decidedly an affection of middle age. The myomata of the skin, however, form a notable exception to this, as they may even be congenital,[5] and some of them have been reported as appearing during the first years of life.[1] Nevertheless, the table of cases collected by Brigidi and Marcacci shows that even the dermatomyomata generally develop in middle life.[2] De Bruyn Kops has recently reported a case in which the muscular ring of the pylorus formed a tumor of cartilaginous hardness the size of a marble, which caused stenosis of the outlet of the stomach, and from which the patient, a new-born male babe, died in three weeks.[3]

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[1] Medical News, vol. lxvi., January 12, 1895.

[2] Rhabdomyoma of the Parotid Gland, by T. Mitchell Prudden. American Journal of the Medical Sciences, vol. lxxxv., 1883, p. 438.

[3] Ueber einen Fall von Teratom des Beckens, von Dr. Fr. Bergkammer. Deut. med. Wochensch., October 29, 1896.

[4] Pathology and Pathological Anatomy, by Richard Thoma. Translated by Bruce, vol. i. p. 559.

[5] Dr. Serg. Marc, of St. Petersburg, has reported a case of congenital leiomyoma in the scalp of the occipital region. Virchow's Archiv, Bd. cxxv. S. 543. The tumor grew very rapidly, but it did not appear to be malignant.

NEUROMA.

Neuroma is an exceedingly rare tumor at any age, and most of the tumors reported as neuromas are either fibromas, myxomas, or painful myomas.

CANCER.

Cancer, of whatever form, is an exceedingly rare disease in childhood. Among four thousand seven hundred and sixty-nine cases of carcinoma treated in the general hospital in Vienna, Gurlt has found that only ten per cent. were in the first decennium of life.[4] Its infrequent occurrence at an early age is possibly because it requires a long-continued, though often slight, irritation to produce the change in the epithelial cells by which they become infiltrated and malignant, and because it is only rarely that such an irritation exists or has time to act in childhood.

There is, however, one disease, xeroderma pigmentosum, that shows that cancer can arise even in childhood when the necessary long-continued irritation is present. Xeroderma pigmentosum is the name given to the intense reaction set up in a peculiarly susceptible skin on exposure to light, especially to the blue actinic rays. When a child having such a skin reaches the second year of life and begins to go out-doors, it is noticed that the face and hands become slightly puffy and diffusely or mottled livid red, and the surface of the skin grows very rough. Afterwards the child becomes very much freckled. This freckling is followed by scar-like areas of atrophy, with streaks or points of dilated capillaries, and the epidermis becomes wrinkled and branny. Flat, wart-like structures, which at first are transient, grow more and more numerous, and towards the sixth or seventh year of the child's life, or later, some of them develop into true flat epithelial cancers, exactly similar to rodent ulcers. The occurrence of these cancers is invariable, so that there is no doubt of the sequence of events.

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[1] In Hess's case, a nineteen-year-old girl had had the affection from her fourth or fifth year. The tumors were situated on her nose, and grew slowly. Ein Fall von mult. Dermatomyoma an der Nase, von Dr. Karl Hess. Virchow's Archiv, Bd. cxx. S. 321.

Babes mentions a myoma in the palm of a young man, in whom it had existed from infancy. Handbook of Skin Diseases, by H. v. Ziemssen, p. 608.

In Jadassohn's second case of multiple myoma of the skin, the patient, who was thirty-seven years of age, remembered distinctly that she had had the affection when she first went to school, in her seventh year. Zur Kenntniss der multiplen Myome der Haut, von Dr. J. Jadassohn. Virchow's Archiv, Bd. cxxi. S. 88.

[2] Annales de Dermatologie et de Syphiligraphie, 1882, p. 119.

[3] Nederlandsch. Tijdshrift voor Geneeskunde, No. 25, December 19, 1896. Abstract in British Medical Journal, January 30, 1897.

[4] Deut. Chirurgie, von Lücke und Zahn, Lieferung 22, 1ste Hälfte, S. 70.

Although cancer is so infrequent in childhood, the foundation for this affection is often laid at a very early age. The cancer itself may actually develop in adult life, yet the true commencement of the affection may date even from before birth, for true carcinoma not infrequently develops in congenital anomalies, and in such a manner as to show that the anomaly itself had something to do with its origin. The following are some of the congenital anomalies in which cancer is said to develop: phimosis congenita,[1] circumscribed hypertrophy or atrophy of the skin, congenital warts with papillary hypertrophy and thickening of the epidermis, pigment-spots, circumscribed congenital albinismus, and hypertrophy of the hair-follicles, of the sebaceous glands, and of the sweat-glands.

It has been asserted that teratoma is one of the congenital anomalies in which carcinoma is apt to develop. There is, however, only one case on record[2] where a carcinoma has developed in a teratoma. In all the other cases of malignant disease developing in teratoma, although the malignant affection has several times contained epithelial elements, it has always borne more resemblance to sarcoma than to carcinoma, and should be so classified. There are, it is true, twelve cases[3] in which cancer has developed in dermoids of the ovary; but dermoids of the ovary, according to Wilms,[4] are not teratomata and do not arise congenitally, but come direct from an egg in the ovary in which they occur. They, therefore, form a class apart from dermoids of other parts of the body, and do not enter into a discussion either of tumor-growth in childhood or of teratomata.

Scars that are acquired in early life may be the site of early carcinoma in the adult. The epitheliomata that arise in the navel cicatrix, in the scars from fontanelle ulcers, and in the scars from burns in childhood may all be included under this head.

W. A. Edwards[5] presents a table of eleven instances of carcinoma of the mediastinum in children whose ages varied from four to eighteen years. The cases were, of course, all fatal, and lasted from a few weeks to about three months. The varieties were: encephaloid, three; medullary, one; lymphoid, one; scirrhus, three; not stated, three.

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[1] A. von Winiwarter says that of thirty cases of carcinoma penis, twenty-three had congenital phimosis, and of these twenty-three, seven had been circumcised before the development of the cancer. Die Chirurg. Krankheiten d. Haut, S. 651.

[2] Entwickelung eines Epithelcarcinoms aus einer angeboren Sacralgeschwulst, von Dr. Vinzenz Czerny. Archiv für klinische Chirurgie, Bd. x. S. 894.

[3] These twelve cases are: I., Heschl's, Prager Vierteljahressch., 1860; II., Biermann's, Prager med. Wochensch., 1885, No. 21; III., E. von Wahl's, Petersburger med. Wochensch , 1883, S. 70; IV., Pilliet's, Session of April 2, 1893, of the Anatomical Society, Paris; V., Tauffer's, Virchow's Archiv, Bd. cxlii. S. 389; VI., Himmelfarbs's, Zur Kasuistik der Dermoid Geschwülste des Eierstocks; VII., Krukenberg's, Arch. f. Gynäkol., Bd. xxx. S. 245; VIII., Veits's, Centralbl. f. Gynäkol., 1890, S. 283; IX., Wilms's, Deut. Arch. f. klin. Med., Bd. lv. S. 368; X., Klein's, Inaug. Dissert., Freiburg, 1893; XI. and XII., Yamagiva's, two cases, Virchow's Archiv, Bd. cxlvii. S. 99.

[4] Deut. Arch. f. klin. Med., Bd. lv.

[5] Ibid.

CONGENITAL DISLOCATIONS.

By ROBERT W. LOVETT, M.D.

THE question of the etiology of congenital dislocations in general has been fully discussed in the original article, to which the reader is referred. The aim of the present article is to record especially the progress made in the study of congenital dislocations, regarding chiefly the practical surgical aspect of the question. So voluminous has been the literature in the last few years that this article must be chiefly an abstract, referring the reader who wishes detailed information on theoretical points to the original sources.

HIP.

Congenital dislocation of the hip is relatively so much more important than all other congenital dislocations taken together that it overshadows them. The view that the cause lies in an arrest of development has gained ground largely to the exclusion of other theories in all but exceptional cases. In Paris the malformation appears to be about one hundred times as common as malformations of the arms and rectum.[1]

The collection [2] of further figures shows the great disproportion between boys and girls; in seven hundred and thirty cases, six hundred and fifteen (eighty-four per cent.) were in girls and one hundred and fifteen (sixteen per cent.) in boys. In six hundred and forty-eight cases, three hundred and ninety-one were single and two hundred and fifty-seven were double dislocations.

It is recognized more fully of late years that, although the head is relatively too large for the acetabulum and often stunted and malformed, the excessive displacement is due to the bearing of the body-weight in walking,—that is, secondary changes result from the simple malformation, and are often confused with it. It is fully recognized also that simple reduction of the head into the acetabulum is not enough. Retention is necessary, and generally enlargement of the acetabulum. Hoffa has taught that the muscles offer the chief obstacle to resistance, but it seems probable that the capsule, and notably its anterior portion, is still more important.[1]

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[1] Broca, Bulletin et Mém. de la Soc. de Chir., 1896, t. xxii. p. 297.

[2] Karewski, Centralblatt für Chirurgie, 1892, S. 36.

The researches of Saniton[2] have shown that in infancy the hip-joint is shallow, and for that reason unstable,—*e.g.*, the relation of the depth of the cavity to the diameter of the head of the femur at birth is one to three; at five years, one to two; and in the adult (Sappey) it is three to five and one-half.

The symptoms have been fully described in the original article on Congenital Dislocations.

The diagnostic points are elevation of the trochanter above Nélaton's line, shortening of the leg, instability of the hip-joint in manipulation, with perhaps a click, and a characteristic yielding limp; in cases of double dislocation, marked broadening of the perineum and prominence of the trochanters in the gluteal region, with marked lordosis and a rolling, swaying gait that is characteristic.

The differential diagnosis must be made from,—

(*A*) Cases in which destruction of the head of the femur has occurred as a result of an acute destructive arthritis of the hip during infancy. The symptoms in later life are identical, but the diagnosis is important, as after the inflammatory destruction of the head reposition is generally impossible on account of the absence of any head or neck beyond a small excrescence, as happened to be demonstrated in a personal case of the writer's, when operation was undertaken in the hope of finding head enough left to work with.

(*B*) Coxa vara[3] or rhachitic bending of the neck of the femur may simulate dislocation, but the head of the femur is in the acetabulum and the joint is not unstable.

(*C*) The gait in severe infantile paralysis of the leg may simulate the gait of congenital dislocation, but the diagnosis is easy unless paralytic dislocation has occurred.

Prognosis.—Without treatment, as a rule, these cases become worse as they grow older. Adults are often so handicapped that they cannot perform manual labor, and inflammatory conditions may occur in the joints. Occasionally children walk better after reaching puberty.

Treatment.—The treatment of congenital dislocation of the hip is the most actively discussed question in the orthopædic surgery of to-day. The widest diversity of opinion exists, the widest disparity of results is obtained by various observers. The literature of the last five years is voluminous, and the writer of an article on this subject at present must leave much unsettled and must warn his readers that it is a time of transition and general distrust. Time enough has not elapsed since the announcement

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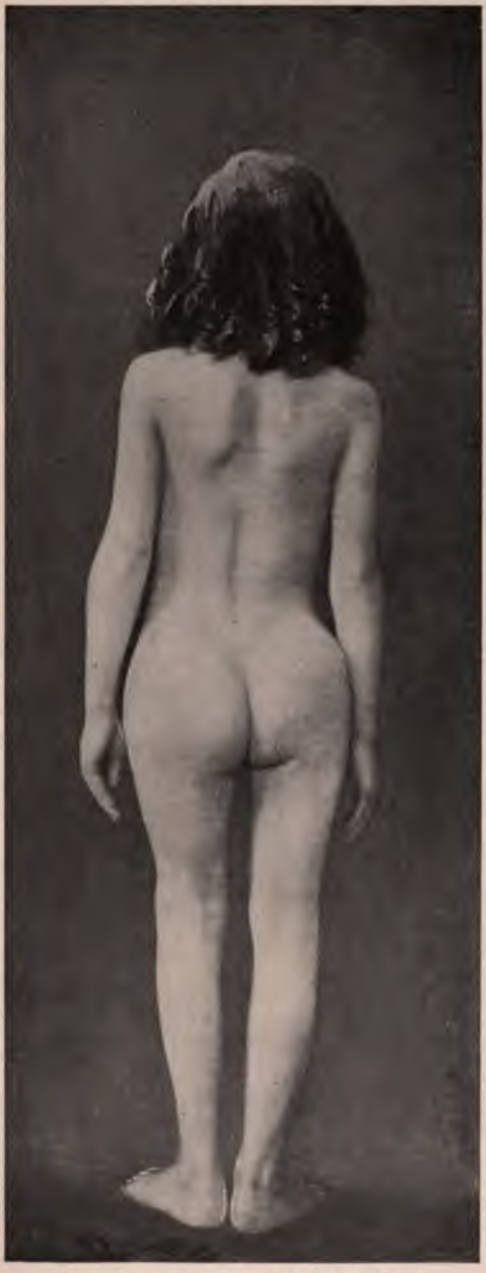
[1] Bradford, Transactions of the American Orthopædic Association, vol. vii.

[2] Saniton, De l'Anatomie de la Hanche chez l'Enfant, Paris, 1893.

[3] Transactions of the American Orthopædic Association, vol. vii. p. 27; Tubby, Orthopædic Surgery, London, 1896, p. 263; Ogston, recent paper.

FIG. 1.



Double congenital dislocation of the hips.

of Hoffa's operation in 1890 to allow the proper working out of so difficult a problem as that of the permanent value of operation.

The main lines of treatment may be considered under three heads:

1. Mechanical treatment.
2. Bloodless treatment.
3. Operative interference.

(1) The record here is, on the whole, one of failure of greater or less degree. Pravaz and Guérin half a century ago failed to convince their colleagues that they accomplished much by continuous extension, and much the same conclusions are held now by competent critics. At the Berlin Medical Congress the statement was made in the orthopædic section that no case could be found recorded in which, ten years after treatment by traction, a cure had been established. The famous case of Buckminster Brown so often quoted relapsed after it was thought that a cure had been effected. Bradford has contributed the final results of four cases carefully treated by extension, and it forms one of the most important contributions to the subject, as showing the real results years after treatment rather than results[1] reported after a short time,—too short to show their real value.

Bradford's cases were treated for years by traction and protection, and later by corsets, as described by him.[2] One girl seventeen years old could walk with little peculiarity of gait if careful, but it could not be claimed that the results were much affected by treatment. One fifteen years old was in no way affected by treatment. One walked better than if no treatment had been instituted. And in the last, in which at eight years of age the trochanters were in place after several years of treatment, at eighteen the trochanters were above Nélaton's line and the deformity characteristic.

Schede's method consists in traction at night and during the day the use of a protective appliance abducting the leg.

Mikulicz[3] reports good results from an apparatus holding the leg extended, abducted, and rotated outward. The children lie in this apparatus from eight to ten hours in the twenty-four, and for the rest of the time they move freely about. Mikulicz reports that up to the end of the second year a cure should result. Three single dislocations were reported wholly cured; treatment had begun at the age of four, thirteen, and fourteen months. In older children an orthopædic corset should be combined with this method. Eighteen cases are reported of single luxation; eight were perfect cures and began treatment before they were four and one-half years old. In ten the functional result is not perfect. The best comment on these results is Bradford's paper, already referred to.

Mechanical treatment, however, finds its place where other measures are not available, or where the children are too old for operation. It probably modifies the lordosis and may prevent the excess of deformity. Corsets with perineal bands which hold the pelvis tightly and press down upon the trochanters are much in favor in Germany, and certain forms of splint are advocated by certain writers. They are generally some combination of jacket and traction splint. Such splints are those of Hessing or the splint of Dolega, holding the leg abducted. Bradford advocates the use of a modification of the Thomas ring knee-splint with traction attachment. Of mechanical treatment it may be said that it is not likely to effect a cure or even any marked permanent improvement. Suitable apparatus may, however, mask or improve some of the secondary effects of the displacement.

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[1] Adams, Transactions of the American Orthopædic Association, vol. viii.

[2] Ibid., vol. iv.

[3] Archiv für klinische Chirurgie, Bd. xlix. S. 368.

(2) Methods of forcible reposition of the head of the femur into the acetabulum are much advocated by certain surgeons. Paci in 1888 advocated reduction under anæsthesia. First, the knee and thigh are both gently flexed; second, abduction is made until the knee is some inches outside of the flank; third, outward rotation is made until the leg becomes perpendicular to the long axis of the body; fourth, and finally, extension of the thigh is made, holding the pelvis firm, and then extension of the leg, maintaining the outward rotation.

This is followed by the use of a plaster of Paris bandage for a month, and by extension, which should continue four months; after four months, crutches and massage; after eight months, canes and an orthopædic corset.

The pathological condition does not lend support to the view that Paci's manipulation can be widely successful. It will be remembered that in these cases the acetabulum is undeveloped, the head displaced and too large for it in any event, the capsule stretched, and the muscles pulled out of their original place with impaired function.

Paci has reported twenty-two cases reduced by this method, in all of which he obtained good results.

Redard has been fairly successful by this method in single dislocations. Italian surgeons were most enthusiastic when Paci's method was announced, and ten were quoted as obtaining good results. Karewsky has not succeeded with it. English and American authors are silent except in the case of Post, where, so far as immediate results are concerned, forcible reduction under ether was successfully done fifteen years[1] or more ago, but the ultimate result was not a cure.

Lorenz's comment upon Paci's operation justifies the theoretical conclusion drawn from the pathology. After making the manipulation described by the Italian surgeon, he has opened the capsule, and, repeating the manipulation with the structures exposed to view, he has in no case succeeded in reducing the deformity. But it must be remembered that Paci's claim is not that complete reduction is produced, but only a pseudarthrosis.[2]

For young children up to the age of six or seven at most Lorenz has proposed a method of bloodless reposition under anæsthesia which is too new to be considered established, but which is reasonable, and in Broca's hands also has proved satisfactory so far as immediate results are concerned. He claims true reduction, which is met with scepticism by Kirmisson and others.

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[1] Abner Post, Boston Medical and Surgical Journal, 1883, vol. cix. p. 193.

[2] Kirmisson, Bull. et Mém. de la Soc. de Chir., 1896, t. xxii. p. 338.

1. The leg is pulled forcibly until the head is on a level with the acetabulum. This is done by a cloth sling around the ankle pulling against a perineal strap for counter-pressure, and by very strong traction, slowly increasing. The tip of the trochanter should reach or pass Nélaton's line.

2. The femur should be flexed to a right angle. This relaxes the capsule and places the head of the femur behind the acetabulum.

3. The femur should be rotated inward slightly, to turn the head of the femur towards the acetabulum pocket.

4. The leg should be then "abducted till an almost right-angled abduction is obtained." The head of the femur should be felt to jump over the posterior rim of the acetabulum. If the abduction is much diminished, the head of the femur again jumps out of the cavity. The more the abduction the greater the safety.

5. The leg is now fixed in this position by a plaster of Paris spica bandage down to the knee.

A high sole is put on the other foot and the child allowed to go about. Weight-bearing tends to bore out an acetabulum. The leg should be fixed in abduction for twelve weeks at least, and walking encouraged.

A critical estimate of these measures cannot be fairly made at present. In the hands of the inventors the results reported are excellent, but the general tone of recent literature is one of qualified scepticism and a desire to wait for the verdict of surgeons in general on these recently introduced methods. That[1] of Lorenz commends itself on scientific grounds, as the method of Paci never could do.

The method of Lannelongue[2] should be mentioned only to be classed among the failures.

After traction an attempt is made to retain the head of the femur by the inflammation excited by the injection of a solution of chloride of zinc around the joint.

(3) Of operative methods only two need be noticed at any length. Excisions of the head of the femur have been abandoned as routine measures, and the operations of Hoffa and of Lorenz are the ones now claiming the attention of the surgical world. These operations in the hands of the inventors yield marvellous results, as reported, but Hoffa's operation in the hands of certain skilful American surgeons has been largely unsuccessful. All this leads the careful observer to withhold his judgment on the general merits of these operations.

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[1] See the discussion at the Twenty-fifth Congress of German Surgeons, Cent. f. Chir., Beitr. z. Cent. f. Chir., 1896, No. 31, S. 177.

[2] Bull. et Mém. de la Soc. de Chir, 1896, t. xxii. p. 300.

Hoffa's operation consists in a posterior incision, as if for removing the joint. The capsule is opened after a dissection exposing it, and extirpated so far as it offers resistance to reduction. With a sharp spoon the rudimentary acetabulum is enlarged and the head of the femur replaced in it. The hamstring tendons and any other resistant structures are divided if necessary.

Surgeons, as a rule, have not been so successful with this operation as was the inventor.[1]

Hoffa reports one hundred and twelve cases with seven deaths (four from operation, one from diphtheria, one from pneumonia, and one from intestinal catarrh), eleven relapses, and nine ankyloses.

As a contrast to these are the American cases:

Bradford reports eleven operations performed by various surgeons at the Children's Hospital, Boston; no cure, three deaths (one from diphtheria, one from diarrhœa, and one from scarlet fever). In no case did improvement follow operation.

Gibney reports six operative cases with two deaths (one from diphtheria and one from sepsis), four relapses, two good results.

Lorenz's operation is more than a modification of Hoffa's in that recognition is made of the fact that the chief obstacle to reduction lies in the capsular ligament, especially its anterior part, rather than in the muscular tissues. The child should be subjected to five or ten pounds' traction about ten days before operation. At operation the leg should be abducted and an incision made downward from the anterior superior spine of the ilium in the direction of the outer edge of the tensor vaginæ femoris muscle. The fascia should be divided above and down to the anterior border of the glutæus medius. The capsule is exposed, the leg twisted outward, and the capsule thus stretched is divided in the direction of the neck of the femur until the ridge of the shaft is reached. The second division of the capsule crosses at right angles over the head of the femur. The head of the femur can then be turned out and examined. The acetabulum should be then deepened, and the head of the femur shaped, if need be, and replaced. Satisfactory reposition of the head of the femur in the acetabulum is essential, and dislocation should not recur at each attempt at manipulation of the leg. If so, the obstructing cause must be identified and corrected.

The limb is abducted and fixed by a plaster of Paris spica, without traction. Lorenz[2] reports brilliant success in over two hundred cases. He performs the operation in from ten to twenty-five minutes, but Kirmisson finds half an hour or more up to one and one-half hours necessary.

It is extremely difficult to estimate the value of operative measures. American experience in Hoffa's operation has been unfavorable, yet Anerer,[1] in reporting three successful cases, says, "With the immediate results f Hoffa's operation one can hardly be too much pleased." The mortality-ate is not high, nor is the operation one of great difficulty.

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[1] Bull. et Mém. de la Soc. de Chir., 1896, t. xxii.; Broca, thirty-seven cases, three deaths; Kirmisson, seventeen cases, two deaths; Bergmann (quoted by Hoffa), two cases.

[2] Quoted by Bradford, Boston Medical and Surgical Journal, vol. cxxxv, pp 9, 207.

Lorenz has a record of two hundred cases. In the first one hundred nd fifty, at least, he reports no death. Up to the end of August, 1894, here had been no failure.[2]

Such extraordinary results as these have not been elsewhere obtained, ut here and there successful cases operated on by this method have been eported.[3] Too little time has elapsed to permit the formation of any eliable critical opinion. The operation undoubtedly has a brilliant future.

Paradies[4] has described an operation applicable to cases too old for the orenz or Hoffa operation, by which he attempts to add to the stability of e joint by cutting down on the head of the femur, opening the capsule, nd removing some of the cartilage from the head of the femur.

The outcome of all this is that, given a young child with congenital islocation, if very young, the apparatus of Mikulicz might be tried. Be-ween two and four years, the bloodless reposition of Lorenz seems by all eans the most trustworthy. If this fails, operation by the method of orenz should be done. From five to ten years, operation is most likely to ucceed. After this, and especially after puberty, nothing seems to remain ut palliative mechanical treatment.

CONGENITAL DISLOCATION OF THE HIP.

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[1] Münchener med. Wochenschrift, April 10, 1894, S. 281.

[2] Mikulicz (Archiv f. klinische Chirurgie, 1894) reports a bad ultimate result in one f Lorenz's patients.

[3] Leser, Berliner klinische Wochenschrift, 1895, Bd. xxxii. S. 981 (twenty-nine cases); ottet, Presse Médicale, February 5, 1896, p. 61; Broca, Presse Médicale, March 11, 1896, 52; Whitman, New York Medical Record, September 12, 1896.

[4] Zeitschrift f. Orth. Chir., 1896, S. 258.

modifies the lordosis and may prevent the excess of
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felt in the course of a year or two.
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a result of the forward dislocation of the
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rarely and in cases with other congenital de-
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resumes the displaced position. This condition is
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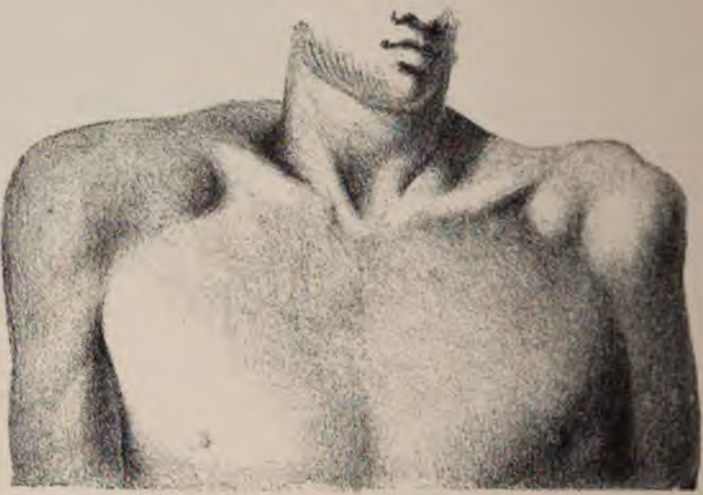
In these cases the attitude of the limb is that of
right angle or beyond. In the worst cases the toe of
groin. The condyles of the femur can be felt at the
space, and the patella, often small, may be absent.
and Krönlein.)

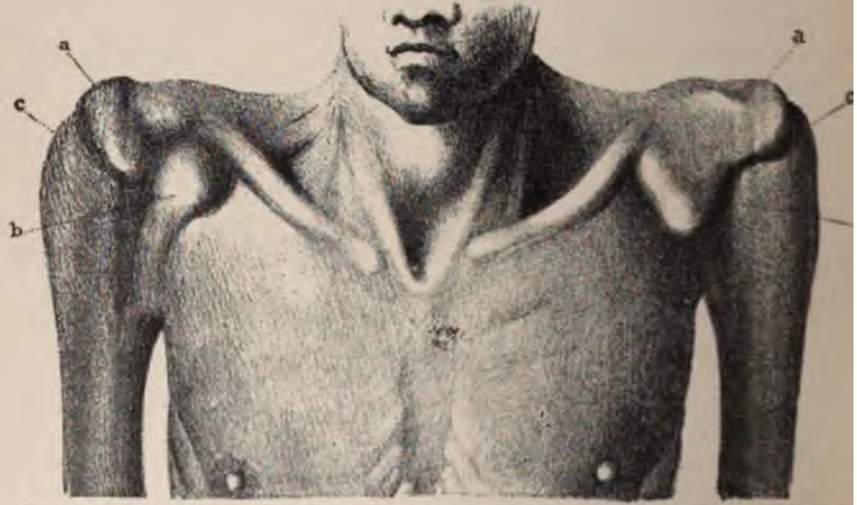
Lovett, Orthopædic Surgery, diagram, p. 525; Mason, New York
1877, vol. xii. p. 42; Hamilton, Fractures and Dislocations, p. 845;
Poliklinik zur Leipzig, 1888; Chaussier, quoted by Hamilton.

FIG. 2.



Subcoracoid dislocation of left shoulder. (Smith.)

FIG. 3.



Subacromial dislocation,—bilateral.—*a*, acromion; *b*, coracoid; *c*, head of humerus. View of patie
from front. (Smith.)

In only one case (Müller) was the absence of the patella anatomically demonstrated.

Lateral mobility is the exception, and, as a rule, is confined to the biateral cases where other deformities coexist. Abduction with eversion is he direction in which mobility is most marked. In a case of Krönlein's, however, there was ankylosis with anterior luxation of both tibiæ.

Pathology.—Pathologically the data are few. (Albert, Müller, and Wolff.) The articular surfaces in contact with the tibia may lie at the nterior part of the lower end of the femur. The condyles may be flattened (Wutzer), the articular facets on the femur may reach higher than normal (Albert), and the facets on the tibia may be small (Wolff). The patella may be rudimentary or absent. The ligamenta alaria have been found absent and the joint cartilages modified.

In Albert's case the vessels lay behind the outer condyle of the femur.

In Wolff's case the luxation in one knee was permanent and in the other could be produced and reduced at will.

Treatment.—With regard to the treatment of the posterior variety, a case has been reported by Hamilton where the hamstrings have been divided and reduction was made. In the discussion of Mason's case, Sayre spoke of having recorded a similar instance.

The forward dislocation of the leg is generally easily corrected by manipulation and the application of splints to the leg in a corrected position. The length of time that this treatment must be continued will depend upon the severity of the case. The excellence of the ultimate result depends upon the amount of deformity of the knee-joint. As no ultimate results have as yet been reported, the final outcome cannot be definitely stated. The immediate result is, however, as a rule, satisfactory without operative treatment.

Wolff found it necessary to replace by an operation a case of this sort n a five-year-old girl in 1887.

Phocas performed manual osteoclasis of the lower end of the femur in order to restore flexion.

Taylor reported a case where a double congenital dislocation of considerable severity showed a good result three years after treatment by braces was begun. In the cases of Bard and Youmans a good result was achieved with every prospect of permanency.

PATELLA.

Congenital dislocations of the patella are of three general types. It is denied by some writers that all of these are present before birth, but the evidence seems to be good that at least the majority are truly congenital.

The three forms are as follows:

1. Displacement of the patella directly upward, with lengthening the patellar tendon.

Zielewicz reports three cases which may or may not have been congenital. No other literature on this form could be found.

2. The second form of dislocation is outward, with absence or flattening of the outer condyle of the femur.

3. The third form consists in outward displacement of the patella, with approximately normal condyles.

In a number of the reported cases of outward dislocation the deformity was not reducible. Where the external condyle is lacking, and in certain other cases (South and Wolcott), the deformity disappears or becomes less marked in extension, reappearing in flexion. The disability from this deformity varies much: in some cases it is of little inconvenience, while in others the patient finds difficulty in going down stairs, especially in carrying weights down stairs; they are, however, as a rule, able to make forcible extension of the leg in going up stairs. Apart from the cases of Stokes, Smith, and Servier, where the external condyle is absent, Appel adduces data to show that a faulty development of the external condyle often occurs, and may be a predisposing factor. The filling up of the intercondylar groove which is noted in some cases is probably secondary. The outward rotation of the tibia which occurs is a mechanical result of the oblique pull of the quadriceps.

Treatment.—No specific treatment has been suggested for these cases, and the course to be pursued must be formulated on general surgical principles. In certain of the cases it would certainly seem that the operation recently proposed of shortening the capsule of the knee-joint might be performed with benefit.

ANKLE.

Congenital dislocations of the ankle are rare. In outward dislocation of the ankle the fibula is absent, at least in part.[1]

Inward dislocation of the ankle is marked when the tibia is absent in part. Here, again, the treatment must be upon general surgical principles.

SHOULDER.

Shoulder dislocations may be divided into two classes,—those in which the glenoid cavity is absent or imperfectly developed, and those in which it is approximately normal.

The first class has been well described by Smith.[2] In this case there was hardly a trace of the normal glenoid, but a well-developed new facet beneath the coracoid process, or beneath the acromion on the body of the scapula, with which the more or less altered head of the humerus loosely articulated.

The biceps tendon arose in each of the cases from a point in nearly

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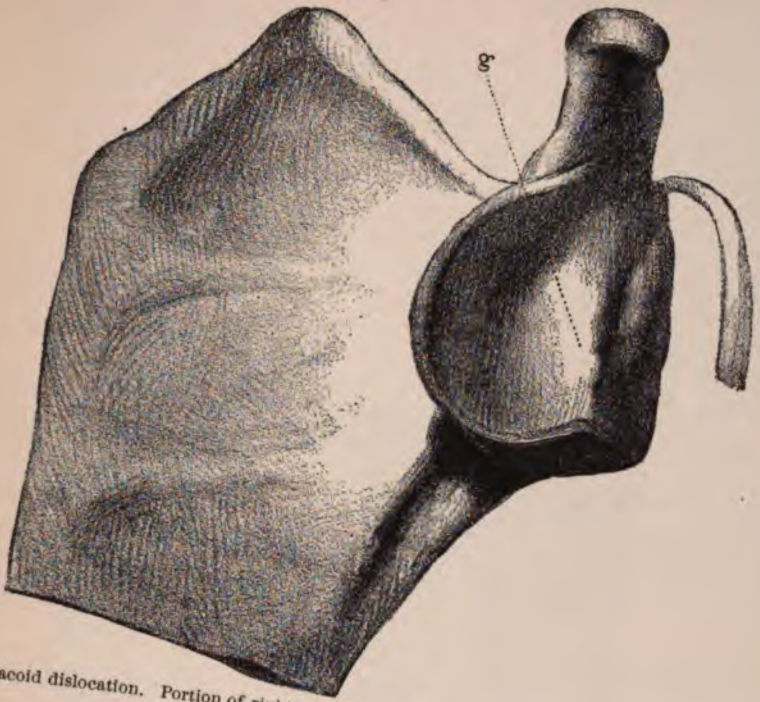
[1] Kraske, Beiträge zum Cbl. f. Ch., 1882, No. 29, S. 85; B. Mackenzie, New York Medical Journal, February 20, 1892.

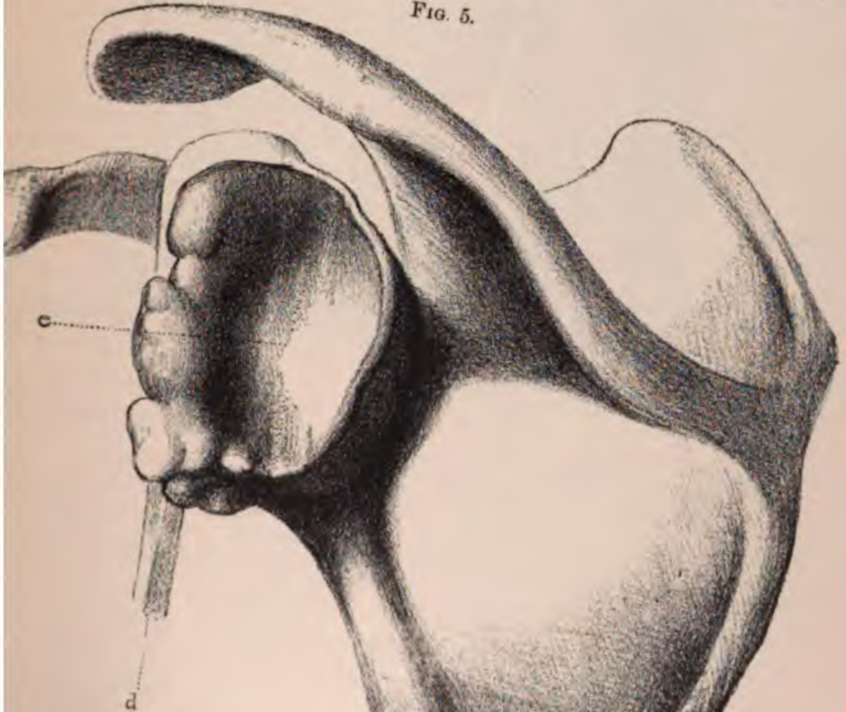
[2] Smith, Dublin Journal of the Medical Sciences, 1839.

FIG. 4.



Subcoracoid dislocation. Portion of right scapula.—*g*, abnormal socket directly under coracoid. Scapula from front. (Smith.)

FIG. 5.



ial dislocation. Left scapula.—*d*, tendon of biceps; *e*, abnormal socket on outer surf
neck of scapula. Scapula from behind. (Smith.)

the external condyle downward, perhaps due to relief from mutual pressure. Cases of this class are reported by Allen, Bergtold, Herskovitz, Phillips, and Symington.

Bessel-Hagen has excised the radial head in a case of this form resulting from overgrowth of the radius in partial giantism with good functional result.

2. Forward and upward dislocation of the radius.

Abbott, Mitscherlich, and Guérin have reported cases of this form. In these cases the head of the radius lies upward and outward from its normal position, articulating imperfectly with a new facet on the humerus; the articulating surface of the ulna is widened.

In one of Abbott's cases there was ankylosis of the radius and ulna in the upper part.

In Mitscherlich's case, as well as in other cases reported by Abbott, flexion was interfered with less than might have been expected from the position of the radius, but pronation and supination were much limited.

In Mitscherlich's case the whole elbow-joint was excised. The early death of the patient prevented any conclusions as to the value of the operation.

3. Backward dislocation of the radius and partial dislocation of the ulna with imperfect development of the internal condyle.

Abbott reports a case of this form where defective ossification of the internal condyle and trochlea had led to a separation of the trochlear portion, on one side complete, on the other incomplete. Dislocation of the radius backward, inward, and upward was present. Flexion and extension were much limited; pronation and supination only moderately limited.

4. Dislocation of both bones backward.

The only case of this form found is reported by Chaussier. This was definitely congenital and referred by him to muscular action *in utero*.

WRIST.

Congenital dislocations of the wrist are for the most part associated with bony deformity, and are classed as club-hand. Of congenital dislocations of the wrist pure and simple two instances may be mentioned.

One was the case of Wolff, where the wrist could be dislocated anteriorly by muscular action, and the other was that of Marrigues, where the two bones of the forearm were separated and the carpal bones lay between. These conditions possess no practical interest.

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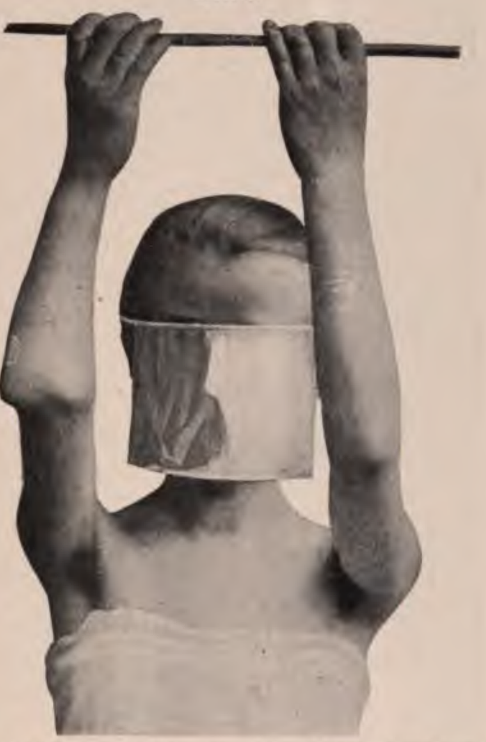
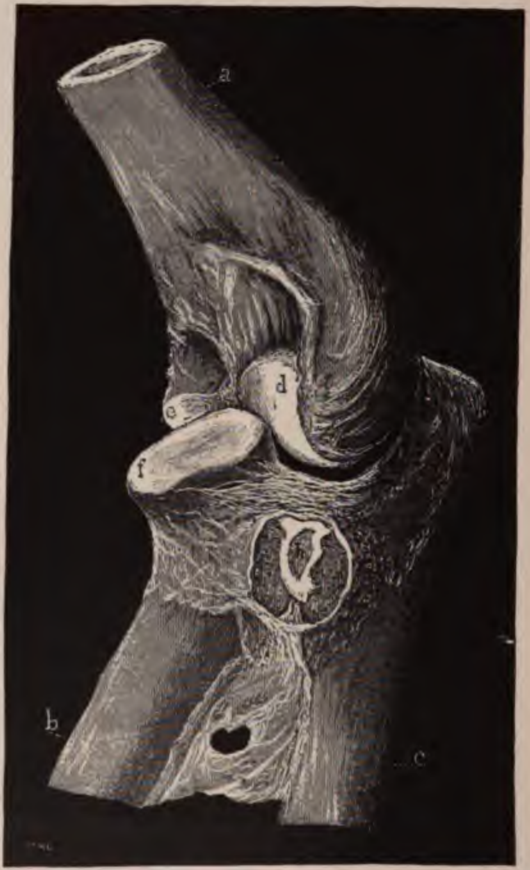
FIG. 7.



Dislocation of right radius in a girl of fourteen. (Bessel-Hagen, from Langenbeck's Archiv.)

FIG. 8.



Forward dislocation of radius,—left.—*a*, humerus; *b*, radius; *c*, ulna; *d*, capitellum; *e*, trochlea; *f*, head of radius; *h*, extra centre of ossification between radius and ulna. (Abbott.)

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ACUTE INFLAMMATION OF BONE.

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Acute periostitis, acute infective periostitis, acute osteomyelitis, acute necrosis, acute epiphysitis, acute subperiosteal abscess.

The above, with others, are names given more or less correctly by different writers to the disease which has been already so fully described by Professor Macewen in this work (vol. iii. p. 992) that little remains to be added to his article. We may, however, consider one or two points not specially dealt with by him, and for the rest refer to his article.

Etiology.—As to the predisposing causes of acute periostitis, epidemic influenza must be added to the list of specific fevers which may be followed by acute bone-inflammation. We have seen the fibula attacked after influenza.

Pathology.—That the inflammation in these cases may begin either in the deep layer of periosteum,—*i.e.*, in the tissue between the fibrous layer and the compact bone,—in the medulla, or at the end of the diaphysis at its junction with the epiphyseal cartilage is fairly certain, though it is not always easy in an individual case to say which has been the starting-point. But it may also begin in the cancellous tissue of an epiphysis or in the superficial layer of periosteum which connects that membrane with the rest of the fibrous tissues of the part, and the resulting abscess has been called parosteal or supra-periosteal.

Parosteal abscess seldom gives rise to necrosis, since it spreads usually in the line of least resistance,—*i.e.*, towards the surface,—but sometimes the periosteum melts away, the bone is exposed, and the inflammation gains access to the subperiosteal area. Nothing more than local exfoliation, as a rule, results from this, though implication of an adjacent joint may occur. It is then a less severe and acute disease than the worst cases of the subperiosteal form, but its treatment by free incision is the same.

While this short article must be considered wholly as supplementary to Dr. Macewen's, and in no sense as attempting to cover the whole ground of the disease, it is perhaps well to point out that a great deal of unnecessary complication has been imported into many of the text-book descriptions of acute bone-inflammation. The very multitude of names applied to the same disease shows what confused ideas have existed on the subject.

If it is remembered that periosteum and medulla, whether of diaphysis or epiphysis, of medullary cavity or of cancellous bone, form one continuous structure connected by bands or columns of tissue penetrating the compact bone by way of the Haversian canals, it will be clear that it is impossible to draw a hard and fast line between acute periostitis and acute osteomyelitis. The two should be considered as pathologically one disease. Given the presence of the necessary micrococci, the exact locality in which the mischief begins and the extent to which it spreads depend upon the existence of an area deficient in resisting power from injury, general disease, exposure to cold, or failing blood-supply from whatever cause. The weak spot may be in the periosteum, in any part of the medullary cavity or of the cancellous tissue at the end of the diaphysis, at the epiphyseal line or in the epiphysis, and the extent and progress of the disease depend simply upon the capacity of the tissues to resist the growth of the organisms. There is no inherent difference between the varieties of the disease, and the writer who insists that one name alone should be given to it—*e.g.*, that all these cases are examples of osteomyelitis—is just as much right and just as much wrong as he who is similarly insistent that all are primarily cases of periostitis. The inflammation may be the one one day and the other or both the next.

The important point is to recognize that as the extent of the disease is different in different cases, so the treatment must differ. In the one, free incision of periosteum is sufficient; in the next, it may be imperative to clear out the medullary cavity.

It is, we think, easier to understand the whole subject of acute inflammation of bone if this view of it is taken than it is if one chapter is devoted to acute periostitis, another to ostitis or osteitis, and a third to osteomyelitis, as if they were separate and independent diseases.

Dr. Macewen has admirably pointed out the difficulty, in some cases, of finding the site of a local acute periostitis, especially in those where the patient is delirious or semi-conscious only, and he mentions a case in which the neck of the femur was attacked and this difficulty arose. We have had recently a case of acute suppuration in the hip-joint in which the symptoms in character and severity most perfectly resembled those of a very acute periostitis. The escape of half a drachm or so of pus from the joint, on making an incision through the capsule and synovial membrane, relieved the symptoms, and the child recovered. We have also had a fatal case of localized acute periostitis of the neck of the femur.

Of the rarer sites for this disease, the spine, the ilium, the os calcis, of which Owen has lately recorded an example, a metatarsal bone, and the skull may be mentioned. The femur and tibia are probably most commonly attacked.

It is usually only in cases which are so severe as to be fatal that joint-invasion or multiple bone-lesions (probably pyæmic) are found, but in the case related below, the knee by direct extension, and the lower jaw, th humerus, and the opposite femur by pyæmic infection, were attacked, and yet the child recovered.

As illustrating two somewhat unusual complications, fracture and hemorrhage from ulceration into a large artery, as well as the connection which we believe exists in some cases between acute periostitis and tubercle, we may mention the case of a lad whom we saw with acute periostitis of the upper part of the humerus. There was a large abscess, which was opened, but a few days later severe bleeding came on. It became necessary to open up the axilla freely to find the vessel, and in lifting the arm gently to expose the armpit, the humerus snapped across. The arm was amputated, saline transfusion was employed, as there had been great loss of blood and the lad was almost dying, and he recovered. The bleeding was apparently from the subscapular, or possibly from the axillary itself.

Some years later, writing from memory, the boy, who had quite recovered, was seen on account of a swollen toe which was evidently tubercular. He was ill and breathing rapidly at the time of the examination, and, as it was suspected that there was acute general tuberculosis, no operation upon the toe was advised, and in a few weeks he was dead of general tuberculosis. (*Vide* also article on Tuberculosis of Bone.)

Fracture in later stages from deficient development of new bone around a separated dead shaft is not uncommon, but it is a rare though by no means unknown event for the bone in an acute stage to be so rapidly disorganized as to give way with no more force than the raising of the arm.

Treatment.—As regards the treatment of these cases, the need of free and early opening is fully insisted upon in Dr. Macewen's article. We do not think it is a good plan to explore freely with the finger after opening the abscess. No good can result from it which will compensate for the risk of breaking through vessels and stripping up periosteum not yet detached, and so still further endangering the life of the bone. The plan of freely opening up the bone at once and scraping out the medulla, just as in osteomyelitis after amputation, is not necessary in the early stages, but may well be employed where incisions have failed to arrest the disease and on gouging or trephining the bone the medulla is found to be involved. So also later, when, if necrosis has occurred and sequestra require removal, it is often necessary thoroughly to clear out the whole medullary cavity of a long bone. Much has been written of late in support of the treatment by immediate trephining by those who believe that the disease is always primarily an osteomyelitis, but, though such treatment is far better in any case than delay or insufficient incisions, we cannot admit that it is necessary in all instances, since cases undoubtedly recover after incision of periosteum only, though we are bound to acknowledge that in the majority some necrosis follows. The fact that the flat bones of the skull and the ribs and the laminæ of the spine may be attacked further proves the existence of inflammation apart from an epiphysitis, though the neighborhood of an epiphyseal line is usually involved. It is very difficult to be sure of the exact starting-point, and it may be that a primary inflammation in the vicinity of the epiphyseal line is the more common lesion. However this may be, it is certainly not in all cases necessary to do more than incise the periosteum.

Amputation is seldom called for in the acute stages of the disease, though it would possibly check the course of pyæmic infection in some cases. Too often the infection is so rapid and severe that the child dies before the local condition is such as seriously to raise the question of amputation.

In making incisions to set free the inflammatory material in the acute stages the openings should be free, but multiple, and on different aspects of the limb, if possible, rather than one long, single incision; and for the same reason, in removing sequestra, especially of the tibia, it is wise, a few days after the dead bone is removed, to bring together the edges of the wound as far as possible, and so to minimize the subsequent scars. Much trouble often arises from the large, tender, thin scar which so often occupies nearly the whole length of the tibia after operation in these cases. The slightest injury in after-years is apt to cause troublesome ulceration, and the occurrence of malignant disease in the cicatrix is no imaginary complication.

It is well, also, in removing large sequestra, not to chisel away completely the sheath of new bone, but, cutting through it on one side, to turn it back as a hinge, as it were, and after removal of the dead bone again lay down the flap of living bone. In this way a stronger limb and more speedy healing and filling up of the cavity are likely to be got. No violence should be employed in chiselling or removing sequestra, as fracture is very liable to occur in cases of extensive necrosis, and may be followed by non-union, requiring subsequent wiring or other plastic operation.

Primary resection of the diaphysis in the acute stage of this disease is a plan which must be considered in most cases wholly unnecessary and likely to result in a weak and distorted limb from deficient re-formation of bone.

As regards the subsequent usefulness of the limb in these patients, it must be remembered that in some, where the growing line has been encroached upon by disease or obliterated by premature ossification, there may be arrest of growth of the bone and resulting deformity. In others a converse overgrowth of the affected bone may occur as a result of the increased blood-supply brought to the uninjured growing line in consequence of the inflammation near it.

In the rare cases of acute periostitis of the larger flat bones, such as the ilium or scapula or cranium and in the spine, it must be remembered that pus may be poured out beneath the periosteum on both aspects of the bone, and it may be necessary to perforate the bone freely to empty the cavity on the deeper surface, or, in the case of the ilium, this may be attempted by working down on the inner side of the pelvic brim, though probably gouging or trephining will better provide for drainage.

The occurrence of late abscess in the interior of bones which have years before been the seat of acute inflammation must not be forgotten. We have met with many such cases, and as long as thirty years after the original attack an abscess may form either as the result of some latent focus of disease, which has been dormant till an injury or failure of the individual's health has renewed its activity, or because a slowly progressive sclerosis has at last so cut off the blood-supply as to starve some area of bone and lead to its death.

It is probable that most of the cases of chronic abscess of bone which are not due to tubercle or syphilis or some of the rarer sources of irritation (actinomycosis, etc.) are examples of a more or less acute inflammation, such as that described in which the resisting power of the tissues has been sufficient to circumscribe the spread of the organisms and diminish their virulence.

Special mention must be made of those cases in which acute inflammation has attacked the popliteal surface of the lower end of the femur. Probably because its blood-supply is somewhat scanty, this area of bone is frequently inflamed, and, unless treatment is early and thorough, a most troublesome condition often results. The bone appears to be unable to recover completely, and at the same time it does not become completely necrosed and detached. Long-continued suppuration with sinuses which perhaps close for a time and periodically reopen is commonly seen. Often attack after attack at longer or shorter intervals occurs with more or less pain and fever, and in many cases the patient becomes a chronic invalid, with a limb useful, it is true, for walking, but a great source of trouble and danger. Extension of the mischief to the knee-joint or upward to the shaft of the femur is apt to occur sometimes years after the original inflammation, and not a few of these cases fall victims to chronic or acute septicæmia in later life.

Exploration of the bone usually reveals a hard bare surface with no definite sequestrum and no line of demarcation making the removal of the disease practicable. We have seen so many of these patients in whom it has been necessary to amputate the limb after years of intermittent suffering that we are inclined to think that, where this chronic condition is once established and a fair trial has been given unsuccessfully to milder measures, amputation is the wiser course. It is possible in some, by very free exposure of the bone, to cut away the diseased area, but in children this would usually mean arrest of growth of the limb to such an extent that it would not be worth preserving.

Acute Epiphysitis.—Mention has been made of the occurrence of localized abscess in the epiphyses and shafts, but to complete this brief and supplementary survey of acute inflammation of bone we must mention the cases which, occurring usually in the first year of life, have been described as acute suppurative arthritis of infants and as acute epiphysitis. For the recognition of the disease we are indebted to Mr. Thomas Smith, though since his paper many cases have been recorded by Poland, the present writer, and others. An acute inflammation of the ossifying centre of an epiphysis, usually spreading more or less rapidly to the adjacent joint and leading to extensive abscess-formation, is the main feature of the disease. The onset may be acute or subacute; often there is a history of swelling and pain in one or two joints before any very definite local mischief becomes established in a particular joint. Pyæmia is a common and usually fatal complication, yet a fair number of these babies recover after free incision and drainage, and that with surprisingly good joints. As a rule, the joint is involved by extension from the epiphysis, and this is almost invariably the case in the hip, in which the whole epiphysis is intra-articular. Occasionally the pus finds its way to the surface without entering the joint.

A similar condition may be met with in older children. Free incision and drainage with abundant nourishment and stimulation are the essentials of treatment. Arrest of growth of the limb may follow, and in cases where the hip has been attacked the ultimate condition closely resembles "congenital dislocation."

This acute epiphysitis of infants in its early stages is often overlooked or mistaken for other ailments. Thus, it bears some resemblance to the congenital syphilitic affection of the ends of the diaphyses, it might be readily confounded with "scurvy rickets," or possibly the pain and disinclination to move the limb might be attributed to ordinary rickets. Like its ally, acute periostitis, it is sometimes considered to be erysipelas or rheumatism. The age of the child, the severity of the local symptoms, and the great disturbance of health in the acute stages, together with the definite implication of one or two joints, usually the hip or knee, will suffice to lead to a correct diagnosis when the liability of infants to this particular disease is kept in mind.

A fairly typical case may be briefly given. Samuel C., aged nine months, was admitted to hospital for an abscess, in the neighborhood of the left hip, of one month's duration. No cause was known for the disease. The abscess was opened by the resident medical officer, who found no direct communication with the joint, and no intra-articular grating was felt. The mischief did not subside, and the joint was subsequently opened up by the present writer, who found the head of the bone gone. Much pus escaped at the time, and, though the child seemed fairly well, it showed no power of repair, and ultimately sank and died. At the necropsy much thickening was found all round the hip-joint. There was an opening at the upper part of the articulation; the head of the femur was completely destroyed, only a stump of neck remaining. The surface of the acetabulum was covered with granulation tissue.

Vertebral Osteomyelitis.—Acute osteomyelitis of the vertebræ is occasionally met with, and is of extreme importance from the difficulty of its diagnosis and its very high mortality. A collection of twenty-one cases by Makins and Abbott[1] shows that any of the vertebræ may be attacked, and that the body or the laminæ or a transverse process may be inflamed.

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[1] Annals of Surgery, May, 1896.

There is the greatest danger of extension to the spinal meninges, and pyæmia is very common (sixteen of the twenty-one cases died). The depth at which the bone and abscess lie, the obscurity of the symptoms, the likelihood of attention being drawn off speedily to other foci of infection, and the similarity to the symptoms of other diseases have prevented the recognition of the nature of the lesion in an early stage in many of the cases. Free incision and perhaps removal of the lamina, to prevent the collection of pus within the spinal column and the development of meningitis, are the main lines of treatment.

A fatal case of inflammation of the occipital is also recorded in this paper, of which the title is an instance of the need for revision of our nomenclature of bone disease. Unless we are to abolish altogether such a term as periostitis, it surely would be more applicable to some of these cases at least.

The lumbar spine appears to be the most common seat of inflammation. Nineteen of the twenty-one cases occurred in children under fifteen years.

A paper by Chipault in *La Gazette des Hôpitaux*, December 12, 1896, of which an abstract by F. T. H. Coutts will be found in the *Medical Chronicle*, January, 1897, deals with a series of cases of this sort. Besides the general symptoms due to septic absorption, local pain, rigidity of the affected part of the spine, tenderness on deep pressure, and swelling may indicate the seat of mischief. Symptoms due to implication of the spinal cord may be present, but these are apt to mislead rather than assist the diagnosis, unless the other evidence is present.

Local Periostitis.—Acute localized periostitis is fairly common in children as a result of injury. A boy gets a kick on the shin at foot-ball, and an immediate swelling from extravasated blood follows; in a few days, perhaps, instead of the leg getting better, a localized but painful swelling is found over the front of the middle of the tibia; there is little, if any, fever, and the mischief is purely local; a few days' rest and the use of lead lotion bring the trouble to an end, though for a considerable time there may be some thickening and tenderness over the bone. This is, of course, a very different type of disease from the terrible cases of "acute necrosis," but all stages between the two may be met with, and neglect of a slight case might lead to the development of the more severe form.

An acute inflammation of the bone is also commonly enough seen in the jaw as the result of alveolar abscess from a carious tooth. This may be nothing more than an ordinary gumboil, or, on the other hand, it may lead to extensive destruction, though wide-spread necrosis of the jaw is more often a chronic than an acute condition. Even when a large area of jaw is laid bare by acute suppuration following an alveolar abscess there is usually little necrosis. It is well also to remember that in pyæmic periostitis it by no means follows that bone that is bare will die; it is remarkable how readily pyæmic lesions of bone and joints often recover, and that sometimes without even an incision, though if pus is present it should undoubtedly be always let out.

Acute Inflammation of Tibia; Pyæmia.—Arthur B., aged seven years, fell and struck the left knee on November 5, 1896. The skin was not broken. On the 13th the knee began to swell, and he was admitted to hospital on the 17th. There was then considerable swelling of the leg below the knee, the skin was dusky and œdematous, and there was a collection of fluid in the popliteal space. The temperature on admission was only 99.2°. The popliteal abscess was opened, but in the evening the child was delirious and his temperature 104.2°. The next day incisions were made over the upper part of the tibia, which was found to be bare. The temperature fell to 100.2°, but rose to 102° on the 20th. On the 22d the knee-joint contained pus, and was opened and washed out. On the 24th an erythematous rash appeared all over the body, and for the next few days the child was very ill. On December 1 the knee-joint was opened up still more fully. On the 2d there was severe pain in the leg about the ankle, which was swollen; the joint was explored, but no pus found. An abscess was opened over the lower end of the fibula; the bone was bare.

The parents were advised to allow amputation of the limb, but preferred to risk the child's life. On December 4 an abscess was discovered beneath the left deltoid, and the margin of the bicipital groove was found to be bare. The boy took food well most of the time, but was not fully conscious. Fresh abscesses were opened in the leg on the 7th. There was slight diarrhœa. The temperature ranged from normal to 103°. There was no albuminuria or chest trouble except a little bronchitis.

On December 10 a cultivation made from pus taken from the leg proved to consist entirely of staphylococci; no streptococci were present. On the 11th ten cubic centimetres of antistreptococcin were injected. No marked change resulted. The injections were repeated on the 16th, 17th, and 18th, twenty cubic centimetres on each occasion being used. It was not thought that any material change resulted from the injections.

There was no great alteration in the boy's condition, but the temperature ranged somewhat lower after December 20. He took food fairly well, but was much weaker. The wounds in the leg began to look better. On the 30th an abscess was found over the ramus of the lower jaw on the left side, which did not increase during the next three days.

On January 4, 1897, an incision was made along nearly the whole length of the tibia. The shaft was quite firm and in part covered by new bone. The medullary cavity was opened up and pus and small sequestra cleared out. The abscess over the jaw was opened and the bone found bare. The new bone in the leg was turned back, hinge-fashion, and none removed that could be saved. On the 7th the edges of the wound in the leg were strapped to bring the new bone into its place. The boy improved both physically and in intellect. On the 13th the wound over the jaw had healed. Subsequently swelling and pain appeared at the upper end of the right femur, but the acute symptoms subsided without operation, leaving, however, much thickening. He steadily improved, and though, of course, his wounds are yet unhealed, he appears to be convalescent at the time of writing, February 21, 1897.

The case, reported from notes by Mr. Izard, resident medical officer, is given as fairly typical of the course of a severe attack of inflammation of the tibia with pyæmia, but, fortunately, with no visceral complications, and hence, probably, his recovery.

TUBERCULOSIS OF BONE.

Pathology and Pathological Anatomy.—An acute miliary tuberculosis of bone certainly occurs as a part of a general tuberculous infection, and is probably much more commonly present than is supposed, since the presence of tubercles in the medulla in the early stages of the disease gives rise to no obvious symptoms and the patient dies before further evidence of bone-infection is shown.

Acute miliary tuberculosis of bone, then, is a matter of general pathological rather than of clinical interest, and is, moreover, not peculiar to childhood and need not be further discussed here.

Tuberculous disease of bone, in which the mischief has no doubt begun in the formation of one or more small foci which have given rise to more or less acute inflammation around the tubercles and more or less rapid degeneration of the deposited material, is exceedingly common in children. The rapidly growing, highly vascular, and unstable tissues, especially in the neighborhood of the growing lines of the bones and the articular surfaces, are very suitable for the development of tubercle, and it is in these sites that it is chiefly found.

The liability to injury common to all children is of importance, but it must be remembered that injury to the bone of a child will be followed by tuberculosis only if that child is made of the special material which is suitable for the growth of the bacillus,—that is to say, that either because its tissues contain some material favorable to the growth of the organism or because they are deficient in some material hostile to the development of the bacillus, one child is attacked by tuberculous disease of bone after an injury which would cause nothing but momentary pain to another child.

Though we have said that acute miliary tuberculosis of bone is of little clinical surgical importance, it must be remembered that a tuberculous disease of bone may be very acute and require active local treatment without any general infection of other organs. Thus, a child with perhaps tubercular disease of the epiphyseal line (tuberculous epiphysitis) may in a few days or a week or two be found to have a similar condition at some other point, tibia or radius or other long bone, and a series of foci in several bones may rapidly appear with or without evidence of tubercle elsewhere.

Again, we are disposed to think that some of the cases of so-called acute infective periostitis or osteomyelitis are tubercular from the first; they certainly develop all the features and run the course of tuberculous lesions afterwards; and though it may be said that the tubercle is grafted into the tissue injured by the previous inflammation, there is at least a possibility of the tubercle being the cause of the whole mischief.

While the epiphyseal line or bone adjacent to it is by far the most common starting-point of tuberculosis in bone, the periosteum and superficial layers of the shaft may be attacked at any point in the length of a long bone, and, on the other hand, the cancellous tissue and medulla may be extensively involved without implication of an epiphyseal line. So, too, the short and flat bones are often the seat of tuberculous disease, the ribs are not uncommonly attacked, and the disease may extend to the pleura and cause an empyema. Occasionally the sternum is affected, but in our experience less often in children than in adults. The carpal and tarsal bones are very commonly the subjects of tuberculosis, but usually the disease spreads from the synovial membrane to the bones, which are thus secondarily affected. The exception to this rule is the os calcis, in which the disease commonly attacks the epiphyseal line and spreads into the bone and from it to the adjacent joints. As to the bones of the skull, tuberculosis of the upper jaw and malar bones is common; the temporal is often diseased as a result of tuberculosis of the middle ear. The lower jaw is sometimes attacked by disease, the parietals and occipitals very rarely, the frontal more frequently, though by no means commonly. In the bones of the vault of the skull tuberculosis, when it occurs, is often circumscribed and leads to perforation of the cranium. Tuberculous disease of the spine is dealt with elsewhere.

In the pelvis the disease is by far most commonly situated in the acetabulum, beginning in the triradiate epiphysis and spreading from this centre to the bone around. Under such circumstances the hip-joint is necessarily involved, and often the disease, which is primarily in the femur, spreads to the joint, and, if not arrested, attacks the acetabulum secondarily by extension. Tuberculosis of the sacrum may be primary or the result of extension from the sacro-iliac joint, which is not very rarely the seat of the disease in children. Sometimes the pelvis is very extensively affected.

The clavicle is rarely attacked, but we have met with disease of the acromial extremity, either primary or as an extension from the acromio-clavicular joint. Disease of the scapula is usually limited to the neighborhood of the glenoid cavity, and is secondary to mischief in the joint, which itself is often the result of tubercle in the upper end of the humerus. The fingers and toes seem specially prone to tuberculous disease, so-called "strumous dactylitis."

The physical characters of tuberculous disease of bone vary somewhat in different cases. Thus, in some instances a superficial erosion (caries) of bone is found in which there is a more or less well-defined shallow pit filled with granulation tissue, caseous detritus, and perhaps small particles of dead bone (caries, caries necrotica).

In other cases a well-defined cavity may be found occupying part of the epiphyseal line and adjacent epiphysis and shaft, in which lies a sequestrum surrounded by caseous detritus. The sequestrum may preserve its form, and show on one aspect the natural surface of bone which originally was covered with the epiphyseal cartilage.

Again, the sequestrum may be eroded and so soft and broken up that its origin is unrecognizable. The most favorable cases are those in which the disease is, as described, well defined and the walls of the cavity are composed of sound bone. In other instances large tracts of bone without any distinct line of demarcation are pale, fatty, and rarefied; the bone is soft, often almost wholly absorbed, and it is difficult or impossible to say where actual disease ends and mere atrophy begins.

In others, again, the medulla for a quarter or half the length of the bone is filled with yellow or greenish pus and caseous material with sequestra of varying size and consistence, and with no line of demarcation between the healthy and the tuberculous bone. Yet in others a cream-like or semi-solid, whitish substance like soft, decolorized blood-clot may fill the medullary cavity and replace the cancellous tissue.

In the short bones the whole of the cancellous tissue may be soft, fatty, rarefied, and easily scraped away with a sharp spoon or crushed by pressure of forceps, so that little but an oily mass with a few osseous spicula may represent one of the carpal or tarsal bones.

In certain cases, notably in the fingers and toes, it is common to find part or nearly the whole of the shaft of the original phalanx or metacarpal lying loose as a sequestrum in a more or less spacious cavity, of which the walls are new bone, and the contents, besides the sequestrum, caseous granulations and débris.

We so often find multiple bone and other lesions in tuberculous children—perhaps a pulpy knee with disease of the lower end of the humerus on the other side, tuberculous dactylitis, a corneal ulcer, and disease of an ankle or one or more tarsal joints, and perhaps glandular disease as well—that we have become accustomed to speaking of these children with multiple tuberculous foci as cases of "general surgical tuberculosis," and in such cases most of the varieties of local tuberculous manifestation may be found. Bacilli are usually few and difficult to find in the discharge or the diseased parts removed by operation, though sections of such bone will show well-defined tubercles scattered among the ruins of the trabeculæ of cancellous tissue.

When once a tuberculous deposit has been formed in a bone it is impossible to say what the course of the disease will be. No doubt the process may be arrested and a practically complete recovery take place, or the vitality of the affected tissue may be so far destroyed that local death (necrosis) occurs. When this is so, if there is little irritation set up and little reaction in the surrounding tissues, there may be but little evidence of disease and no collection of fluid or "abscess"-formation. Such are some of the cases of "caries sicca" and "quiet necrosis," the difference being simply one in tuberculous cases of the extent to which death of the part goes. When, however, there is much outpouring of fluid and breaking down of tissue a so-called "abscess" is formed. In some cases there is formation of a true abscess; in others the fluid is serum, holding in suspension caseous material derived from the disintegration of tuberculous matter and dead tissue. In the soft bones of children destruction is naturally more rapid and extensive than in older people, and, on the other hand, repair is more complete and new bone-formation is often very abundant. It is, however, well to bear in mind that, while abundant new bone-formation may take place about the shaft of a long bone in which there is a tuberculous deposit, this growth of new bone is much less abundant and often deficient in the short and flat bones, and is usually absent altogether about the articular extremities until active disease has ceased and repair is going on.

This is important to remember, since the thickening about a joint is often spoken of as an enlargement of bone and taken as evidence of bone disease. The enlargement is simply of the soft parts covering the bone, and is no index of the extent of bone disease nor indicative of repair.

The so-called "expansion of bone," "spina ventosa," which is so evident in tuberculous disease of the phalanges, as well as elsewhere, is an example of destruction of bone, with new formation of periosteal bone around the diseased part. The new layer is again absorbed and replaced by fresh bone, and the process goes on till a large cavity, containing the remains of the original bone, with caseous material and granulations, is found surrounded by a thin layer of young bone formed in the way described.

Symptoms and Diagnosis.—A diagnosis of tuberculosis of bone in a child may usually be made if swelling involving the extremity of a long bone comes on slowly without great pain and with no tendency to rapid formation of abscess. If there is a tubercular family and personal history, or the child has the appearance indicative of a tendency to tuberculosis, or if the disease has, in a child apparently healthy, followed gradually measles or whooping-cough, the case is pretty certainly tuberculous. Chronic simple or traumatic bone-lesions are comparatively rare in children. Pyæmic bone disease is either acute or marked by a general failure of health and tendency to early suppuration not usually met with in tubercle. In congenital syphilis there is generally unmistakable evidence of the taint elsewhere, and while in infancy the ends of the diaphyses are affected, there is a more rapid arrest and a more speedy yielding to treatment. The evidence of the rash, snuffles, and general appearance prevents mistake in most instances.

A warning must be given as to the possibility of failure of treatment in cases where both syphilis and tuberculosis are present. We believe that some of the more intractable cases are unsuccessfully dealt with because the disease is a mixed one, and it must be remembered not only that it is very difficult sometimes to distinguish between tubercular and syphilitic lesions, but also that children often require very large doses of iodide to produce an effect. The chance of mistaking cases of actinomycosis for tubercle should not be forgotten; it is by no means improbable that the mistake is not very rarely made. If suspicion is aroused, the microscope will usually speedily settle the question, and energetic local treatment, with administration of iodide of potassium, will give the best prospect of cure.

Acute rickets, especially when accompanied by hemorrhage ("scurvy rickets"), is sometimes mistaken for acute tuberculosis, pain on moving a limb, with swelling in the neighborhood of joints, and failure of health leading to a supposition of the presence of tubercle. The general appearance of the child, the evidence of rickets, the acute onset, and the history of the upbringing of the child will suffice to clear up the case.

Occasionally much difficulty arises in the diagnosis of sarcomata of bone in children in their early stages. As these tumors are usually of very rapid growth, the doubt is likely to be felt only at the outset. Since tuberculosis is infinitely more common than sarcoma, the probabilities in a given case are all in favor of tubercle; but this is not sufficient. Sarcoma often, tubercle rarely, attacks the bones of the skull. Sarcoma growth is more rapid than tuberculous deposit, and enlarged veins coursing over the surface of the swelling suggest great vascularity. Sarcoma often follows injury, but so may tuberculosis. Tuberculous foci are often multiple, but there may be secondary deposits of sarcoma. In such cases, however, there are often failure of health and evidence of visceral new growth. Sarcomatous swellings attain a greater size than tuberculous ones and do not soften into "abscesses." Sarcomata are usually better defined than tuberculous foci, and, if they are central in origin, may cause "expansion" of bone, with the feeling of "egg-shell crackling," which is rarely found in tuberculous disease. Family history is of importance, but in any case of real doubt the right course is to cut down upon and examine the swelling, when the difficulty can usually be speedily disposed of.

Treatment and Prognosis.—The treatment of tuberculosis of bone in children must be based on general surgical principles, good hygiene and local rest being the main features of treatment in the early stages. Thus, careful feeding, fresh air, tonics of one sort or another, with attention to the digestive organs, are the main features of the general management, while the application of an efficient splint and of soothing lotions is all that is necessary locally. Should the mischief go on, or if it has lasted for more than a few weeks when first seen, a trial may still be given to splints and to inunction of oleate of mercury. If, in spite of treatment, the swelling and tenderness increase or remain persistent, it is probable that caseation of the inflammatory material has taken place and resolution is past hope. Under such conditions it is well to wait no longer, but to cut down upon the diseased bone and freely gouge or scrape it away till sound bone is reached. After free application of iodoform, the wound should be closed completely, or, if there is a doubt about the asepticity of the wound or the complete removal of the disease, it is better to drain it. In any case, whether acute or chronic, if there is any evidence of softening and breaking down of the tuberculous material, it should be at once and completely cleared away. Operation too early is better than too late.

It is very seldom necessary to amputate a limb in a child, and the question can hardly ever arise in a case of tuberculous disease of bone unless a joint is involved. In certain cases of extensive tuberculous osteomyelitis of the femur spreading downward after excision of the hip or upward from the knee, or in case the disease, beginning in the epiphyseal line, has spread to and destroyed a neighboring joint, it may be necessary in a feeble child to amputate, but, as a rule, conservatism should be pushed to the utmost and every means adopted before resorting to the removal of the limb, and in all cases the tendency of tuberculosis to exhaust itself and die out after a time should be borne in mind. Most surgeons can recall cases where limbs condemned to amputation have been preserved, whether wisely or not is another matter, and have, though usually crippled, become sound by the spontaneous dying out of the tuberculous process.

In any case of tuberculosis of bone it is unlikely that one operation will be sufficient to eradicate the disease. After removal of all that is obviously unsound, the wound may be packed with iodoform or iodoform gauze and allowed to granulate. Healing at first will probably be rapid, but in two or three weeks' time it is likely that progress will cease and the wound will take on an indolent aspect, with a sinus discharging seropurulent fluid, or it will be covered with prominent, coarse, feeble granulations. In such case stimulants and astringents of various kinds may be tried; not the least valuable of these is powdered nitrate of lead. Sublimated sulphur, recommended by Mr. Lane, is too uncertain and sometimes too violent in its action, but may be tried with caution in cases where a superficial cavity exists and the disease is intractable. As soon as it is clear that no progress is being made, it is better, before the mischief has time to spread and make headway, again to repeat the scraping out and applications, and again and again to attack the tubercle till finally the wound heals. We have met with success in these cases even where the result of treatment for months has been most unpromising. After a time, sometimes without any obvious reason, the extension of the process has ceased and the wound has rapidly closed and remained sound. Of the many applications employed in these cases besides scraping, the actual cautery, chloride of zinc, iodoform in powder or emulsion, nitrate of silver, nitrate of lead, powdered sulphur, and caustic potash may be mentioned, but many others have been recommended. The essence of the matter is perseverance. General hygiene and sea-air are among the most important matters.

Though caries is the type of tuberculous bone-affection in tuberculosis, it is not uncommon to meet with necrosis. The existence of a well-defined sequestrum is a satisfactory feature of the case, for it implies a power of forming a line of demarcation separating the healthy from the diseased tissue, and the infiltrating form of tuberculosis is less likely to be present in a case where necrosis exists than in one where the bone is merely eroded and carious. While in acute non-tubercular bone disease the rule should be to remove too little rather than too much, the converse is the case in tuberculosis; the operation should go beyond the obviously diseased area and well into sound tissue where this is possible.

In some cases, where many foci of disease exist or the mischief is very extensive and the child's health much broken down, unfitting him alike to bear an operation or to repair an extensive lesion, it is well to refrain from any operation and to send the patient away to the sea-side, to try what improved conditions will do in the way of enabling him to throw off, or at least localize, the disease; but, as a general rule, it is better to remove the diseased tissue first and send him away to become convalescent afterwards.

Where, as in the case of tuberculosis of the middle ear involving the temporal bone, or in perforating caries of the vault of the skull, or where, again, in disease of a rib or of the sternum, there is danger of extension to neighboring vital organs, special care will, of course, be necessary. It is all the more needful under such circumstances to arrest the morbid process, but caution is required to avoid doing harm by the operation itself.

Mention must here be made of those unfortunate cases in which, after an operation upon a tuberculous focus, an outbreak of acute general tuberculosis occurs, perhaps most often affecting the brain as a tubercular meningitis. There can be little doubt that some, at least, of these cases are due to direct embolic tubercular infection from the operation-wound. The opened blood-vessels or lymphatics take up tuberculous matter left in the wound, and so infection results. These misfortunes are an argument, not against operation, but against imperfect operation,—*i.e.*, they point to the necessity of the complete removal of all tubercular matter, so that the greatest pains should be taken thoroughly to clear out all the disease and to use such applications as are likely to destroy any infective particles that may not have been mechanically removed.

Where it is reasonably certain that all tuberculous tissue has been taken away not only from the bone, but, by excision of the walls of all sinuses and of the margins of openings in the skin, from the soft parts also, it is a good plan to close the wound with sutures without drainage. No better safeguard against recurrence of the tuberculous process in a wound can be provided than healing by primary union. It is true that many cases in which the superficial tissues heal by primary union show again evidence of tubercle left behind, but this is less likely to occur than in those where feebly vitalized granulation tissue exists, as in the track of a drainage-tube, and a free excision of the tissues with closure of the wound again, and, if necessary, yet again, is the best method, where it is practicable, of arresting the progress of the disease.

Sufficient indication has been given as to the line of treatment of tuberculous bone disease in different regions, but a word may be said as to the management of tuberculous fingers and toes and of epiphysitis threatening invasion of a joint. As to "tuberculous dactylitis," a fair number of cases, if taken early, will get well without deformity. If, however, the tuberculous deposit breaks down or involves the epiphyseal line, arrest of growth and permanent distortion are likely to result. Total resection of a phalanx in an early stage is not to be recommended, while later, amputation of a finger gives speedier results and not a more unsightly hand than one with a short, distorted digit. We prefer, as soon as caseation occurs, to open up and clean out the diseased material, leaving the shell of new living bone without a formal resection, and wait for amputation or total resection till this plan has failed.

Tuberculous disease of the cancellous tissue of an epiphysis may, and often does, spread to the adjacent joint, and the possibility of this should lead to careful attempts—repeated, if necessary, again and again—to eradicate the disease before the joint is reached. The limb should be kept still upon a splint and no trouble spared to avoid what must always be a most serious aggravation of the mischief. Where disease has spread from the os calcis or metatarsus to the adjacent joints and extensive tarsal disease is set up, amputation should on no account be practised, but the disease freely exposed by an ample incision, and all tuberculous matter, with, if necessary, the whole tarsus, removed. A thoroughly useful foot may still be retained if nothing of the tarsal bones but part of the os calcis remains. It is only after failure of "total tarsectomy" that amputation should be performed.

LEONTIASIS OSSEA.

The disease to which this name was given by Virchow is a diffuse hypertrophy of the bones of the skull. It affects perhaps most often the upper jaw, and is apparent in the earlier stages as an enlargement of the nasal processes, giving an appearance somewhat like that of a mandril ape. The other cranial and facial bones may become involved.

Leontiasis usually begins in childhood, and is supposed to have some relation to rickets. The bony overgrowth is slow, and, besides the terrible deformity, the pressure upon nerve-trunks may produce pain and even blindness. Though a rare disease, we have seen two or three cases. In one instance pain and nasal discharge, without great bony deformity, led to the exploration of the maxillary antra, both of which were opaque to transillumination. Both antra were found obliterated, and the bone was apparently quite solid. In this patient the skull bones have enlarged and the head has assumed the appearance of rickets; the mental powers are failing, probably from intra-cranial pressure, and there is much headache. It seems probable that death will result from brain-pressure, though there is only slight external deformity at present.

No treatment, except in some instances removal of prominent bony masses, is known to be of any avail.

The disease is rarely recognized in childhood, from the exceedingly slow rate of progress made by the overgrowth.

LATERAL AND FUNCTIONAL CURVATURES OF THE SPINE.

By JAMES K. YOUNG, M.D., AND JOSEPH M. SPELLISSY, M.D.

Definition.—While rotation of the bodies of the vertebræ on the vertical axis usually accompanies lateral curvature, it is not essential and need not form part of the definition.

A large number of synonyms are used to express the deformity; among these may be mentioned *Scoliosis;* French, *Scoliose; Déviation latérale de la Taille;* German, *Seitliche Rückgratsverkrümmung oder-Verbiegung; Bogenförmige Deformität der Wirbelsäule; Seitliche Verbiegung.*

History.—Since the original article was written by Professor Gibney (vol. iii. p. 1001), the distinction between lateral and functional curvature of the spine has been more insisted upon by systematic writers and orthopædic specialists. They prefer to divide lateral curvature into functional and rotary. The past eight years have been marked by increased interest and many decided advances in this important department. Attention has been directed to the fact that the modern orthopædist has received too much material upon the subject of scoliosis at second-hand without sufficient research. The clinical observations of the earlier writers, as Copeland (1815), Sheldrake (1816), Bamfield (1824), Shaw (1827), Coulson (1839), Rogers-Harrison (1842), Tamplin (1846), and Lonsdale (1857), were very thorough, and their careful study by the modern orthopædist has enabled him to avoid repetition. The abandonment of tenotomy by Jules Guérin, the prince of tenotomists, and his contemporary, Louis H. Sayre, together with the increased importance given to exercise in the treatment of these affections, has led to the almost entire rejection of apparatus except in cases of advanced rotary deformity.

RELATIVE FREQUENCY.

The frequency of lateral curvature is greater than would be imagined by the casual observer, as the clothing can be arranged in a manner to hide the deformities of a lesser degree. Without doubt it is the most frequent of all orthopædic diseases, but the estimate of Werner that Prussia has six thousand five hundred people with this affection has little weight. According to Drachman, there were examined in the schools of Denmark twenty-eight thousand one hundred and twenty-five children, and three hundred and sixty-eight were found to be suffering from this deformity. Taken, however, in connection with eight thousand cases of deformity examined by Schilling, Berend and Langaard, and Fischer, of whom two thousand five hundred and fifty-three suffered from scoliosis, we have sufficient to indicate the frequency of this affection. In surgical practice the relative frequency of scoliosis is shown by the comparison of cases treated at the orthopædic dispensary of the hospital of the University of Pennsylvania.[1]

The relative frequency among boys and girls—a larger percentage of the latter, about four or five girls to one boy—possibly may be more apparent by greater attention being given to the development of the forms of the latter and consulting the surgeon earlier upon the recognition of deformity. The relative proportion is found by consulting the following table:[2]

	NUMBER EXAMINED.	BOYS.	GIRLS.
Kölliker	721	144	577
Roth	200	17	183
Wildberger	120	19	101
Lonsdale	170	21	149
Ketch	229	40	189
Berend	896	123	773
	2336	364	1972

Among children under five years of age the number of boys is reported to equal or even exceed that of the girls.

The aggravated forms appear to be in excess among males, although there are severe cases with females.

Scoliosis appears more frequently among the more enlightened, but, as has been stated by a number of authorities, that this is due to civilization cannot be readily admitted, as several of the causes—congenital, traumatic, and pathological—occur in the savage state as well as in the civilized. However, if varieties only were considered, as habitual, static, and professional, the statement would be correct. The relative frequency at different ages is shown by Ketch.[3] From one to twelve years of age, fifty-two per cent.; from twelve to eighteen, forty-one per cent.; and after the latter age only three and one-half per cent. For this reason Ketch has divided them into three classes:[3]

1. Those in whom the deformity was first observed from the first to the twelfth year, or the age of childhood.

2. Those in whom the deformity was first observed from the twelfth to the eighteenth year, or the age of puberty.

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[1] Young, Orthopædic Surgery, 1894, p. 170.

[2] Medical Age, June 25, 1890, article on Lateral Curvature of the Spine, James K. Young, M.D., p. 269.

[3] Ketch, New York Medical Journal, May 19, 1888, p. 8.

3. Those in whom the deformity was first observed from the eighteenth year and upward, or the age of complete development.

In age not stated he found two and one-half per cent.

Etiology.—The production of scoliosis by faulty habitual positions in standing results in an inequality in the lower extremities which may become permanent if not corrected. The relaxed limb tends to become longer and thicker than the limb upon which the superincumbent weight is borne. In this manner a long limb is produced. The tendency to stand upon the left limb may be responsible for the greater frequency (sixty-six per cent.) of right dorsal scoliosis.

Enormous morbid growths, as encephaloid, enchondroma, or sarcoma,[1] springing from the sides of the pelvis, may by their weight cause scoliosis. Scoliosis may also arise in sacro-iliac disease from the habitual faulty position assumed to relieve suffering.[2]

Pathology and Pathological Anatomy.—The first pathological change is probably in the intercartilaginous disks. In advanced scoliosis the changes include alterations in bone, ligament, intercartilaginous and muscular structures, in the order of importance. From the difficulty of obtaining specimens of the early stage the lesions of this period are but poorly understood. In the original article a full description and illustration are given of a specimen of advanced lateral curvature. The accompanying cuts from Hoffa will help to demonstrate more minutely the changes that are seen in wedge-shaped deformities of the individual vertebræ. These illustrations show the body to be atrophied on the concave side (Fig. 1), and in some cases ossified to the adjacent vertebræ by a mild pressure osteitis, resulting in the wedge-shaped body with the base towards the convexity (Fig. 2). Pressure shortens one of the roots of the vertebral arch, imparting an ovoidal form to the canal. The bony fibres of the body occupy an oblique instead of an upright position. The description of a pathological bone specimen might be supplemented by an account of the alteration in the ligamentous, intercartilaginous, and muscular structures. The constant pressure on the intervertebral bodies causes them to become wedge-shaped and to lose their elasticity and ability to reassume a normal position. Ligaments connecting the vertebræ are lengthened on the convexity and shortened on the concavity of the curve. The muscles on the concavity are contracted and atrophied to a less degree by their osseous attachments being brought too close together. On the convexity the muscles are relaxed, stretched, atrophied, and fatty degenerated. While the muscles of the convexity are considered the weaker muscles, in restoring the curve equal attention must be paid to the muscles of the concavity.

Symptomatology.—The symptomatology of this disease can be thoroughly studied only by reference to minutiæ and accurate records of a large number of cases. The taking of these records has been much facilitated by publishing the painstaking methods of several observers. These writers have employed some ingenious mechanical devices which record accurately the *status præsens* of a case, also elaborate, uniform, and tabulated methods of mensuration, and by no means of least importance in the recording of symptoms is the use of the camera and anthropometrical charts. Whatever method be employed, the system, to give reliable and useful data, must be uniform. Photographs should be taken of the front, back, and sides of the body in the faulty and corrected positions; also two lateral views and a rear view of the body in Adams's position. The value of the photographs is improved by photographing through a wire screen after the method of Brackett, of Boston.[1]

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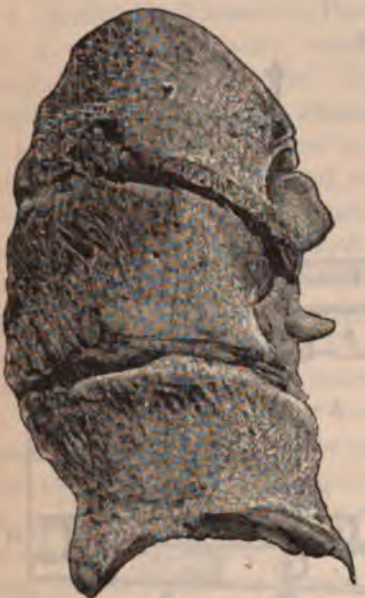
[1] Shattuck, Boston Medical and Surgical Journal, January 10, 1889.

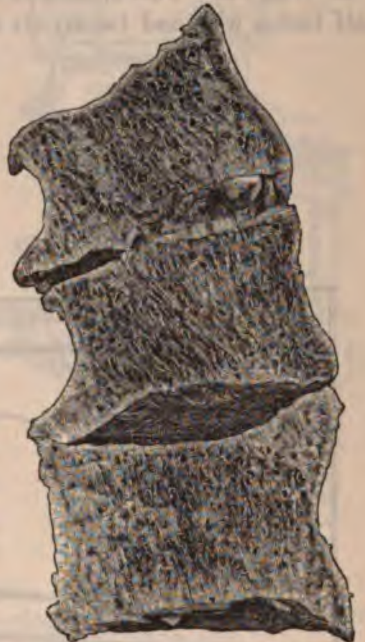
[2] Young, Orthopædic Surgery, 1894, p. 175.

FIG. 1.



Osseous changes in vertebral bodies. (Hoffa.) From Young's Orthopædic Surgery.

FIG. 2



Section of vertebra showing condensation on concave side and rarefaction on convex side. (Hoffa.) From Young's Orthopædic Surgery.

APPARATUS FOR MEASURING SCOLIOSIS.

Out of a large number of apparatus for recording the rotation in scoliosis we mention the following:

1. Hawksley's cyrtometer.
2. Weigel's.
3. Bradford's.
4. Beeley's scoliosometer.

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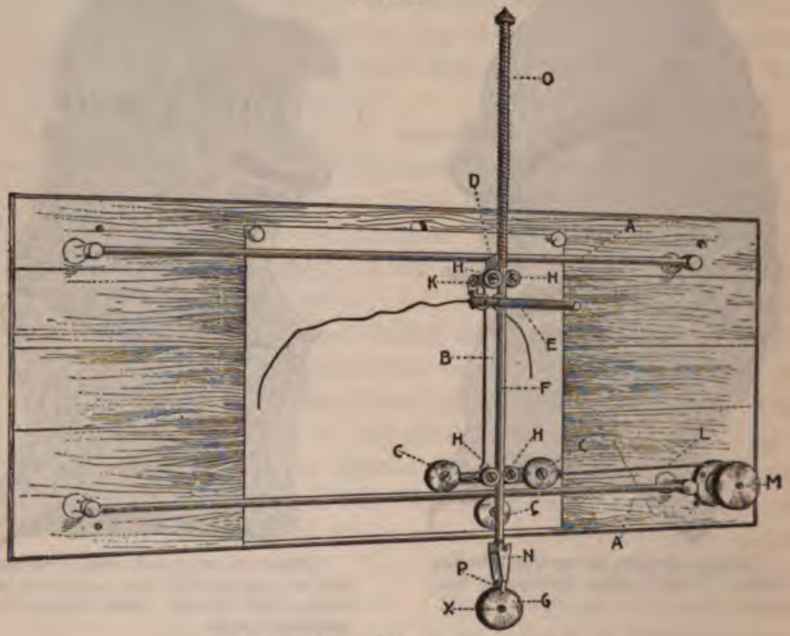
[1] Children's Hospital Reports, 1894.

These are recommended in the reverse of the order in which they are given here.

1. *Hawksley's Cyrtometer.*—This is a narrow band of lead long enough to pass around the body and allow the ends to overlay each other, and of the right thickness to allow it to be readily formed to fit all the curves of the back and sides. This band is marked in a scale of inches. It is the instrument generally used by clinicians.

2. *Weigel's.*[1]—This apparatus is mounted on a thin board ten and one-half inches wide and twenty-six and one-half inches long, and consists of two rods attached to the board within about an inch from the upper and lower edges, and so arranged that they are connected with the board at each end only. Upon these rods a trolley moves horizontally. To the trolley is attached a pencil, which is held against a paper that is fastened to the board back of the rods. At the lower end of the trolley-rod a roller is attached. The pencil is held against the paper by a light spring. The movement of the apparatus across the board is controlled by a helical spring. These springs render the instrument almost automatic in its action, it being necessary only to guide the roller across the back of the patient. The marking pencil, taking the movements of the roller through the trolley, reproduces on the paper the outlines of the surface over which the roller moves.

Fig. 3.



Weigel's apparatus for measuring scoliosis.

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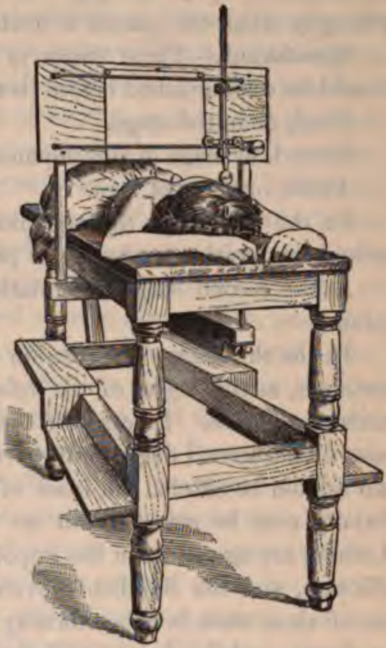
[1] American Orthopædic Association, 1893.

3. *Bradford's.*—This consists of two steel bars arched in the centre and fastened together at one extremity by a pin, having a quadrant at one end and a spirit-level at the other. The patient being prone, the instrument is applied across the deformity from side to side, with the attached end at the convex side. The arm with the spirit-level is raised until the level is attained and the degree of deformity can be read from the quadrant.

4. *Beeley's Scoliosometer.*—This consists of a steel framework containing a series of narrow steel rods. By two plummets and a spirit-level attached to the instrument it may be used to take outlines in a vertical, horizontal, or lateral plane. The rods are pressed into position by a roller and secured by a brake. The outline is impressed upon paper by pins attached to the lower surface of the ends of the rods. For practical purposes this is the best instrument for recording the amount of rotation, because the steel points can be applied more closely to the skeletal outline and the error of the thick muscular pad on the convex be eliminated. An improvement on this would be a circular measuring appliance on the same principle, fashioned after a hatter's measuring machine.

FIG. 4.



Weigel's apparatus in use.

Diagnosis.—A careful diagnosis of this disease in its early stages is most essential. The body should be placed in a strong light, and all its curves and lines carefully examined from various positions, both in front and rear. To facilitate this examination the body of the patient, in the case of a child, should be uncovered as far down as below the trochanters. The rotation of the spinous processes should be carefully noted and marked with the patient in an erect posture and also bending forward in Adams's position or self-suspended from a bar. Any difference in the length of the lower extremities may be ascertained by the use of books or blocks under the feet in connection with a spirit-level. If by this method there is any doubt, or if any difference is detected, the body may be placed upon a suitable couch and the lower extremities measured. Among the many apparatus for measuring and recording the deformity, Young has a list of thirty-three,[1] but an ordinary lead measure, heretofore described, the writers find answers all purposes. In the diagnosis careful attention is called to cases of *lateral bending of the spine* caused by debility. This condition is without rotation, and can generally be remedied by appropriate constitutional treatment. Attention should also be directed to *lateral deviation of the spine* occurring in the early stages of Pott's disease. This is due to unilateral spasm during exacerbations of the disease, and disappears promptly when the patient is treated in a prone position.

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[1] University Medical Magazine, February, 1898.

Treatment.—Three stages or degrees of deformity are recognized and should be distinguished before treatment is instituted:

First, or initial stage.

Second, or stage of development.

Third, or stage of arrest.

In the first stage the rotation can be corrected by suspension, by the recumbent position, or by slight pressure of the hands.

In the second stage the rotation cannot be entirely corrected by these means.

In the third stage the rotation cannot be affected in the least by these measures, as the degree of the deformity has a direct bearing on the prognosis of the case. In cases of the first degree a cure may be expected; in cases of the second degree improvement may be promised, but a perfect cure can seldom be effected; in cases of the third degree the progress of the deformity may be arrested, but no improvement in the stature will occur. Authors are agreed upon the importance of exercise in the treatment of this affection, and the Swedish movements are usually preferred; but in the use of these there is no uniformity at the present time. Some writers insist on the general development of the body, first by the use of heavy weights. The objection, however, to the heavy weight system is the strain on the heart and lungs and the short time devoted to the course of exercises, which is not sufficient to permit of the compensatory action of the heart being established.

Others, by employing the eight physiological movements of the spine as a basis, attempt to form a rational system of exercises.[1] These eight primary movements include,—

1. Bending forward.
2. Bending backward.
3. Bending to the right.
4. Bending to the left.
5. Combining these movements in circumduction to the right.
6. Combining these movements in circumduction to the left.
7. Rotation of the spine upon its vertical axis to the right.
8. Rotation of the spine upon its vertical axis to the left.

Our method consists in developing first the relaxed and weaker muscles until the curve is slightly over-corrected, then by general systematic exercises to improve and maintain a perfect physical development. This plan, given in detail, is to find the position that can be the most readily assumed by the patient to overcome the deformity, and then, maintaining this position as nearly as possible, perform all the exercises, constantly endeavoring to bring the body to a correct position in the use of all the movements, thus tending to the developing of the muscles on the concave side of the deformity, also of the weaker muscles on the convex side. These exercises are to be continued until the curvature is over-corrected in a slight degree. A treatment tending to the development of the general physical condition is then commenced and kept up until the deformity is permanently overcome. Primary forms of curvature being exceedingly rare, each case requires particular study, and while sets of exercises are given for individual cases, most of the forms of exercise will be made up of a combination taken from them all. The use of these prescribed exercises must be under the personal supervision of the surgeon in charge or of a trained instructor or masseuse. They require to be performed slowly once a day, and each movement made from five to fifteen times, and, at the least, one hour before or not sooner than two hours after a meal. Massage of the weaker muscles on both sides of the curve should follow the exercises, and the patient must occupy the prone or key-note position for at least half an hour or an hour after. Any necessary medical treatment should be prescribed and any menstrual irregularities corrected. The exercises are to be continued daily for from four to six months, omitting Sundays and the menstrual periods, and to be given by the surgeon in charge, three or four at a time, until the whole number has been learned. They should last from twenty minutes to half an hour, and are not to be continued to the point of fatigue. During the exercises the breathing should be regular, and one or two full breaths taken at the end of each exercise. The most important element in all these exercises is time. From four to six months will be necessary to cure mild cases, and two or three courses of the same period will be required for medium cases. The exercises are usually under the direction of a masseur or a masseuse, the surgeon supervising the treatment from week to week. More importance is attached to the direction than to the personal conducting of the treatment.

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[1] Rugh, Philadelphia Polyclinic, December 5, 1896.

This is the European method of treatment. Where the hours of school interfere with the treatment, in order to derive the fullest benefit it will be necessary to omit the school altogether or limit the attendance as much as possible.

A consideration of the special treatment of primal right dorsal scoliosis will best illustrate this method. In right dorsal scoliosis the right shoulder is elevated with the scapula projecting backward; the left shoulder is depressed, the scapula flattened, and the spinous processes of the dorsal vertebræ rotated to the right, the weaker muscles being considered on the concave side. The pelvis is inclined to the left and downward. The right leg is elongated and the spinous processes of the lumbar vertebræ are rotated to the left, the contracted muscles being, as before stated, on the right, or on the side of the concavity. The weaker muscles are first subjected to the treatment by putting the skeleton in such positions as to render the muscular attachments more normal, and consequently strengthened and contracted. Further, the longer or right leg is shortened by special exercises, which contract the muscles about the hip-joint. The fact that this long limb can be shortened is conclusively established by the number of successfully treated patients, as in the case of a college student who was injured and where elongation had occurred from relaxation following the injury. Should the inequality of the lower extremities result from other causes, as flat-foot, asymmetry, or fracture, these must be corrected. The foregoing remarks particularly apply to elongation occurring in atonic cases.

That elongation and rotary lateral curvature may be caused by incorrect postures continued during a long period is shown in the case of an artist's professional model, who in two years has developed a left dorsal scoliosis from standing resting the weight on the right leg.

The special exercises in right dorsal scoliosis[1] are as follows:

1. Best standing position.
2. Key-note position.
3. Key-note, forward bending.
4. Key-note, sideways bending, right.
5. Key-note, forward sideways, turning right.
6. Key-note, heels rising.
7. Key-note, heels rising, knees bending.
8. Key-note, fall out, right limb forward.
9. Hanging (left hand higher), right limb rising, with resistance.
10. Hanging, knees abducting, with resistance.
11. Hanging, knees adducting, with resistance.
12. Right hip flexing.
13. Right hip extending.
14. Right hip rotating.
15. Prone key-note, backward sideways, turning right.
16. Salute, right arm, from five to ten times, arms extended.
17. Left shoulder elevated, right scapula depressed.

Exercises for the right lumbar secondary curve which usually appears in these cases are also included. The exercises for treating left dorsal scoliosis are the reverse of those given.

For lumbar scoliosis, right or left, the treatment includes the exercises given in the foregoing for the development of the lower extremities, adding exercises for the special treatment of the lumbar muscles. After the removal of the cause of primary cervical curves, special exercises with resistance are employed to develop the muscles of both the concave and the convex side of the curve; when associated with right dorsal scoliosis they are directed to the right side of the neck, the head being carried to the left.

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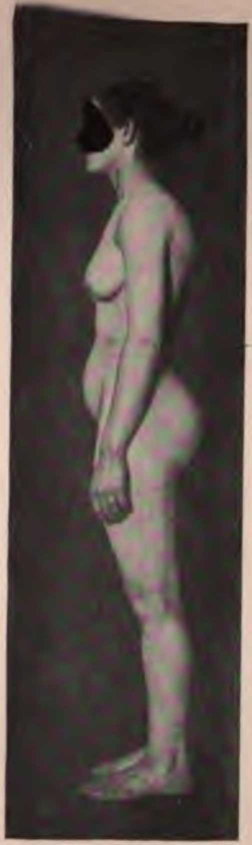
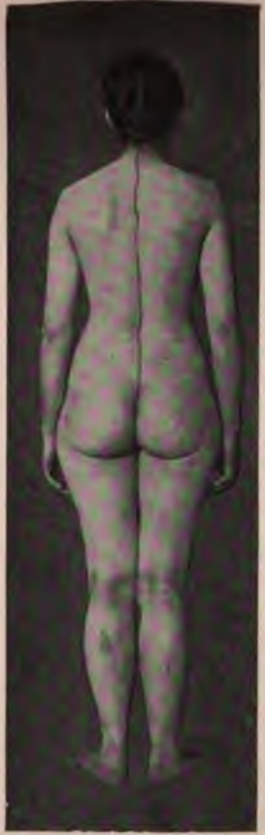
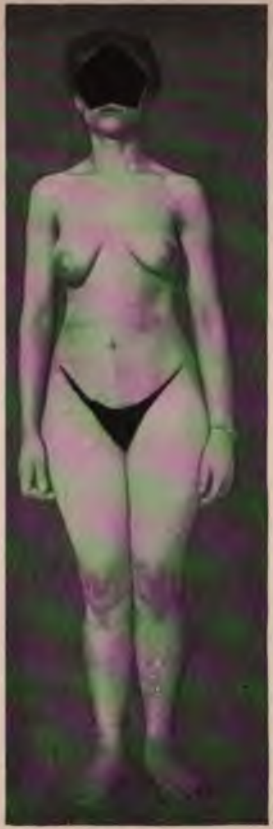
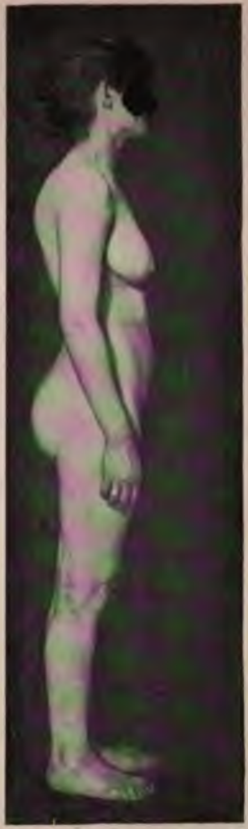
[1] *Vide* International Medical Clinics, vol. ii., Eighth Series, 1898.


Faulty standing position. Side view.


Faulty standing position. Rear view.


Faulty standing position. Front view.


Faulty standing position. Side view.

After the over-correction of the curves by the employment of method, general exercises are used, with or without apparatus, for the s metrical development of the body; these are taken with both arms tended, with both arms elevated, and with both arms in the neck-rest po tion. Then general muscular development is maintained by eight especi exercises originated by Drs. Keating and Young eight years ago, and pub lished at that time.[1] They are as follows:

1. Neck-rest, heels rising.
2. Alternate trunk twisting.
3. Stretch, standing, sideways bending.
4. Stretch, standing, falling out.
5. Half-stretch, standing, falling-out position.
6. Stretch, turn, falling out.
7. Falling out forward.
8. Stretch, standing, heels rising and bending.

No mention has yet been made of the different apparatus for correcting and maintaining the correction of this deformity. Among these apparatus the best results have been obtained from the Shaffer brace modified by Young.[2] The design of this apparatus is to suggest constantly to the patient the necessity of maintaining a correct position rather than to compel it by direct pressure, and also to support the body slightly between the times of exercise and reclining.

Mechanical Correction.—In the medium degree of this affection mechanical means are needed as supplemental to the treatment by exercises, and of these, means for forcibly correcting rotation are very valuable. The same principles are in them all, and Beeley, Loring, Redard, and Hoffa all show to what degree force can be used to advantage without resultant injury. Two kinds of apparatus are in use:

1. Those placing the body in the best posture of correction.
2. Those bringing to bear direct pressure of correction.

Of the first, that of Beeley is the best. This is a square framework in which the patient is placed, and weights and straps used to assist in the correction. The apparatus of Schede, of Hamburg, modified by Bradford and Bracket and by Weigel, represents the second form, and can be used for this purpose.

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[1] Vol. iv. of this Cyclopædia.

[2] Young, Orthopædic Surgery, 1894, p. 191.

SYNOVITIS.

BY H. M. SHERMAN, M.D.

It is necessary here only to recall the fact that synovial membranes have no true epithelial lining,[1] and that where there seems to be a definite layer of cells on the surface, it is due to a simple aggregation and stratification of the ordinary connective tissue cells of the part. A synovial membrane, therefore, is not to be thought of as a modified serous membrane, but is to be classed as a simple connective tissue structure, not differing anatomically or physiologically from an adventitious bursa.

Synovia is a viscid, slightly yellowish fluid, alkaline in reaction, and contains fat molecules, amœboid corpuscles, and surface cells from the membrane. In solution is a mucin-like nucleo-albumin, but no true mucin.[2] In pathologic synovia has been found a mucin-like body which is not a nucleo-albumin, and which has been named synovin.[3]

Inflammation of the synovial membrane does not differ in character from inflammation of other connective tissue structures, and it may result in the production only of new connective tissue cells, or of serum, or fibrin, or pus. Clinically, we encounter in children chiefly the forms producing serum or pus, sero-synovitis, or suppurative synovitis (pus-joint), and the synovitis due to tuberculosis.

Sero-synovitis, acute simple synovitis, occurs as the result of a trauma, such as a contusion or a sprain, or of over-use which amounts to a trauma, or of exposure to cold. It may follow any of these causes in rather a short time, reaching its maximal intensity in from twenty-four to thirty-six hours. At this stage the membrane would be congested and swollen, but the chief pathologic feature is the great increase in the amount of the synovia. This increased synovia is apt to be thinner and more watery than normal, and, as stated above, it has accidental ingredients. It is probable that with the increase of serum a production of a certain amount of fibrin occurs, for flakes of this are always found floating in the fluid. In the severer types the number of surface cells cast off may be so great as to give the fluid a milky or creamy appearance, but this is not to be confounded with pus in an infected joint. After a certain time there is a subsidence of the inflammation, and by retrogressive steps the joint returns to its normal condition. The whole process has probably been reparative in its action from the moment of the full effect of the injury. There is usually comparatively little pain in connection with this condition, but there is discomfort which finds expression, and there is disability, use of the limb being guarded or avoided. Inspection shows a diffuse swelling of the joint, limited in its extent by the limits of the synovial sac, and usually obscuring the ordinary bony outlines of the part. Palpation finds the swollen joint warmer, and at the same time easily discovers fluctuation of the contents of the sac. In cases of ordinary intensity, when the intra-articular pressure of the effusion is not excessive, the position of the limb is not necessarily rigid, but in the severer types the limb is permitted to move very little from the position in which the joint-cavity can accommodate the most fluid, and in these cases there are more pain and local temperature, and there may be a general fever.

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[1] Quain's Anatomy, tenth edition.

[2] Hammarsten.

[3] Salkowski.

Of all the joints the knee is most often affected, and here the fact of a given swelling being due to fluid within the synovial sac can almost invariably be demonstrated by the phenomenon of the floating patella, and the appreciation of fluctuation between the extreme limits of the synovial sac. Synovitis at the ankle is usually the accompaniment of a sprain, and its signs are merged in those of the greater injury. Synovitis at the hip is apparently rare in children, though Gibney has mentioned cases. The effusion causes fulness both anterior and posterior to the trochanter, and if it is sufficient in amount, fluctuation can be appreciated. In synovitis at the shoulder the effusion makes the shoulder appear deeper antero-posteriorly, and fluctuation can be best felt over the front of the joint. At the elbow the swelling more evidently obscures or changes the contour of the part, espepecially filling out the depression on either side of the triceps tendon, and it is here that fluctuation is to be looked for. The swelling of synovitis at the wrist is outlined by the limits of the synovial sac, extending across the limb in distinction from the swelling of a teno-synovitis.

The diagnosis is primarily simple, and is made on the general and local symptoms and on the evident relation between the trauma and the condition as cause and effect. Doubt enters only in those cases where the trauma was slight and the resulting inflammation not particularly acute, and in such cases the possibility of the trauma being the occasion of the development of a tuberculous synovitis, and not the cause of an acute simple synovitis, must be entertained. The treatment is the classical one of immobility for inflamed joints, supplemented by such accessories as special indications require. Nothing secures immobility so perfectly, no matter what joint may be affected, as a properly applied and well-fitting plaster of Paris splint, and the effects on the limb of both heat and cold may be obtained through the splint so long as no wet applications are used. To apply heat, flat bags of sand, heated in an oven, are packed about the splint over the joint and changed for fresh ones as soon as they become cool, and to apply cold, bags of ice are used in the same way.

The question of hastening the often rather slow process of absorption of the effusion has of late more especially engaged attention. The operation of aspiration may be done, or the fluid may be expressed by a snugly applied rubber bandage through a small trocar thrust into the joint-cavity between the layers of the bandage. After either of these, firm compression of the joint must be made or the effusion will be repeated. Massage, by stimulating the local circulation in the lymphatics, is also useful, but it must be so given that it is not in itself a trauma, and it is quite inapplicable during the time of acute symptoms. For some time afterwards the joint must be considered as especially vulnerable, and therefore entitled to particular care.

Synovitis with the production of pus can occur only as the result of infection with pyogens, ordinarily the staphylococci and streptococci. These usually gain admission to the joint by a direct penetrating wound, but the admission may be indirect, or the synovitis may be a secondary lesion, as when an abscess or abscesses exist in other parts of the body, or when the joint-inflammation is sequent to an acute infectious disease. Pathologically the synovial sac then becomes the sac of an abscess. The contents are synovial fluid and pus. The synovial membrane is congested and has a velvety or granular surface, or the membrane may be pale instead of reddened, and covered with a smooth, fibrinous exudate.[1]

The intensity of the symptoms, local and general, in comparison with those seen with simple sero-synovitis, will lead to the diagnosis of a pus-joint. If there has been an accidental wound opening into the joint as the cause of the synovitis, or if the synovitis comes during the course of or during convalescence from an acute contagious disease, more especially scarlatina, the presence of pus in the joint must always be thought of as a possibility. The diagnosis can always be confirmed or disproved by an exploratory aspiration, securing material for bacterioscopic examination.

The fact of the presence of pus in a synovial sac having been established, the condition demands the same prompt treatment as any other acute abscess. Free incision, thorough washing out, and afterwards efficient drainage are imperative needs. In the ordinary healing of an acute abscess the obliteration of the abscess-cavity is a matter-of-course result, and the same is true in the great majority of cases of pus-joints. In such cases the joint affected should be laid open by the ordinary resection incisions, so that every part of its cavity can be reached, and after its contents have been evacuated it should be thoroughly packed with gauze and the whole treatment such as is given an open wound,[2] especial care being taken that the limb is in proper position when ankylosis takes place. If the case is recent and it seems possible that ankylosis can be avoided, the plan followed at the Johns Hopkins Hospital may be tried. As applied to the knee, the joint is opened by two long incisions, one on either side of the patella, and the cavity thoroughly irrigated with several gallons of hot sublimate solution, 1 to 1000. No drainage is arranged in the dressing, but the openings are not sutured, being allowed to close by granulation. Through them subsequent irrigations may be made during the process of healing. Passive motion carefully done may prevent ankylosis.[1]

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[1] Finny, Johns Hopkins Hospital Bulletins, Nos. 54 and 55.

[2] Mays, Annals of Surgery, vol. xxi. p. 87, and Gerster, ibid., vol. xxii. p. 503 *et seq.*

GONORRHŒAL SYNOVITIS.

Synovitis due to infection by the gonococcus is not, as was once held, unknown in infants and children. It may occur in conjunction with a gonorrhœal ophthalmia in the new-born,[2] or later, at any time during the course of a gonorrhœal vulvo-vaginitis. It has not yet been reported as occurring during the course of a gonorrhœa in a small boy. The joints most frequently affected are, in the order of preference, the knee, the wrist, the tarsal joints, the smaller articulations of the hand and foot, and the hip. Neighboring synovial sheaths may be affected simultaneously, or the teno-synovitis may be the principal lesion. When the lesion is monarticular, the knee is the joint affected. The inflamed joints are painful, swollen, hot, and the skin is shiny and red. The appearance is very much that of a suppurative synovitis. The presence of effusion is, in all superficial joints, usually demonstrable. During the invasion there are often fever, anorexia, and prostration, or vague and severe pains. These symptoms after a little subside and the local inflammation appears. The synovitis may last from five to fifty days, but the usual duration is fifteen. Complete recovery from the synovitis is the rule, the joint not being left ankylosed, as in the adult; on the other hand, death may occur in an infected new-born child. The diagnosis should be suggested by observing in a case of apparently ordinary rheumatism an ophthalmia or a vulvo-vaginitis, and the bacterioscopic examination of the discharge from these would determine their character.

In the case of a severe monarticular synovitis at the knee where a pus-joint was feared, the exploratory aspiration of the joint and the finding of the gonococcus would establish the comparatively benign character of the inflammation. Free incision is not indicated in a gonorrhœal joint unless there are, after one aspiration, reproduction and persistence of the effusion. In such a case incision, as in the case of a pus-joint, should be practised. When this is not needed, local treatment should be immobilization and heat or cold, dry or moist.

Internally, the usual antirheumatic, salicylic acid in some of its forms, is advantageous, and the original infection, ophthalmic or vulvo-vaginal, demands attention. During convalescence, massage and douches will hasten the re-establishment of full function. For a sequent muscular atrophy faradism has been advised.[3]

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[1] Finny, loc. cit.

[2] Tyrrell, Medical News, 1896, vol. lxviii. p. 271.

[3] Martan, Journ. de Clin. et Thér. Infantile, September 3, 1896; quoted in Journ. des Praticiens, November 7, 1896.

SYPHILITIC SYNOVITIS.

Synovitis due to syphilis may now be quite definitely recognized. It may be the only manifestation of the disease, in which case the etiology would be very difficult to discover, or it may be one of a group of symptoms, when the diagnosis should be easier. The condition may be met with at any time in the course of the syphilis, whether that be hereditary or acquired, but, uncomplicated, it is more frequently seen early; the synovitis that occurs later is apt to be only the accompaniment of periostitis and gummata close to the joint.

The lesion itself is never an acute one, rarely attaining even subacute intensity, and there is not usually much pain or deformity or disturbance of function; but there is effusion which has come on gradually and without much notice of its presence apart from the swelling. Moreover, the condition affects more than one joint ordinarily, so that there may be symmetrical lesions, or various joints may be affected simultaneously or consecutively. This multiplicity of joints attacked is a point of diagnostic value,[1] but there can usually be discovered some other stigmata of the disease, such as interstitial keratitis,[2] or, more likely, a periostitis with the deposit of new bone,[3] or enlarged glands, though the enlargement of lymphatics is so common in other conditions that its import here is lessened.

The affection occurring in this way yields, though sometimes slowly, to mercury and iodide of potassium, and may, even in cases of hereditary disease, subside spontaneously.[4] It is right to recognize the joints as inflamed and to give them the proper mechanical support and rest from function.

In cases of acquired syphilis in older children synovitis may also occur at any stage. Shortly before the skin lesions there may be an attack like a polyarticular rheumatism, the joints being hot and red and swollen, with limited motion and painful points, and these symptoms may be quite severe. Later in the syphilis the symptoms are more likely to be severe and are chronic. There are thickening of the capsule and crepitation, both due to villous hyperplasia, and there may be erosions of the cartilage. If in these cases the thickening of the capsule is localized, it is probably due to a gumma.

Here, too, the diagnosis, in the absence of other marks of syphilis, depends in great measure on the affection of many joints and the absence of a heart lesion, and it is to be noted also that the joints are the seat of comparatively little pain. The prognosis is good in both groups of cases, but it is better in the earlier and milder lesions. The later lesions may leave impaired or suspended joint-function.[5]

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[1] Bowlby, Lancet, 1893, vol. ii. p. 1062.

[2] Hutchinson, Jr., British Medical Journal, 1892, vol. i. p. 797.

[3] Trinkler, Berliner klinische Wochenschrift, 1890, vol. xxvii. p. 590.

[4] Hutchinson, Jr., British Medical Journal, 1892, vol. i. p. 797.

[5] Lang, Vorlesungen über Path. und Therap. der Syph., 1895.

HÆMARTHROSIS.

The effusion of blood into a joint is the result of a trauma which has lacerated a blood-vessel of size, the blood escaping into the joint-cavity. The distention of the capsule is rapid and may be very great, the tension in some cases preventing the pressing of the floating patella back to its place on the femur and making a click. Rarely there may be felt a crepitation in the joint. The condition is generally painful, there is tenderness over the entire region, and there may be an irregular fever, but the chief symptoms are the local ones.[1]

The diagnosis is from synovitis, and must rest on the rapidity and extent of the effusion. In known cases of hæmophilia hæmarthrosis was recognized long ago,[2] and attention has been called to the dangers of operative interference. In hæmarthrosis in children not the subjects of hæmophilia the operation of aspiration is rendered difficult because of the clotting of the blood in the needle.

In each case the preferable treatment is by rest and even compression at first, and later by careful and gentle massage and passive motion to prevent the adhesion of a clot to any one part of the capsule of the joint, where it may become organized and cause localized thickening and loss of elasticity.

ACUTE EPIPHYSITIS.

This condition merits consideration here because of the relation the epiphysis has to the articular cavity. The different parts of the joint have all been formed by tissue differentiation from the same original material, and looked at from this stand-point the capsule of the joint is seen to be a continuation of the periosteum from bone to bone, modified, however, in transit.[3] During development it is most intimately attached to the circumferential margins of the epiphyseal cartilage, and consequently the epiphysis, both the cartilaginous and osseous portions, is practically within the joint, and the passage of substances around the margins of the articular cartilages from the epiphysis to the articulation, or *vice versa*, is not difficult.

The lesion itself is a staphylococcus osteomyelitis, not differing pathogenically or pathologically from other forms of staphylococcus infection, and varying from them only in the tissue affected and its anatomical environment. The condition almost always occurs in children under one year old, and this fact has led to its being classified apart from other forms of osteomyelitis; but it is a possible occurrence during childhood and adolescence,—in fact, until the bone has become an adult, and the epiphysis and diaphysis, having united, have lost their separate identities.

Occurring in the new-born, the infecting pathogen may have entered through a suppurating umbilicus[1] or by the ordinary modes of ingress, as in older individuals.

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[1] Broca, Presse Médicale, Paris, 1894, 397.

[2] Steele, Cyclopædia of the Diseases of Children, vol. iii. p. 1138.

[3] MacNamara, Diseases of Bones and Joints, third edition.

The epiphyseal lesion may be single, or quite frequently two or many epiphyses may be affected, or the bone lesions may be but local symptoms of a general pyæmic infection. It affects by preference the hip, about half of the cases; the knee, about one-third of the cases; and the shoulder, about one-sixth of the cases. But it may occur in any epiphysis, and in somewhat more than one-third of the cases a plural number of joints is concerned.[2]

The pathology is hardly unique. It is a classical staphylococcus osteomyelitis which runs a rapid course. Transitional forms of tissue during constructive processes have notoriously feeble powers of resistance to interference, and in this instance there are rapid necrosis of bone and cartilage and separation of the epiphysis from the diaphysis. The infection invades the synovial sac, and the whole joint is an abscess with the epiphyseal sequestrum loose in it, or the epiphysis may disintegrate entirely and form part of the fluid pus.[3] This total destruction of the epiphysis and joint may take place in thirty-six hours,[4] but this is usually in the youngest patients or in an overwhelming infection, the process being slower in older children or in those in whom infection is not so virulent. The local signs are those commonly seen with a pyogenic process. The limb becomes swollen and hot, and may be red and œdematous or brown and glazed, with the superficial veins marked under the skin. Fluctuation in the joint can be felt, and the joint itself is habitually flexed;[5] any effort at extension of it causes pain and is resisted. As disorganization progresses the bones may be moved on each other with crepitus of bare bony surfaces, and finally a pathologic dislocation may result. The general symptoms begin with a chill, but in infants and younger children this may be absent or replaced by an attack of vomiting. The temperature rises rapidly and high, and the individual is very quickly and plainly seriously ill. The initial chill may be followed by others with temperature exacerbations, and there may be delirium, convulsions, and coma. The mortality is very high: of Townsend's collected cases forty-five per cent. had died. On the other hand, if the lesion is not an expression of pyæmia, and if it is recognized early enough, it yields surprisingly to treatment. Townsend makes the diagnosis from rheumatism, sprains, periarticular abscess, malignant growths, and syphilis. The condition might also be mistaken for a suppurative synovitis. Of all these it is most likely to be confounded with a syphilitic epiphysitis, more especially if the acute epiphysitis affects more than one epiphysis or joint, and the differentiation must be made by finding other specific stigmata. Rheumatism may simulate it, but in this the joint-effusion is not pus and the general symptoms are lighter. The ultra-careful distinction between a periarticular abscess, a suppurative synovitis, and an acute epiphysitis is not a clinical necessity, as the treatment in all these conditions is the same. Still, the diagnosis can usually be made by careful attention to all the details of the development of the lesion.

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[1] Townsend, Acute Arthritis in Infants, Transactions of the American Orthopædic Association, 1889.

[2] Ibid. [3] Ibid. [4] Ibid. [5] Townsend, loc cit.

The treatment can be on but one line. The epiphysis or joint must be freely cut into, pus and necrotic tissue removed, proper drainage established, and the complete technique of an antiseptic dressing practised. If this is done before there has been much destruction of tissue the result may be a saving of the limb, and the articulation may be left with apparently perfect function; but if the epiphysis has necrosed and separated, the ultimate result can only be an incompetent joint and limb, for ankylosis is not the rule, and instead we have "flail joints," "dangle limbs," and pathologic dislocations.

When the hip-joint has been destroyed, it may in later years, after muscular and ligamentous attachments have stretched under undue use, resemble in appearance and disability a congenital dislocation.

TUBERCULOSIS OF JOINTS.

In what is to be written on this subject the stand will be taken, in accordance with the universally accepted teachings, that tuberculosis is due to the bacillus of tuberculosis, and that without this infective agent the disease does not occur. If there is in the history of the development of any particular case an injury, a wrench, sprain, contusion, or wound, the general organism immediately and as a natural matter of course institutes reparative processes, which are carried on to completion or to the point where the infecting pathogen interferes and, by adding to the remaining damage of the first injury the poisonous effects of its products, inhibits further recovery. From this time, no matter whether the incidental trauma is entirely recovered from or not, the pathologic and clinical conditions are the effects, directly or indirectly, of bacterial life, and the injury itself, as a still potent causative power, is to be entirely disregarded. No blow or other injury can by unhappy fatality be so cunningly delivered or so unfortunately received as to set in action a lasting and increasing train of evil effects. The chronic, continuous, and usually increasing effect can be due only to a continuously acting and increasing cause. At any period of the case, if this cause can be eliminated, pathologic processes will cease and recovery ensue, its completeness limited only by the amount of destruction already accomplished. The selection of any particular site of infection is controlled in the first place by feeble powers of resistance, innate or accidental, on the part of the tissues to any injury, mechanic, thermic, or bacterio-chemic; and in the second place by the coincident presence of the bacillus.

Tuberculosis as it is seen affecting the bones and soft tissues which constitute a joint differs in no particular, pathogenically or pathologically, from the same disease affecting other organs. For the review of the general subject the reader is referred to the chapter on "Tuberculosis," by George Blumer, M.D., page 323 of this volume.

The special tissue-changes as they occur in tuberculosis of the synovial membranes will be reviewed, and then the subject of joint tuberculosis from a clinical stand-point, the pathology of bone tuberculosis being considered in the chapter on "Tuberculous Diseases of Bone," by G. A. Wright, page 1000 of this volume.

TUBERCULOUS SYNOVITIS.

Tuberculosis of the synovial membranes may occur as a primary local lesion, but it is more likely to be, in accordance with the general rule,[1] a secondary lesion, the primary lesion being a glandular or visceral tuberculosis. The disease may, too, be due to a direct extension of the process from the neighboring epiphyses, but it is then to be considered only as a portion of the more general joint-infection.

Occurring in conjunction with and secondary to a bone lesion, the infection attacks the whole of the synovial surface as the contents of a tuberculous abscess are discharged into the joint. Occurring independently of a bone lesion, the infection may be from the first diffuse, but more commonly it begins at one or more points and from these spreads over the entire membrane.[2] The infection and the earlier tubercles are located just under the surface of the membrane, and the products of their activity may be increased synovial fluid or tuberculous granulation tissue. If the tubercles are comparatively few the membrane may not be at first appreciably thickened, but there is effusion in the joint, and this may resemble the normal secretion or be thinner and contain shreds of lymph, the condition being termed tuberculous hydrops.[3] The amount of this effusion may vary from a little to a great deal, and if it is removed the normal contours and sizes of the joint itself are practically restored, but the effusion in a short time returns to about the original amount. If the tubercles are more numerous, scattered through the whole membrane, the latter becomes thickened to a lesser or greater degree, in the former instance the surface being generally left smooth, but thin layers of granulation tissue extending from the borders of the articular cartilages towards their centres, and in the latter instance the whole synovial surface being covered by a thick, spongy layer of granulation tissue. In neither of these forms is there any excess of synovia, and they are probably different stages of the same process, the earlier stage being called a "pannous synovitis" and the latter being the "fungous synovitis" of Billroth.[1]

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[1] König, Senn's Tuberculosis of Bones and Joints, p. 20.

[2] Cheyne, Lancet, 1890, vol. ii. p. 1018.

[3] Senn, loc. cit., p. 136.

The ordinary coagulation necrosis of tuberculous tissues follows, but with varying rapidity, the pannous and fungous forms being slower and the hydrops more rapid. The result in each case is the usual serum containing the detritus of the tuberculous tissues, a pus-like fluid, but not pus. As this accumulates there is an extension centrifugally of the tubercular process, the ligaments are involved, and the tuberculous granulations erode, undermine, and detach the articular cartilages, and attack and invade the vascular bone, causing caries.[2]

There is another form of tuberculous infection of the synovial membrane in which the lesion may be confined to one locality and result in the development of subsynovial masses of tuberculous tissue, perhaps as large as a walnut, which project into the joint, occupying some of the normal recesses of the part, a tuberous synovitis; or the affection may be diffuse and the surface be covered with small papillomatous growths, some of which, becoming detached, form some of the so-called rice bodies.

The invasion of a primary synovial tuberculosis is slow, and the exact date of its origin may be with difficulty fixed upon. The patient, a child, will show the general symptoms of being in a vague way ill by loss of appetite, restless sleep, and mental irritability. Gradual disability of the limb comes on, finally amounting, if the joint is in the lower limb, to a limp, and at the same time there is complaint of pain; but this is an exceedingly variable symptom, for pain may be present from the start and severe, or it may be entirely absent. Generally it may be said that pain is more usually present in cases where the granulation tissue is less in amount, and is not so marked in cases of fungous synovitis.[3] Swelling of the joint affected occurs in proportion to the amount of effusion or of hyperplastic tissue changes; it is most in those cases where there is effusion, and after this the greater swelling is seen in fungous synovitis. With effusion fluctuation can be appreciated, and with a fungous synovitis there is also a sense of fluctuation upon palpation which may be mistaken for the evidence of fluid in the joint. A little exploratory aspiration with a small syringe will easily settle the question. The swelling may seem greater than it really is because of the atrophy of the segments of the limb contiguous to the joint. This atrophy, for a long time termed "joint atrophy," is a very constant and early symptom, and it affects all the tissues of the limb. The early date of its appearance and its amount led to its being, on purely clinical grounds, referred to a disordered nervous influence.[4] The mode of operation would be by the irritation of the articular nerves affecting their spinal centres and then reflexly the centres of origin of the muscular nerves.[1] It has, too, been shown experimentally that injuries to bones, more especially when they involve the epiphyses, affect the growth not only of that bone but of all the tissues of the limb.[2] In the bone itself the effect locally is to interfere with the proliferation of cartilage-cells, or in some instances, where the amount of infection is slight and the resistive efforts of the general organism are prompt and vigorous, the local irritation may stimulate constructive processes and result in overgrowth. Other experiments have shown that there are direct tissue-changes in the nerves from an inflamed joint, the number of nerve-fibres being diminished, and some of them having lost their axis-cylinders.[3] The total evidence shows that the complex phenomenon of growth and nourishment in a limb is affected through the spinal nervous system when any bone or joint becomes the seat of an irritative and destructive process. The atrophy is, however, much augmented by the disuse necessitated by the disease of the joint, and due weight must be given this factor in estimating, from the amount of atrophy, the probable amount of disease-injury in the articulation. On the other hand, two conditions tend to make the atrophy, whatever its totality, seem greater. One is the swelling of the diseased joint, its increased size making the wasted limb, by contrast, look smaller. The other is the increased size of the other limb due to its compensating extra use and hypertrophy, which makes it an unreliable standard of comparison.

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[1] Senn, loc. cit. [2] Ibid. [3] Ibid., p. 174.

[4] Paget, quoted by Park, Transactions of the American Orthopædic Association, 1891, p. 103.

Sequent in point of time there come deformities of the joint which are at the very first purely postural, but which later, as tissue changes take place, become definite alterations in the shape of the limb and the joint. These changes are not, however, seen in every form of synovitis, for they are absent in the hydrops form, and are not usually seen in the earlier stages of the fungous form, probably not appearing until there is some invasion of the bone, and their development and the explanation of it will be better discussed in connection with the symptoms of bone cases. The tale of symptoms of synovitis closes with the mention of abscess of the joint, and it is really but a more advanced stage of the tuberculous process. In the hydrops form the change would be noticed in the character of the fluid, which would become thicker and more like pus, less like serum. This change would of course be due to the admixture of the detritus of the tuberculous tissue, broken-down cheesy nodules, shreds of necrotic connective tissue, pus-cells, and granular matter, with the fluid of effusion.

In the pannous and fungous forms, where effusion is not common, the whole of the abscess contents are of this tissue-detritus material floating in a serum, and with the advent of this the pseudo-fluctuation would change to a real fluctuation, and an aspiration would prove the existence of fluid. Clinically, this symptom shows that absorption and elimination of the products of disintegration are not progressing sufficiently rapidly, and while its local evidence is the more obvious, its general bearing is the more important, for it shows that the patient's organism has reached the limit of its ability to cope with the disease, and that assistance of some sort is demanded. If there is no interference, the joint opens spontaneously at some point by a subacute inflammatory process in the walls of the sac and the contents are discharged, the sac being partially or wholly emptied, and the opening not usually definitely closing, but remaining as a sinus, discharging at intervals later accumulations.

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[1] Vulpian, quoted by Senn, Tuberculosis of Bones and Joints, p. 102.
[2] Ollier, quoted by Park, loc. cit., p. 100.
[3] Duplay and Cazin, quoted by Senn, loc. cit , p. 103.

Superinfection with the staphylococcus, development of a genuine abscess in a tuberculous joint, or empyema of the joint is a comparatively rare occurrence, and it changes at once the entire clinical picture, the acute symptoms, both local and general, of suppuration replacing the chronic symptoms of the tuberculosis.

The matter of treatment opens up at once the vexed question of protective *versus* operative measures. If it is possible to appreciate early in a case the existence of a local synovial tuberculosis, it is advised that the affected tissue be excised.[1] This is, perhaps, more especially applicable to the tuberous form when this rare type presents itself, but is at the same time of force in regard to the other forms where localized thickening and tenderness in conjunction with slight disability lead to a fairly definite diagnosis. It is very rare, however, that a case of synovial tuberculosis is seen in which this diagnosis can be made and early radical treatment followed. Failing this, the ordinary conservative treatment must be practised. This treatment is founded upon the fact, learned in the first place by experience, and confirmed later by biologic investigations, that there is in living tissues an inherent antagonism to all forms of infection, and that this antagonism is best exerted by tissues that are not functionating and are at the same time well nourished. The function of a synovial membrane is the production of sufficient synovia, and it does this in response to the motions of the joint when the membrane is alternately folded and unfolded, stretched and relaxed. Suspension of the function of the synovial membrane necessitates immobilization of the joint affected. This must be accomplished by splints, preferably properly fitted plaster of Paris splints, and to the immobilization should be added such auxiliaries as compression and counter-irritation to control not so much the tuberculosis as the accompanying inflammatory processes.[2] If these measures fail to control the process, the simple operation of arthrotomy—an incision into the joint—may be done on the same theory as a laparotomy is done in cases of tuberculous peritonitis, but the evidence of benefit to be expected from this is based on a single series of a few cases.[3] The preferable plan is to do an arthrectomy, an erasion of all the synovial and capsular tissues which are infected, and secure ankylosis of the bones thus deprived of their proper inter-attachments.

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[1] Cheyne, Lancet, 1890, vol. ii. p. 1094.

[2] Ibid., p. 1018.

[3] Ibid., p. 1019.

The technique of the operation is well described in the original article on this subject.[1]

TUBERCULOUS OSTEO-ARTHRITIS.

Tuberculous osteo-arthritis is the form of joint-tuberculosis which begins as a tuberculous osteomyelitis, with a later infection of the other joint-structures, the result being a panarthritis. Clinically, it is usually impossible to differentiate between cases that begin in the synovial membrane and those that begin in the epiphyses, one or both, unless the patient is seen early and the symptoms are very well marked, with no overlapping; and in the later stages, when all the tissues are implicated, the exact location which the initial lesion had in the joint does not matter. But as in cases of primarily synovial infection there is a brief time during which a radical operation may, by removing a small amount of tissue, prevent a general arthritis, so in cases of bone-infection there is a time during which the bone-focus may, if it can be located and is accessible, be removed and the integrity of the joint itself be preserved.[2] In patients under the age of puberty, when the various epiphyses are still not united to the diaphyses and the processes of cartilage proliferation and ossification are going on, the new vessels in these regions, "on account of their imperfect structure and irregular contour, furnish the most favorable conditions for the mechanical arrest of floating granular matter and the localization of pathogenic microbes."[3] Consequently at these ages the number of cases in which the bone is primarily affected exceeds those in which the lesion is primarily synovial, and this is an important matter to bear in mind when the question of the exact location is being considered preparatory to an early operation.

Bone cases in general are, however, like synovial cases in that they are usually secondary lesions, the primary affection being in some of the glands or viscera of the body. It is an exceedingly common thing to see in the necks of children with joint tuberculosis the scars of healed abscesses of the cervical lymphatic glands, and, according to Kummer, other and older foci than that in the bone are found in sixty per cent. of the cases; in the lungs, twenty-five per cent.; in other joints and bones, twenty per cent.; in lymphatic glands, ten per cent.; and in the pleura, two per cent.[4] These statistics are evidently not drawn wholly from children, else the percentage of gland cases would be larger, for "certainly in a very large proportion of all cases of tuberculosis in children it would appear that the first infection was in these structures, while common experience shows . . . that the glands may be involved without any local lesion in

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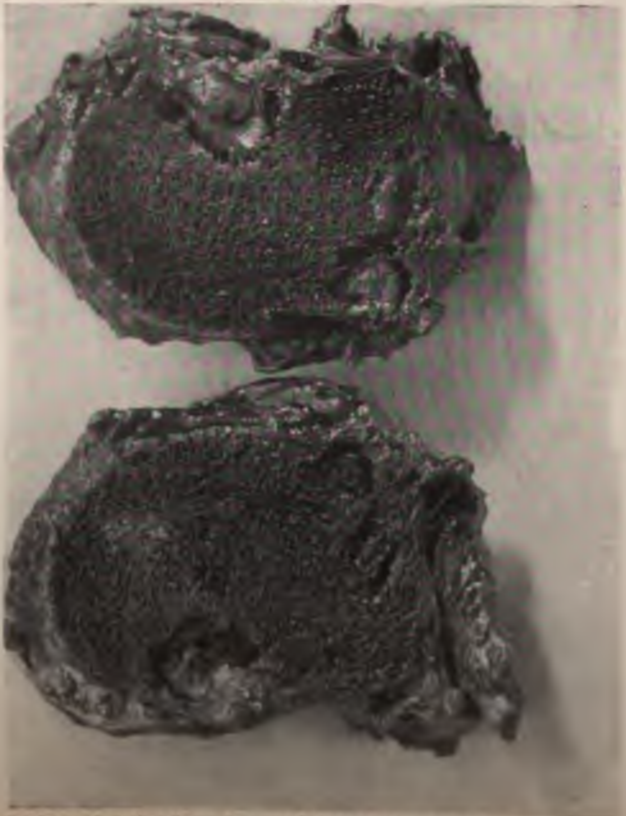
[1] Poore, vol. iii. p. 1214 of this Cyclopædia.

[2] Cheyne, Lancet, 1890, vol. ii. p. 1095.

[3] Senn, Tuberculosis of Bones and Joints, p. 65.

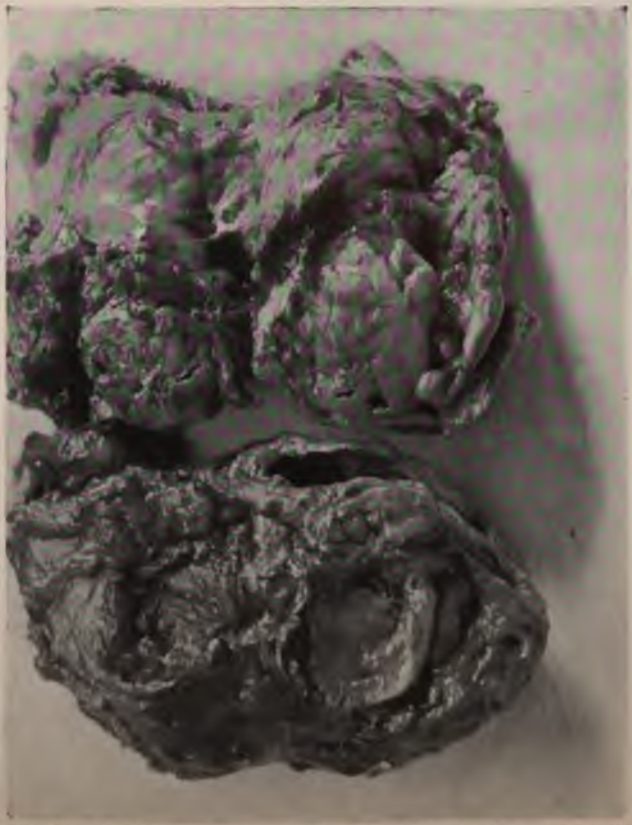
[4] Quoted by Senn, loc. cit., p. 92.

FIG. 1.



Surfaces of section through femoral lower epiphysis, showing tuberculous foci and rarefaction of bone.

FIG. 2.



Articular ends of femur (above) and tibia (below), showing fungous synovitis and cartilage exfoliation over tuberculous foci in bone underneath.

the lungs. Of one hundred and twenty-five cases examined by Northrup, the bronchial glands were tuberculous in every case."[1] As there are no definite symptoms by which any but an extravagant amount of infection of the bronchial glands can be recognized, it is important that the great percentage—one hundred per cent.—that they supply should be emphasized, to impress the fact that either in them or in some other of the glands or viscera of the body is a focus which antedates the occurrence of the bone disease, and which would probably persist even if the latter were healed.

Beginning in the bone, the disease process advances through the same series of tissue-changes as in any other organ, and the result always is the destruction of a certain amount of normal tissue. This is equally true of those cases where the local lesion is a granulating focus, from which the infection may spread and make other granulating foci, and of those where the lesion is an infarct due to the plugging of an arteriole by a tuberculous embolus, resulting in a necrosis and, later, the formation of a sequestrum. Around the granulating focus and around the sequestrum, if there is local dissemination, is always a region of osteo-porosis due to a decalcification of the trabeculæ, and this is likewise a destruction of bony tissue, and the result is that there is taken from the bone a part of its power of resistance to pressure and strain; the further the process advances and the wider the infection spreads the greater is this weakness of structure; as bone is a living tissue the function of which is to resist pressure and strain, the greater the structural weakness the greater the functional disability. (Figs. 1 and 2.)

If the granulating focus or the necrotic area is close under the articular cartilage, the latter will necrose and become detached and a direct way be opened into the joint cavity by which the infecting organisms pass in; or the liquefied caseous material (the contents of the tuberculous abscess, constituting the so-called tuberculous pus) may work its way to the surface of the bone and penetrate the compact layer inside the attachments of the joint-capsule, and the discharge into the joint-cavity take place in this way. If the entrance be through the articular cartilage and in a gradual manner, the synovitis developed may be distinctly chronic in its type; but if the entrance to the joint be by the discharge of a tuberculous abscess, a tuberculous synovitis of an acute type, with considerable effusion, may be lighted up, and in this case the signs would be so definite as readily to explain the condition. In either case a tuberculous osteo-arthritis results, with its carious and softened bone, unable to bear weight; its ligaments relaxed, incapable of holding the bones in their normal relations; and its synovial membrane ulcerated and containing a fluid mass of tissue detritus, or converted into a thick layer of granulation tissue, unable to fold and unfold, to stretch and contract, as the normal membrane,—in fact, a complete suspension of function.

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[1] Osler, American Text-Book of Diseases of Children, p. 109.

Clinically, this functional disability is the most prominent local symptom from the beginning to the end of the case, and in cases where the destroyed bone is not replaced by bony tissue, as in the ossification of a cicatrix, the disability persists after the cessation of the pathologic process as an actual or potential impediment, which determines to an accurate degree the amount of ordinary or extraordinary work the member may accomplish.

Special Symptoms.—There are certain definite symptoms of osteo-arthritis which are developed during the increasing interference with the function of the affected articulation, some of which have their cause in a spontaneous effort to protect the part both during rest and while it is at work, others represent the interference with the general nutrition of the limb, and still others testify to the special pathologic state. Pain plays a comparatively minor *rôle*, for it is often entirely absent, and is practically never the initial symptom; and yet there is in these cases an unconscious appreciation of the fact that there is something wrong with the joint, and an involuntary but none the less active carefulness in the use of it, and it is this that is first noticed by those around the patient. This carefulness is exactly what one would expect of an individual who had a definite knowledge of a weak point somewhere in his anatomy, and who intentionally adopted positions and modes of progression that would put the least work on the weak area. But this same carefulness is seen to be practised by little children who can know nothing of their physical state unless there is pain as a telltale; and even when it is practised by those older it is quite involuntarily and usually unconsciously. It seems right to infer, under these circumstances, that there is a sense of local well-being or of disability in the bones quite apart from pain, just as there is in the experience of every one a general sense of well-being in a state of health and a sense of discomfort in a state of disturbed functions or of disease. In cases of osteo-arthritis in the lower limbs the carefulness exercised in their use produces irregularity of gait, the affected limb being spared either by having the step on it made shorter or by having the body balanced on it in some particular manner, while the sound limb is used more vigorously than normally, being made to do the major part of the work of progression.[1] In tuberculous osteo-arthritis at either hip or knee or ankle this limp is the initial symptom. Later, when more bone-tissue has become affected and the bone-disability is greater, the watchfulness over the limb is practised not only during work, but during rest, and even during sleep, and definite positions are assumed and maintained which arrange for the bone and the joint, whether the latter is definitely involved or not, doing the least amount of work. Just as a bone is at work when it is resisting strain, so a joint is at work when it is moving, and at the stage when these positions are taken the conditions of the joint demand lessened work or cessation of work, restricted motion or immobility. If the organism is unaided, this restriction of motion or immobility is attained by an involuntary contraction of the muscles around the joint, which occurs when the limitation of permitted motion has been reached and prevents further motion, or which is present in all the muscles around the joint as a tonic contraction and practically inhibits motion. Later yet, when these positions have existed for some time, the postural deformities become definite deformities, changes taking place in the relations of the bones to each other by subluxations, or by alterations in the shapes and dimensions of the bones as different portions are destroyed by the tuberculosis.

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[1] Cf. Sherman, Cause of the Limp of Hip-Disease, Transactions of the First Pan-American Medical Congress, vol. i. p. 606.

It is not possible for these states to succeed each other in a joint without their having put a certain amount of unavoidable work on the bone, for no bone can have its motion in any direction checked or be prevented from moving in any direction whatsoever by the muscles around it without giving those muscles points of attachment. The last arrangement especially maintains, in the effort to immobilize, a certain definite pressure, and the work it entails on the bones increases and emphasizes the demand for rest for the part. In response to that demand the muscles are still more rigidly held in their tonic contraction, and in this way a pathological circle is developed, each element giving and in turn receiving an added stimulus. The pressure, furthermore, aids materially in the advance of the tuberculous process and the destruction of bone,[1] and increases in that way the totality of the resulting deformity.

Another symptom of tuberculous osteo-arthritis related to these etiologically is the occurrence of "night-cries," a phenomenon due to the temporary relaxation of muscles contracted to produce immobility and the occurrence of a prohibited motion. The result is an instant starting of the patient from sleep, together with a sharp cry. The awakening may be complete, and there may be a great complaint of pain, or the protective contraction may be so quickly resumed that the patient does not wake completely, but falls back into sleep. These "night-cries" or "night-startings" are held to be indicative of perforation of the articular cartilage and the exposure of the cancellous bone to direct pressure and friction, and their absence may, therefore, be considered as indicating, in part at least, that this has not yet occurred, and that the disease is still limited to the bone; but their absence by itself, and not supported by the concurrent testimony of other symptoms, is hardly to be so interpreted in all cases, for the same effect may be produced by a motion which puts distressing pressure on affected but not yet denuded bone, and, on the other hand, perforation may take place without developing this particular symptom.

Atrophy of the segments of a limb contiguous to the diseased joint occurs in every case of tuberculous osteo-arthritis, and has already been described in the section on Tuberculous Synovitis. As seen in a case of bone-infection it does not differ from that seen in the synovial cases. It is one of the permanent marrings of the limb, for it is never completely recovered from even in light cases that run a short course to practical recovery.

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[1] Bradford, Transactions of the American Orthopædic Association, 1893, p. 127.

With the atrophy there is a condensation of the panniculus adiposus, and in some instances an hypertrophy of it, the latter being present more especially over or near the tuberculous area in the bone.[1] This condition can very easily be appreciated by pinching the skin and subcutaneous tissues between the fingers, when the change in the texture of the panniculus is felt, especially if the normal texture over the sound limb be used as a standard of comparison. If the hypertrophy is marked and localized, a sense of false fluctuation may be got by palpation, leading to the idea of a tuberculous abscess. A little exploratory aspiration may then be needed to settle the doubt.

Incidental to the course of tuberculous osteo-arthritis of whatever joint may be the occurrence of tuberculous abscess. This occurs in a large percentage of cases; and while it has for this reason, and also because of its being a condition which is obvious, palpable, and unmistakable sometimes even by laymen, been accorded a prominent place in the history of the case, it must still be recollected that it is only a symptom of the pathological course of the disease, and represents the detritus of tuberculous tissue which has undergone coagulation necrosis, caseation, and liquefaction. If this process takes place so slowly that the products of disintegration, more especially the fluid parts of them, may be absorbed, removed, and eliminated as they form, there is no abscess; but if the process is more rapid, or if, being slow, it is long continued and the absorbent passages, vessels, and glands gradually become loaded and blocked, then there are accumulation of necrotic and liquefied products and the formation of a definite mass in the centre of the tuberculous tissues. Resulting from the breaking down of a single granulating focus, this accumulation is small, but the same process has been going on in contiguous foci, and these gradually coalesce, the tuberculous abscess increasing thus in size. It is possible for this mass to remain stationary, or nearly so, in the location where it formed, or it may slowly flow, moving always in the direction of least resistance, pushing its way along cellular planes between different organs, and finally appearing under the skin at some quite distant spot, forming a larger or smaller swelling, without heat or pain or redness. It is not uncommon for the subcutaneous swelling to be rather quickly formed by the sudden passage of a large amount of fluid from a deeper to this superficial location, but still there will be no heat nor redness, and usually no pain. At first the cavity containing this mass has no definite lining: it is simply a space in the areolar tissue formed by the hydrostatic pressure of the fluid. Later there is a definite layer of granulation tissue which is of the typical tuberculous structure, being composed of heaps of miliary tubercles, and contains the bacillus of tuberculosis.[1] Technically, this mass of fluid in the tissues does not constitute an abscess, for it is not pus. Under the microscope it is seen to be a turbid fluid in which fragments of dead tissue, particles of cheesy matter, shreds of fibrin and pus-corpuscles, and granular detritis are suspended; but custom still dominates the nomenclature, and this fluid tumor is still designated as an abscess. If the term is used it should always be qualified, and the fluid tumor be called a tuberculous abscess and the fluid tuberculous pus.

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[1] Alexandroff, Presse Médicale, December 9, 1896; quoted in the Journal of the American Medical Association, January 30, 1897.

The natural termination of the tuberculous abscess in its superficial location is that it shall increase in size until it ruptures, the opening in the skin being made by a subacute inflammatory process; after the discharge of the contents the cavity may contract to a sinus which furnishes permanent drainage for the original focus, and may last for months or years, or it may heal comparatively early, to remain closed or break down again. On the other hand, the abscess may, after reaching a certain size, gradually recede by absorption of the fluid parts, the solid portions condensing into a cheesy mass. The comparatively benign course of a pure tuberculous abscess may be interrupted by a secondary infection with pyogens, an event that may occur at any time in its history, even before it has opened, and the mixed infection inaugurates a much more serious state of affairs, the local condition becoming progressively worse, and such an abscess, if left undisturbed, inevitably goes on to spontaneous opening.

There is always depreciation of the general condition in cases of bone- and joint-tuberculosis; the appetite is capricious, breakfast being usually a light meal, while a noon dinner may be quite hearty. Even in the less serious cases there is a loss in body-weight, and it will be noticed that these children may do well as regards the disease, and increase in height, but remain the same in weight for a long time, probably until local repair is completed. The more serious the local condition the greater the constitutional disturbance, and this is especially true of cases with tuberculous abscess which has become secondarily infected, when the symptoms of acute or chronic sepsis are added to those of tuberculosis. In these cases, in addition to the more obvious changes, is developed a leucocytosis not found with pure tuberculous lesions, and its presence may consequently be taken as probable evidence of the secondary infection of abscesses which are still unopened.[2]

There are certain special therapeutic measures based on the symptomatology, and tending, all of them, to aid in the recovery of the part affected. The general law of rest for a diseased part is, of course, applicable here, and as the functions of these parts are chiefly mechanical, the rest must be a cessation from mechanical work. The idea of this can be got from the conduct of the patient, all of whose actions are bounded by the necessities of sparing the affected region. Recognizing first the structural change in the bone, by which it becomes weaker, one appreciates that to give rest to this part not only must the limb do no work in the ordinary sense of the term, but also arrangements must be made to relieve the bone of that pressure and strain which inevitably come upon it because of its position in the living organism; for, though this pressure is perfectly natural for the healthy bone, and borne by it without fatigue, it may, and usually is, more than the affected and weakened bone can bear without doing excessive physiologic work. Bone, too, is a living tissue, and work must physiologically be followed by fatigue and the need of rest. Tissues that are fatigued or physiologically exhausted are specially vulnerable and have diminished reparative powers. Work, then, whether it is ordinary mechanical work or the normal tone or tension of the special part, which has become excessive because of diminished strength, lessens distinctly the ability of the part to recover from the disease.[1]

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[1] Senn, loc. cit , p. 56.

[2] Dane, Boston Medical and Surgical Journal, May 28, June 4 and 11, 1896.

Furthermore, the pathogen under consideration, the bacillus tuberculosis, is non-motile; it has of itself no power to change its location in the tissues, and its movements are only as it is borne here and there in the current of the circulating fluids. Work of any part is accompanied by an increased amount of blood in it and by the more rapid movement of all the circulating media. It is inevitable that in the case of bone its normal use, with the changes in position and the constant concussions it is subject to, must increase and lessen the amount, hasten and retard the flow, and at times even change the direction of the current of the blood and of the lymph circulating in its rigid tissues, and by just so much it will tend to the local dissemination of the passive bacillus and the infection of a greater area.

What has been said here of bone applies with equal force to the synovial membrane and its functional activity and rest, and the mechanical distribution of the bacilli over its surface by any movements of the joint is too obvious to need more than the merest mention.

The attainment of the rest required—a rest so complete as to have a therapeutic value—is either by fixation alone of the joint affected by means of splints of any suitable material or by traction and counter-traction applied below and above the joint, the apparatus at the same time conferring on the joint a greater or lesser degree of immobility. The former method secures rest from ordinary work; the latter aims to give as nearly as possible complete physiologic rest, and in so far as it more nearly complies with the requirements of the proposition it is the superior; wherever it fails because of inaccurate or imperfect application, it is probably the inferior. That plan by which absolute fixation and just sufficient traction to make the part entirely passive in the economy could be combined would be the ideal local treatment.

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[1] Cf. Sherman, Journal of the American Medical Association, August 4, 1894.

As applied to an individual case of any joint, either method arranges for the correction of any postural deformity which may have been developed,—the former by simply gradually changing the position of the limb until it has been returned to the normal one, the latter by traction and traction-fixation in the direction and position of the deformity until with cessation of pressure comes rest from bone- and joint-work, and with the latter a suspension of the demand for immobilization by tonic muscular contractions. At this period there can be made without the slightest force and its possibly accompanying trauma a partial correction of the deformity,—at any rate, a correction to the point of an inhibiting muscular contraction; then could come a repetition of the traction in the altered direction, and the recurrence of traction and correction until the position of the limb was normal. But in that traction relieves the muscles of the need of holding the joint immobile, it does away with the possibility of their failing in their office during sleep, and relieves entirely the symptom of "night-cries," and often its efficacy may be tested by this symptom being held in abeyance.

The treatment of the tuberculous abscess is that part on which there is most difference of opinion and of practice; in truth, ample authority can be found to support any line of treatment that can be selected. The minor part that tuberculous abscess should play has been mentioned. It is a symptom only, and never should be permitted to assume the rank of a primary pathologic or clinical condition. On this understanding its mere presence may be disregarded, and the plan of treatment of the original lesion may be simply continued or somewhat modified; and this method has given a very satisfactory measure of success, the fluid portions first, and later, through fatty degeneration, the more solid portions being absorbed, until, finally, there was left no trace of tumor or swelling. Finally, at the end of the successful cases, the patient was considered to be none the worse for having had the whole of the mass of tuberculous tissue-detritus pass through the circulation on its way out of the body.[1] A larger number of those submitted to this treatment have spontaneous opening of the abscess, and of these the majority close after a longer or shorter period. In cases where a leucocytosis can be demonstrated, the ultimate opening of the abscess can be anticipated and the abscess incised, and in imitation of natural methods, and because these frequently lead to happy results, the operation may be limited to this incision, the evacuation of the abscess-contents and its final closure or persistence as a sinus being left to natural processes aided by the proper treatment of the original lesion. In contrast to this plan is that in which the abscess is directly attacked at a formal surgical operation; it is incised, its contents removed, its wall scraped and cut away, and it is either drained by being stuffed with gauze or it is sutured without drainage. In a minority of cases these abscesses heal and remain healed;[2] in the majority the symptom recurs from persistence of the original trouble. This lack of successful and permanent healings must not count against the operation if it is done with the proper object,—that is, of removing from the patient's organism an amount of necrotic material which is a menace to the general health. Whether the wound heals or not, whether a sinus remains or not, the object of evacuation is attained. Often the general improvement will reward the patient for undergoing the operation even if the nearest approach to healing is a small sinus with a little daily discharge. In the opening of a tuberculous abscess connected with any bone two things must be well considered. First, it is the rule for the condition exposed by the incision to be much worse than ordinarily would be supposed from the external appearances. There is hardly any condition in surgery more difficult to estimate accurately than the real extent and seriousness of a tuberculous lesion, and it is always wise to be prepared to do a radical operation on the original focus should it appear advisable after the evacuation of the abscess. Second, no class of cases suffers more seriously from secondary infection with pyogens than the tuberculous, and the operation, even for the opening and radical evacuation of a small abscess, must be done with full aseptic technique. It is the secondary infection and the possibility of its occurrence that must often decide the question for or against the operation. If pyogenesis and sepsis can be assuredly prevented, the very fact of an accumulation of necrotic tuberculous tissue is an indication for operation, for this necrotic tissue is in large part in the form of minute sloughs, each separated from the living tissues, and each is a foreign body. But if, on the other hand, asepsis is not a possibility, the condition after an operation and the occurrence of a septic infection may be much worse than with the tuberculous abscess intact.

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[1] Shaffer, New York Medical Journal, February 29, 1896.

[2] Lovett, International Encyclopædia of Surgery, vol. vii. p. 512.

The treatment of the tuberculous abscess and the tuberculous tissue itself by iodoform is a method which has been practised with varying degrees of success. Consequently there are some who advise and practise it and others who consider the procedure useless. The plan contemplates the injection into the cavity of an abscess which has been evacuated by aspiration and then washed out through the needle of a certain amount of iodoform suspended in glycerin or oil or dissolved in ether, expecting that the drug will exert a germicidal power on the bacillus. Or the injection is made directly into the tuberculous tissues, soft or bony, the drug being deposited in the closest possible contact with the pathogen. There is danger in the method lest pyogens be injected with the drug or in its vehicle, and sepsis ensue; consequently the substances must be sterilized before they are used. This, in the case of iodoform and glycerin or iodoform and oil, can be accomplished by keeping the mixtures in a boiling-water bath for two hours or longer, and, as iodoform does not volatilize below 115° C., there is no decomposition of the drug. The solution of iodoform in ether does not need sterilization. Of these the iodoform and glycerin is the preferable. In spite of favorable opinions as

Fig. 3.

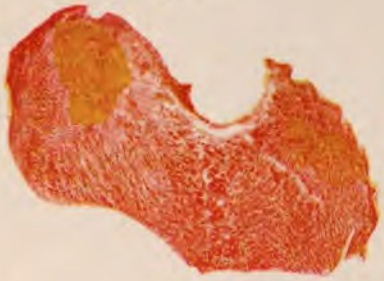
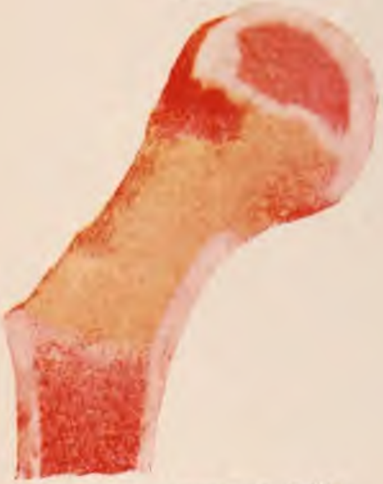


Fig. 4.



Tuberculous osteo-arthritis.

to its value,[1] the method has been a disappointment in the hands of most who have used it, and there are but few now who practise it.[2]

The early and late results of tuberculous osteo-arthritis are such as would be expected from the nature of the disease. In infection of the synovial membrane alone it is possible that there is frequently a "true recovery, though in others there often remains, somewhere or other, an encapsuled tuberculous mass. On the other hand, where there are deposits in the ends of bones, encapsulation is the rule in the great majority of cases which recover without operation or abscess."[3]

Of those who practically do recover and those who do not, the expectation may be that the number will be about even, and of the recovered cases the majority die later of tuberculosis of other organs, most frequently of the lungs.

TUBERCULOUS OSTEO-ARTHRITIS AT THE HIP.

This is next to the most common bone and joint location of tuberculosis, 30.2 per cent. of the cases of the first decade and 20.3 per cent. of those of the second decade being at the hip.[4] The location of the primary articular focus is usually in the neck of the femur, on the diaphyseal side of the epiphyseal cartilage, and from this point the infection may involve the rest of the upper end of the bone,—a local dissemination. Cases have been seen—but their diagnosis before the actual inspection of the sawn bone is practically impossible—where the infection was located in the epiphysis of the head alone, or in the trochanteric apophysis alone (Fig. 3), or in this and also in the epiphysis of the head (Fig. 4). Diagnosis of a lesion as having been originally in the pelvic bones is impossible, and yet this is an occasional primary location.[5] A tuberculous synovitis at the hip in an individual under puberty is rare.

Symptoms.—The symptoms are best studied individually, and there are but few points to be here touched on. Almost invariably the earliest symptom is the limp. Early in the disease, shortly after the pathologic process has begun, the limp is not constant, but is noticed only in the morning, or after some period of rest, or when the child is fatigued. It is then a slight increase of the normal lateral sway of the body towards the affected side while the weight is on that limb, and nothing more, and may be so insignificant as to require special observation of a nude child walking at a moderate gait to exhibit it. Later, as muscular rigidity lessens joint-motion,—the area of affected bone having increased,—the pelvis can be seen to follow the movements of the limb, the lateral sway is greater and is easily ap-

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[1] Senn, Tuberculosis of Bones and Joints, p. 247 *et seq.*

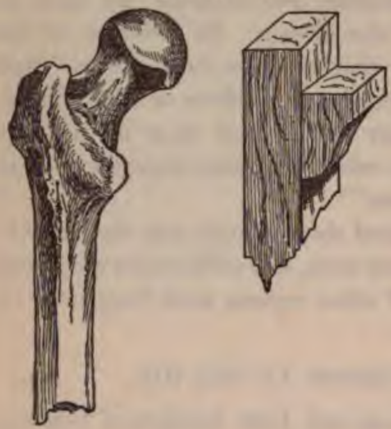
[2] Cf. Sherman and Walker, Transactions of the American Orthopædic Association, 1895, p. 47.

[3] Cheyne, Tuberculous Disease of Bones and Joints, p. 127.

[4] Ibid., p. 115.

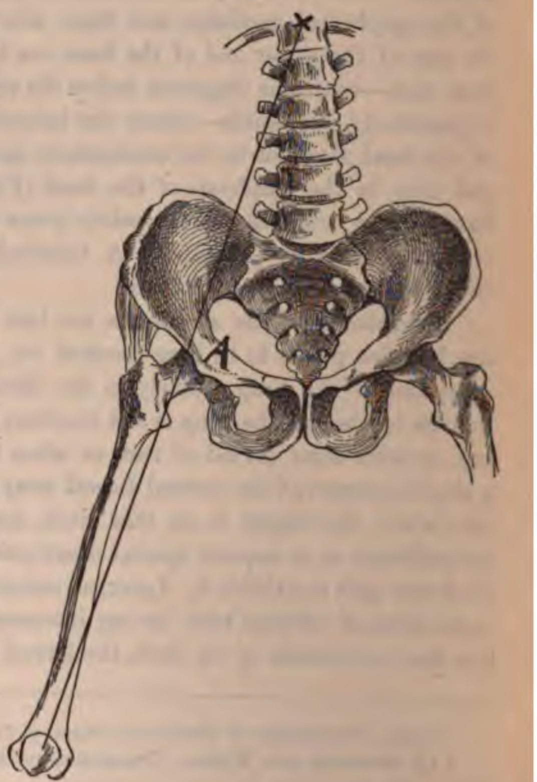
[5] Bradford and Lovett, Orthopædic Surgery, p. 256.

FIG. 5.



preciated, and the step on the limb is shortened. Still later, when the limb has passed into a position of adduction, the amount of flexion has so increased that the heel cannot reach the floor, and the patient walks on the toes with a decided up-and-down motion from step to step; the lumbar spine is thrown into violent lordosis and at the same time sharply flexed laterally to compensate the flexion and adduction and keep the body over the limb and the femoral head, and there is a little swing of the whole body on the tiptoed foot to accomplish some progression. These rather complex motions, which take place suddenly and in a jerky manner, and are typical in their combined occurrence of tuberculous osteo-arthritis at the hip, are reducible to two elements, both of which tend to lessen bone- and joint-work,—namely, lateral sway and restricted motion. The mechanism of the hip-joint is such that the body-weight is carried on the head and neck of the femur, jutting out from the shaft, as a bracket (Fig. 5); furthermore, the point in the body receiving the support is eccentric, in the acetabulum, and the upright position of the trunk is maintained by the action of the thigh abductors. This makes of the os innominatum a cantilever, its fulcrum in the acetabulum, its weight carried by its articulations with the sacrum and its mate, and its anchorage represented by the thigh abductors. Any motion tending to shorten this lever and bring the centre of gravity of the body over the fulcrum will lessen the work done by the head and neck of the femur in bearing the weight, and this is exactly the

FIG. 6.



effect of the lateral sway (Fig. 6). The combination of this lateral sway and the inhibition of motion, which has already been explained, constitute the irregularity of the gait which is known as the hip-limp.[1]

Quite as early as the limp suggests to parents the possibility of something wrong and leads to an examination of the child, there is some atrophy discoverable. If the fact is not appreciable to the eye, the tape will always show it. Certain definite measurements are commendable, because of the possibility of making them at uniformly the same place for purposes of comparison. The first is from the tip of the anterior superior iliac spine around the thigh and back to the same point; the second is directly around the thigh at the level of the gluteal fold; the third is directly around the thigh, midway between the perineum and the inner condyle of the femur; and the fourth is around the lower part of the thigh, just above the condyles. These measurements should be taken with a steel tape, which must be pulled just tight enough to lie in contact with the skin without making any compression. The atrophy of the buttock, which results in the dropping of the gluteal fold and the loss of the natural expression, must be considered part of the "joint-atrophy" of hip disease. The first of the measurements mentioned is an excellent gauge of swelling around the joint and in the groin.

The condensation or hypertrophy of the panniculus adiposus is usually well marked, and lessens the amount of apparent atrophy. This condition has been claimed to be the initial symptom of the local lesion, and the amount of it to be an index of the severity of the disease. Its constancy as a symptom is certain, and its absence may be taken in doubtful cases as counting against an osteo-tuberculosis.[2] Its gradual disappearance may likewise be indicative of the progress of repair.

During the progress of the disease the limb assumes certain positions which are peculiarly definite and which undergo changes with almost mathematical regularity. The motive for their assumption is the desire to lessen work for the bone and joint. In health the position of rest for the hip is one of slight flexion and abduction and rotation outward, thus relaxing the tension of the anterior part of the capsule and the tonic pull of the fully extended flexors and lessening the thrust of the head into the acetabulum entailed by full extension and normal adduction. This is the first position assumed by the limb when there is a tuberculosis in the femoral neck, and as the disease progresses the motion in all three directions is increased, the resultant being a position still more removed from the line of direct force. This position of flexion, slight abduction, and outward rotation makes the limb look to be longer, and if the limbs are put parallel the appearance of increased length is still more marked. Mechanically this appearance is due to the fact that abduction has taken place at the hip-joint and is fixed; that the motion of adduction of the limb with no motion at the hip causes a shifting of the centre of motion from the hip to the lumbar spine and a canting of the pelvis, the side of the disease descending and thrusting the limb downward, and the opposite side ascending and pulling its limb upward.

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[1] Sherman, Causes of the Limp of Hip Disease, Transactions of the First Pan-American Medical Congress, vol. i. p. 606

[2] Alexandroff, Presse Médicale, December 9, 1896; quoted in the Journal of the American Medical Association, January 30, 1897.

The limb may remain in this position a long period of time, but as the amount of flexion increases the functions of the other muscles change by that motion, and the whole mechanical arrangement around the joint is modified, for by this time there has usually been some destruction of the neck of the femur and also of the upper posterior portion of the acetabulum, and the limb gradually passes on into the position of flexion, adduction, and rotation inward, where it is still further removed from the line of direct pressure. In the instinctive selection of these positions there is a vital as well as a mechanical factor concerned, and the exact part played by each is difficult to determine; the chief point to be noted is that, with progressive disability on the part of the joint-tissues, the limb assumes positions which withdraw it more and more from the possibility of efficient function. The position of adduction is one that gives the appearance of being shorter even in a limb of normal length; for if, with rigid adduction at the hip, the limbs are put parallel, the centre of motion has again shifted from the hip-joint to the lumbar spine, and the canting of the pelvis has drawn up the adducted and thrust downward the other limb. At this period, however, there is usually some definite shortening of the limb due to the destructive processes taking place in the joint, the reflex nervous influences checking development, and the atrophy of disuse.

There are certain atypical positions assumed in which the leg is rotated inward instead of outward, or in which adduction does not take place at the usual time, but abduction increases. These are possibly due to uncommon locations of the lesion in the bone, or to the effects of confinement to bed and the changed mechanical relations of the parts when the patient is supine.[1]

The mensuration of the possible motion of an affected joint is of value, giving an approximately accurate idea of the joint condition, and enabling one to estimate progress by comparative measurements taken at different times. With the limb in line with the trunk, the angle of extension of the femur on the pelvis is considered to be one hundred and eighty degrees. Flexion is measured from this point, and may be any amount up to about one hundred and forty degrees, when thigh and trunk come in contact. Extension is measured from the point of full flexion, and as this is about forty degrees short of one hundred and eighty degrees, extension begins with forty degrees to its credit, and when the limb is at right angles to the trunk it is considered to be in the position of ninety degrees of extension; and this position is, too, one of ninety degrees of flexion. Every other position the limb can assume in the same plane has also two readings, depending on whether one is measuring flexion or extension. Abduction and adduction are estimated by measuring the angle of the arc travelled by the limb moving from a position of right angles to a line passing through the anterior superior iliac spines.

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[1] Phelps, Transactions of the American Orthopædic Association, 1892, p. 261.

To measure flexion the patient lies on a firm surface and the knee of the unaffected side is held firmly down. The limb of the affected side is flexed at the hip so far as the joint-condition permits, and the arc through which it passes is measured by Knight's goniometer. This reading will often include the amount of hyperextension at the sound hip, for the pelvis will tilt up with the affected thigh when the limit of motion has been reached; but, as this is a constant value, it does not disturb the comparative measurements.

To measure extension the pelvis is fixed and prevented from tilting downward, with accompanying arching upward of the lumbar spine, by fully flexing the sound hip and locking the forearm into the flexed knee, according to Thomas's method. The arc through which the diseased limb can now be moved in the direction of extension is measured by the goniometer, the reading being taken as if the limb had started from a position coincident with the trunk. In measuring abduction the pelvis is fixed by abducting the sound limb to its limit. The arc through which the diseased limb can then be abducted is measured, taking the middle of Poupart's ligament as the centre of motion.[1] In measuring adduction the pelvis is fixed by adducting the sound limb to its limit, always passing it behind the affected limb.

In a majority of cases of tuberculous osteo-arthritis at the hip tuberculous abscess occurs, and the event has the usual pathologic and clinical significance. Usually the fluctuant tumor appears anterior to the trochanter, but it may be behind that bone, or in the region of the adductors, or even above Poupart's ligament, or two or more abscesses may be in two or more of these places at once. From the superficial major cavity a comparatively small sinus leads to the bone or to the joint, and if to the latter, it usually enters it behind the neck of the femur. In cases which have gone to the stage of abscess-formation that fact is of chief importance, and the details of location and size are of less moment.

Pain is a symptom upon which no dependence can be placed, for it may be entirely absent or be present only for a time, or it may be the predominant symptom through the whole case. When pain does exist, it is usually the expression of some injurious pressure on the affected area; it can then be entirely relieved by protective treatment. This is true of the pain which occasions the "night-cries" and also that which is complained of in the daytime.

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[1] Judson, Transactions of the American Orthopædic Association, 1895, p. 252.

The disease once having begun, it may run one of four courses, the classification being purely a clinical one.

"These four types are:

"(*a*) The destructive form, where the disease is rapid, severe, and but little influenced by ordinary treatment; extensive infiltration of the soft parts takes place, and in most instances the disease passes on to a fatal issue.

"(*b*) The painful form, where pain is a prominent symptom and exacerbations are common.

"(*c*) The quiet or painless form, where pain is an unimportant factor or is entirely absent.

"(*d*) The transient or ephemeral form, where the symptoms are mild and the course of the disease is run in a few months."[1]

The type assumed must be the resultant of the two forces, the attacking organism and the resistant power of the individual. In the "destructive form" there is very probably a mixed infection with the staphylococcus, or possibly a pure tuberculosis, in an individual with other deposits and very feeble resistance. The "painful form" is by far the most common, and it may have any of all possible endings. The "quiet or painless form" is a well-recognized type, very likely, however, to be overlooked in the earliest stages by a careless observer. Favorable cases lead invariably to ankylosis,[2] and the recognition of the type is valuable for prognostic purposes. The "transient form" will cause a later doubt as to the tuberculous character of the trouble, but observations of patients who have recovered will usually show the unmistakable stigmata of a past epiphyseal or joint affection in a limitation of motion in some direction, more likely in the direction of internal rotation, or in some atrophy or shortening.

The treatment of tuberculous osteo-arthritis at the hip, as it is practised in the United States, calls into use the principle of traction to secure rest for the diseased bone and joint, the apparatus conferring at the same time a certain amount of immobility. The method known as the Thomas method is still the only rival of the traction plan, and it "is intended to immobilize the hip-joint by a direct antero-posterior leverage action. By this action it reduces the deformity, or opposes a tendency to deformity. By its weight and rigidity it steadies the inflamed articulation and quickly relieves muscular tremor."[3] The method cannot, however, entirely eliminate work by the affected bony tissue, and so far it is deficient, even though it confer immobility. Comparative tests, to be valuable, should require a large number of cases similar in general constitution and environment and treated by equally careful surgeons, for either method may fail in careless hands when under other circumstances it should succeed. The splint and the method of application are described in the original article.[4]

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[1] Lovett, Transactions of the American Orthopædic Association, 1892, p. 60.

[2] Marsh, Lancet, 1893, vol. ii. p. 791.

[3] Ridlon and Jones, Chronic Joint Disease (preliminary papers), p. 106.

[4] Poore, vol. iii. p. 1183 of this Cyclopædia.

In the consideration of the method of traction the two chief points are the amount and the direction of the traction. Of the former it can be said at once that, to be most thoroughly efficient, the amount must be enough to remove all work—that is, all pressure—from the bone. This, then, should demand distraction. Experiments[1] seem to show that this is feasible with amounts that can be tolerated a long time. The same experiments showed that the greater the traction the more certain the distraction. It is right, then, in all stages of the disease when traction is used, to apply all that the patient can stand, having regard here to the skin as well as the other tissues, and for children at the ages at which this disease generally begins that amount will usually be found to lie somewhere between five and fifteen pounds. This refers to traction in the line of the shaft of the femur, whatever may be the position of the joint, and objection has been raised to this direction on account of the obliquity of the neck of the femur with the shaft, and also because certain muscles around the hip-joint are horizontal and not longitudinal in position;[2] consequently, traction should be in the line of the neck of the femur, it representing more nearly the resultant of the forces of the longitudinal and horizontal muscles, and theoretically nothing can be said against the plan, but sufficient experience with it is yet lacking to show it an improvement. Moreover, some doubt is cast on the necessity of the lateral element by the fact that the acetabulum "wanders" upward and backward, and that this extension is not deeper than the original socket.

The method of grasping the limb is of moment when the maximal tolerable traction is to be used. A rubber plaster of good adhesive powers and free from chemical irritants must be selected. The plaster must be adherent to the limb from just below the perineum and trochanter to the ankle. It is well to adjust the thigh portion first, make a little traction, and then adjust the leg portion, thus insuring a greater pull above the knee than below it. Before the attachment of the plasters the leg must be washed in warm water and soap and then with alcohol to make the skin quite clean. In removing the old plasters lucine can be used to detach them, and then all old gum and dirt should be washed off with it, and after that use warm water, soap, and alcohol to prepare the leg for the new plasters. Usually one set can be kept on from three to four weeks. Excoriations occurring under or at the edges of one set will heal under another if they are well cleaned and then dusted with bismuth subiodide. On skins too tender to stand this the following method may be tried. Pieces of white felting are cut of the proper size, and along the whole length of one side of each is stitched a piece of ordinary webbing, long enough to extend below the foot. In between the skin and the felting—the latter held just in position to be immediately applied—is thrown a fine stream of the following mixture:

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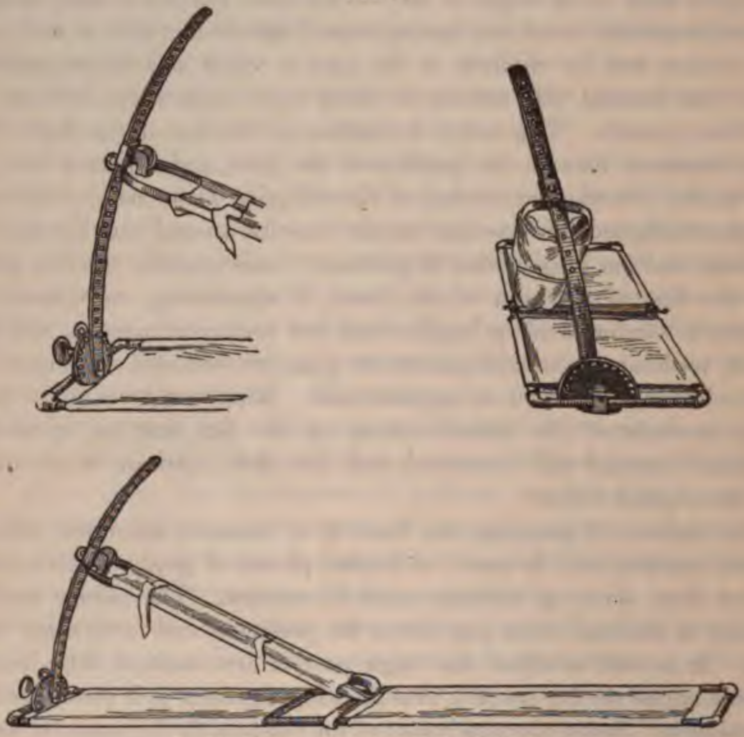
[1] Bradford, Transactions of the American Orthopædic Association, 1893, p. 127.

[2] Phelps, New England Medical Monthly, January, 1894.

cera flava, resin dammara, colophonium, of each, 10; terebinth., 1; ether, alcohol, and ol. terebinth., of each, 55. Mix and filter. Around the feltings are put gauze and crinoline bandages. The amount of adhesive mixture is slight, and risk of eczema is lessened. The feltings do not slip, and yet are easily removed. They are to be removed once a fortnight, the leg washed, and the same pieces of felting reapplied.[1]

FIG. 7.



Bradford frame with adjustable inclined plane.

Traction can be made and continued either with the patient in bed or up and about. The former plan is imperative in painful or otherwise serious cases, or if there is a postural deformity. The bed must be smooth and firm, preferably a woven wire mattress and some blankets, but no hair mattress. If there is a postural deformity, the limb is put on an inclined plane, which holds it so that the whole trunk is naturally supine, and traction is then made in the direction of the axis of the femoral shaft. Any attempt to correct the deformity at this time results in increasing the pressure in the joint and on the bone, for the flexors and abductors are rigidly contracted, and, as force in the direction of extension is used at the knee, the insertions of the flexors and abductors supply a fulcrum and the head is thrust more firmly into the acetabulum. After a few days of traction he shaft in the position of deformity the tonic contraction ides and a certain amount of correction is possible, and eated to the normal position. A most useful apparatus s the Bradford bed-frame, made of gas-piping and canvas, size for the child, and fitted with a holder for the leg, adjust- deformity. This may or may not be fitted with perineal coun- n, and the traction may be by elastic or by webbing fastened to d apparatus, or by a weight and pulley. On a frame of this sort hild may be moved about the house, itself motionless, or may go out doors, and even spend most of its time there. One excellent point in he use of these bed-frames is that the child does not have to be propped with the pelvis up on a bed-pan for the act of defecation, an opening in the canvas arranging for all this without disturbing the child. (Fig. 7.)

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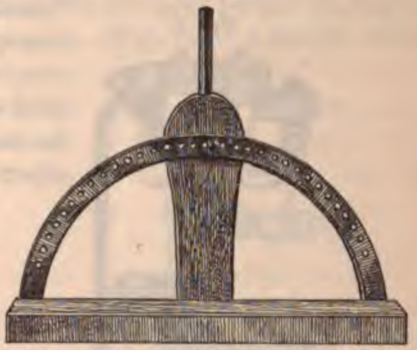
[1] Hoffa.

If, after the correction of the flexion and abduction, there is left some rotation outward, the foot should be fastened to the adjustable sole of a foot-holder. (Fig. 8.) The fastening is to be done with a wetted crinoline bandage or a single plaster of Paris bandage, and as this immobilizes the tarsal joints, and rotation does not take place at the ankle or the extended knee, any turning in or out of the foot causes rotation at the hip. By successive slight inward rotations, made during the continuance of longitudinal traction, the position can be easily corrected.

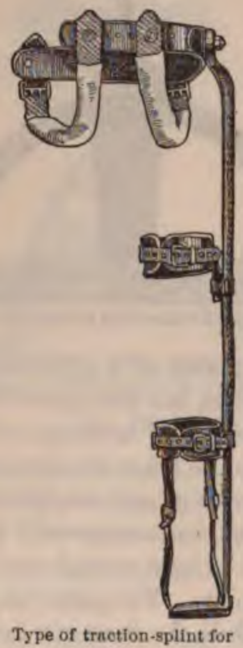
FIG. 8.



Foot-holder for correction of rotation of limb.

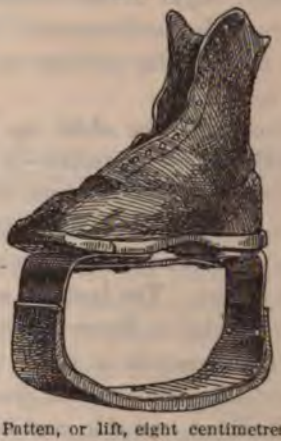
Traction with the child up and about—ambulatory treatment—is obtained by means of a splint or brace. There are many forms in which this brace appears, but the type of all must be the same: a pelvic band of steel, perineal straps of webbing or leather, a side bar of steel reaching from the pelvic band to below the foot and then turning it under. The best of to-day is the evolution and simplification of the Davis-Taylor or Sayre splint. It does not matter especially how the detail of the apparatus is made, so long as it is light enough and strong enough and takes into account the following points. The pelvic band must encircle the front, one side, and back of the pelvis, the front arm lying just below the anterior superior iliac spines, and the side being just above the trochanter. The posterior arm may be on the same level or may be dropped so as to cross the sacrum as low down as possible. A well-fitting pelvic band of this pattern prevents, in a measure, the tilting forward of the pelvis under the pull of flexion at the hip. The perineal straps must be so adjusted that they leave plenty of room between them for the genitalia in front and yet do not press against the tendons of the adductors at the sides. Between the thighs they must each pass backward so that they lie under the tuberosities of the ischia and then ascend to the back arm of the pelvic band. In this way the counter-traction is made, in great part, on the tuberosities of the ischia and not on the perineum chiefly, and the point is emphasized here because the ordinary instrument-maker puts on the market an apparatus in the construction of which none of these points are considered, and usually arranges his perineal straps so that one pulls directly across the anus. The joint between band and bar must be solid, and not the adjustable type, for the splint should not be put on a patient who has any postural deformity. The bar must carry, at a point opposite the junction of the middle and lower thirds of the thigh, a light steel band to encircle and steady the thigh, and another below at a similar place in relation to the leg. There is no absolute need for a ratchet, and often a decided advantage in its absence. The straps below, to buckle to the traction plasters, should be of the finest rawhide and punched with holes five-tenths of a centimetre apart to permit of an accurate adjustment of the traction. (Fig. 9.) In all cases but those where the disease is of the "transient" or "painless" forms the child who wears a traction splint should go about on crutches, and even in these cases this should be done if the child weighs over forty-five pounds. Against the use of the splint as a walking-splint the objection that when the child's weight is on the splint the traction is lessened, to become efficient again when the child steps on the foot, is valid, for under this use the traction is a series of intermitted pulls, and not a constant one;[1] but, perhaps in spite of this, the treatment is an efficient one. The child wears on the foot of the sound side a patten of light steel, eight centimetres high (Fig. 10), and if the

FIG. 9.



Type of traction-splint for hip.

FIG. 10.



Patten, or lift, eight centimetres high.

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[1] Ridlon and Jones, Chronic Joint Diseases (preliminary papers), p. 104.

splint is to be used as a walking-splint the length of the side bar must be sufficient to match this. If greater fixation at the hip than is given by this splint, properly applied, is required, the side bar can be extended upward to the side of the thorax and the latter be encircled by a steel band, or steel straps may extend upward from the posterior arm of the pelvic band, lying close to the skin on either side of the spine, and carry a thoracic girdle. The splint is to be worn day and night, but morning and evening the perineal straps are to be loosened, one at a time, and the skin washed with alcohol and water and then powdered with a talcum powder.

In cases where it is desirable to use traction in the axis of the femoral neck, the splint devised and recommended by Phelps is suitable. In this the side bar is on the inner, not the outer, side of the leg, and at its top it carries a padded and leathered metal ring, which encircles the extreme upper part of the thigh and, by pressing against the tuberosity of the ischium, affords counter-traction. The splint has also a pelvic and thoracic girdle, and finally has a special bar which descends from the pelvic girdle, above the trochanter, to a point opposite the upper part of the thigh, and by this the lateral traction is developed. (Fig. 11.) A similar effect is obtained by a simpler apparatus, also devised by Phelps, in which the traction-bar is wholly on the outer side of the leg. (Fig. 12.)

FIG. 11.

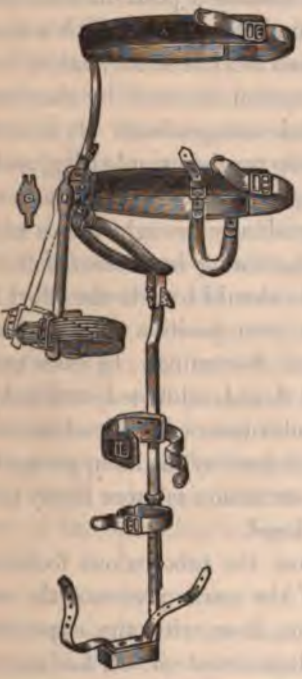
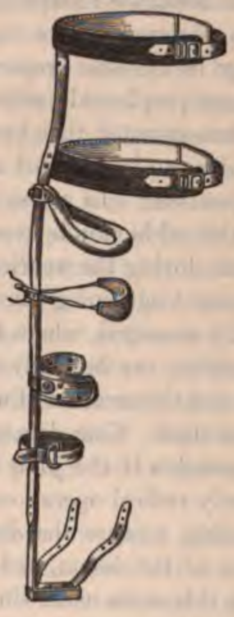


FIG. 12.



This treatment must be kept up uninterruptedly until the patient is definitely convalescent, and then the traction-splint, patten, and crutches may be discarded for a convalescent splint. This is in effect an ischiatic crutch, and does no more than protect the bone and joint from excessive work and accidental jars, the whole weight of the patient falling on the femoral neck only at the end of the step. This splint must be worn for fully two years after the disappearance of active symptoms.[1] All treatment may be discontinued only when there is no expressive attitude or gait (cautious) and no reflex tonic contraction of muscles protecting the joint. But in all cases, it must be remembered, and at all stages of convalescence, or even at any time after apparent perfect recovery, relapses are possible.[2]

Under this treatment a certain number of cases will recover with practically perfect joints, the slight limitation of motion being in usually unused portions of the motion-arcs; or they may recover with joints more or less crippled by destruction of bone and ligament, and with motion limited by changes in the articular surfaces and cicatricial contractions, and often the limb may be held rigidly in positions, such as adduction and flexion and inward rotation, which prohibit useful function. Ordinarily, after the lapse of a few months or a couple of years, the range of motion is lessened and the postural deformity greater. The aim of mechanical treatment is that the joint shall recover with the limb in a satisfactory position, but after treatment has ceased the limb must assume a position determined by the then mechanical conditions in and around the joint. With a shortened femoral neck, the tendency to adduction and flexion is inevitable, for these groups of muscles have not had their function lessened by shortening of leverage in the same proportion as have their antagonizers. It is noticeable that these people walk with a very short step on the crippled leg, and it has been demonstrated that by teaching a proper rhythm of step the positional deformity can be lessened and the limb gradually brought into a more efficient position.[3] In a case where actual shortening has occurred the knowledge that adduction is eventually probable should lead to the effort to keep the limb during the wearing of the splint in a position of abduction, that functional lengthening may offset the actual shortening. In cases untreated or badly managed, where the limb is left flexed, adducted, and ankylosed, the position can be easily rectified by a subcutaneous subtrochanteric osteotomy and the creation of a compensating deformity in the upper part of the femoral shaft. Complete and permanent correction is more likely to follow this operation if the joint be firmly ankylosed.

Early radical operations directed against the tuberculous focus are disappointing, because the deep situation of the parts prevents the accurate location of the lesion, and such operations, done with the expectation of finding this at its usual site, may fail to demonstrate it, or, finding and removing this, may fail to find others lying deeper. Relapses of these cases are common.[1] The operation, if it is thought wise to undertake it, gains access to the neck by trephining the trochanter; in ordinary cases all of this portion is osteoporotic and very easily removed by a sharp spoon. The cavity should after an aseptic operation be allowed to fill with blood-clot and the skin be sutured. If there has been any chance infection of the wound, it should be packed and allowed to heal by granulation.

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[1] E. H. Bradford and R. W. Lovett, in The Children's Hospital, Boston, 1895, p. 176.

[2] Shaffer, New York Medical Journal, 1891, vol. liv. p. 510.

[3] Judson, American Medico-Surgical Bulletin, January 1, 1895.

What has been said of tuberculous abscess in general in another part of this work applies to that occurring in the course of hip-joint infection: it may, in patients who are otherwise doing well, be left alone; or if a leucocytosis develops, it may be incised and allowed to empty, the sinus to afford drainage for the joint-disease; or, in exceptional cases, it may be formally opened and its walls dissected out; but this last operation should not be undertaken unless full provision is made to extend the operation to the joint and to remove any sequestrum which may be there, or all diseased tissues, whether detached or adherent. Any plan short of a radical treatment of the original joint-lesion, when radical treatment of the abscess is practised, leaves a fresh wound opening outside on the skin, a possible source of pyogenic infection, and opening inside on a certain source of tuberculous infection. This leads at once to the question of operation on the joint. Arthrectomy of the hip is unsatisfactory in that it is "impossible to decide as to the limit of the disease, and the continuation of the process ultimately demands excision."[2] The operation of excision of the hip may be done for two objects. It may be done to cut short the pathologic process by removing diseased tissues and secure an earlier healing, with a limb that must be crippled, but which had, at best, no chance of ever being more than partially competent for support and progression, or it may be done as a simple life-saving measure. Against the operation done for this latter reason nothing can be said; against it as done for other reasons, it is urged that the removal of the upper epiphysis of the femur in children interferes seriously with the growth of the limb both because of the loss of what growth would have taken place there and because of the reflex interference with growth at the lower epiphysis, as well as that of all the other tissues of the limb; that the operation often fails to end the disease, sinuses remaining for long after; that the best result after an incision is inferior to an ankylosis with the limb in a proper position; and that the mortality of the operation is higher than that of protective or mechanical treatment.[3]

These statements are true and must count against any but a life-saving operation, and the whole question, then, is resolved into one of time. How long may a life-saving operation be postponed? How soon may a life-saving operation be done? The earlier the operation is done, the better are the chances for quick, sound healing with a fairly useful limb; the longer it is delayed, the worse is the outlook in every particular. The decision for or against it must lie in the estimate of the powers of the individual patient to recover after a long period of illness. If the patient carries his local disease well, the general condition not being markedly depreciated, the operation may be withheld; but if at any time the individual resistance fails, if the disease begins to gain on the patient either in the joint or in some visceral location, if conservatism is not conserving, the operation is indicated, and under the circumstances is a life-saving operation, no matter how early it may be done. (Fig. 13.)

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[1] Poore, New York Medical Journal, 1892, vol. lv. p. 449.

[2] Willard, Journal of the American Medical Association, 1895, vol. xxv. p. 184.

[3] Lovett, Transactions of the American Medical Association, Surgical Section, 1894, p. 138.

In the detail of the operation it is always advisable to remove the trochanter. The additional shortening from so doing is but little, nor is much control of the limb conserved by retaining it. The wound should always be packed with gauze and made to granulate, and the presence of the trochanter is a great obstacle to dressings and to proper drainage. Other points in the technique of the operation do not need to be repeated here.

Amputation at the hip is to be done when the amount of disease in the femur and pelvis precludes the likelihood of benefit following an excision, or when, an excision having been done, the disease has still advanced and the condition of the patient has not been improved, there being in neither instance evidence of other diseased organs which would make the case appear to be a fatal one shortly after the operation. The operation is usually done on extremely bad cases, and, in that it removes a large part of the infected tissue, it gives the patient a better chance of combating the remaining disease. Modern technique has much lessened the mortality, that of the last series of cases collected being but fourteen per cent.[1] In the management of the hemorrhage every effort must be made to save all the blood possible. This is best done by Wyeth's method of transfixing the muscles close to the pelvis by long needles and applying an elastic tourniquet above them. The detail of the formation of the flaps is controlled in great measure by the location of abscesses and sinuses, and the incision should be planned to remove, if possible, all the infected soft tissues and still leave enough to cover the stump. The exarticulation may be done early, as if the intention was to do an excision, or the limb may be amputated very high, as in Wyeth's plan, and the head be dissected out afterwards. After the removal of the limb the acetabulum must be cleared of all diseased tissues and the flaps sutured, arrangement being made for drainage of the cavity of the acetabulum. Another plan permits the flaps to be sutured at either end, but leaves open a space in the centre, through which the cavity of the acetabulum may be packed with gauze,—a preferable method of treating a tuberculous bone-cavity.

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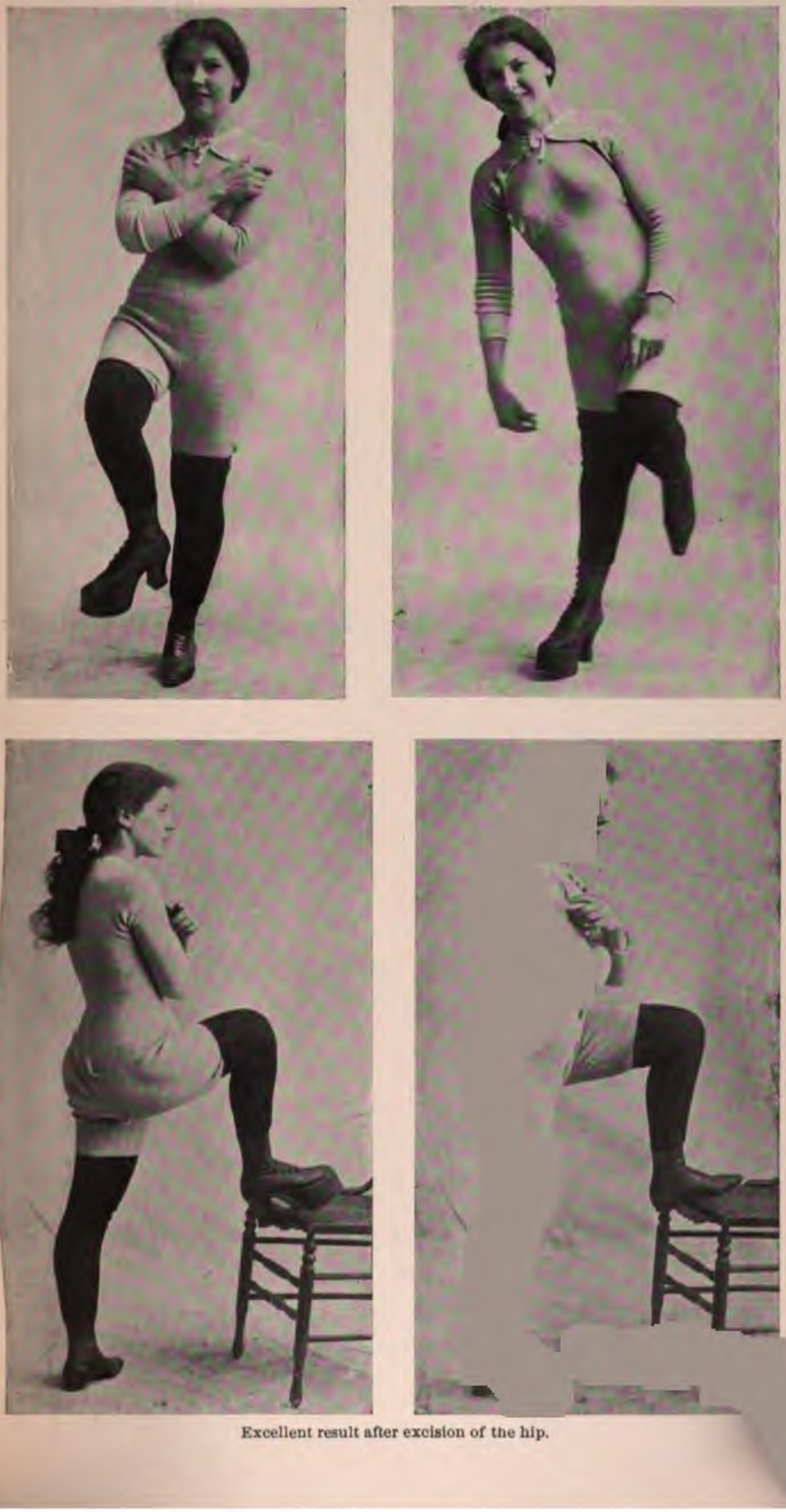
[1] Bradford and Lovett, p. 356.

Fig 13.



Excellent result after excision of the hip.

TUBERCULOUS OSTEO-ARTHRITIS AT THE KNEE.

The knee is the most common location, though by but a small amount, for tuberculous osteo-arthritis, supplying 29.5 per cent. of the cases of the first decade and 22.8 per cent. of those of the second.[1]

The primary location of the infection in tuberculous osteo-arthritis at the knee is, in children under ten, in the bone in about sixty per cent. of the cases; after ten years and until twenty the cases are about evenly osseous and synovial.[2] The locations selected are the internal femoral condyle most often, next the head of the tibia, and lastly the patella. In the tibia and femur the lesion, primarily epiphyseal, but rarely attacks the diaphysis.[3]

Symptoms.—The earliest symptom is the limp, the patient usually stepping with the knee slightly flexed and held rather rigidly, and the weight being passed very quickly to the sound limb, which has advanced to receive it. Later progression becomes practically a series of hops on the sound limb, the toe of the affected limb being barely touched to the floor. The whole endeavor is to avoid bone-work, and in so far the picture is different from synovial cases, in which, so long as the bone is intact, pressure is willingly borne, though motion may be limited. In more advanced stages of synovial tuberculosis, when the epiphyses are affected, the condition is in effect an osteo-arthritis. As the case develops the flexion becomes permanent and increases, and with it is a subluxation backward of the tibial head and a rotation outward of the leg on the tibial axis.

The position of rest at the knee is one of slight recoil from full extension, this permitting relaxation of the stretched flexors and a lessening of the pressure between the bones. Increased flexion takes the leg more and more from the line of bearing weight, and, as the position persists, the direct pull of the flexors on the tibial head and the relaxation of ligaments incidental to the disease result in the partial dislocation of the joint. Rotation outward of the leg, in that it relaxes both the crucial ligaments, also tends to lessen pressure and work. Disease of the patella alone does not entail any change of position or postural deformity. Coincident with the postural symptoms are limitation of motion; swelling due to effusion in the synovial sac; condensation and hypertrophy of the subcutaneous celluloadipose tissues; pain, diurnal and "night cries;" and atrophy of the limb.

Treatment.—Treatment must take cognizance of the pathological condition. Early in the course, if the lesion is so situated as to be appreciable, or even if one can only have an approximate idea of the exact location, it is good surgery to trephine the epiphysis and with gouges and sharp spoons to remove all infected bone.[4] If this is done before caseation has occurred,

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[1] Cheyne, Tuberculous Disease of Bones and Joints, p. 115.

[2] Willemer, quoted by Senn, Tuberculosis of Bones and Joints, p. 452.

[3] König, quoted by Senn, Tuberculosis of Bones and Joints, p. 453.

[4] Cheyne, Lancet, 1890, vol. ii. p. 1094.

the wound may be closed without drainage, the bone-cavity filling with blood-clot. If caseation has occurred, the suture must arrange for temporary drainage with a strand of gauze. The limb must be kept in a splint and out of use long enough to permit definite healing and replacement of the blood-clot, and function must be very gradually resumed. If the lesion is too extensive to permit early radical methods and yet not serious enough to demand the sacrifice of the joint, the protective plan must be adopted. This contemplates the complete suspension of function for the infected tissues—mechanical and physiologic rest—by fixation of the joint, with possibly traction and counter-traction and non-use of the limb. Fixation is attained by a splint which must grasp the limb with absolute accuracy from the toes to the gluteal fold. This is preferably to be made of plaster of Paris, though, if it is desirable, leather or paper splints may be made, using the plaster one as a model. If the plaster of Paris splint is to be the permanent one, it is well before applying the bandages to stick to the whole length of the leg a strip of adhesive plaster which shall be longer than the splint. When about half of the bandages have been put on, the ends of the plaster above and below are turned back and incorporated in the splint. This gives the splint a more definite location or "anchorage" and contributes much to the immobility, but does not exert any traction. If traction is necessary, it may be secured by applying adhesive plasters to the thigh from above the knee upward, and to the leg from below the knee downward, the extension of the plasters beyond the proposed ends of the splint being made with ordinary webbing. A plaster of Paris splint is now applied, and incorporated in it, projecting at either end, are metal pieces carrying little rollers over which the webbing may be folded back and fastened by a buckle on the side of the splint. To make traction and counter-traction on the segments of the limb it is only necessary to pull tight and buckle the webbings.[1] In buckling webbing into ordinary buckles there is a certain loss of tension, and this may be compensated by using heavy elastic instead of webbing at one end of the splint. This apparatus is simple and effective and everywhere attainable, but it must be acknowledged that the effect is probably a more perfect fixation, with but little lessening of pressure between the bones. If there is subluxation of the tibia, this deformity must be corrected at once, and this, in the plan of protection, is best done by the Billroth brackets. The lower segments of these are simple, the arms of the upper segments curve forward and the curved portion is slotted, the screw joining the segments moving in this slot. By this means there is possible a shifting forward of the centre of motion as the tibia advances on the femur. In application, the skin over the femoral condyles in front and the head of the tibia behind is protected with felting. The rest of the leg is wrapped in cotton wadding, and then the plaster of

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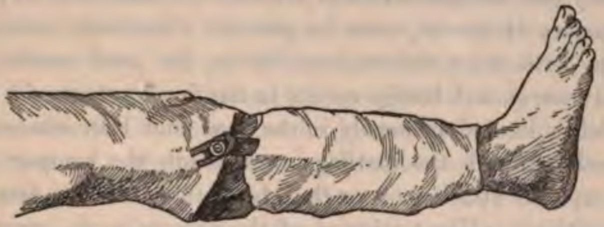
[1] Cf. Taylor, Ready Method for Extension at the Knee, Transactions of the American Orthopædic Association, 1890, p. 40.

Paris splint, with the brackets properly located and incorporated, and with separate "anchorage" plasters for thigh and leg, is put on, except that a little gap is left in front just over the line of articulation. After the plaster is set and dry the segments are separated by a transverse cut behind on a level with the gap in front. Extension of the leg on the thigh is now gradually made by putting at intervals progressively larger plugs of cork into the posterior cut, and at the same time the subluxation is little by little reduced by advancing the centre of motion along the curved slot. (Fig. 14.) The changes must never be so extensive as to be traumatic, and if there be much rigidity about the joint the process may take months, but the method is a very satisfactory one.

FIG. 14.



Leg in Billroth bracket. Extension completed.

The Thomas splint is another very efficient method of securing fixation and traction at the knee. The splint is somewhat modified from the original form of the deviser, and at present is used in two forms, "the bed-splint" and "the caliper." "The bed-splint consists of a ring of round iron to which is welded a long loop of the same material. . . . The ring, in shape, is an irregular ovoid flattened in front and drawn out at the posterior and inner outline of the thigh, and the inner rod of the loop is joined more anteriorly than the outer rod. The ring slopes from without inward, and from before backward in such a way that the point upon which rests the tuberosity of the ischium is the lowest part of the ring."[1] The loop extends well below the foot. The ring, properly leathered, is slipped over the leg and fits about the uppermost part of the thigh, pressing against the tuberosity of the ischium. The leg is held in its place in the loop by bandages or girdles of leather. By plasters adherent to the leg traction can be made from the end of the loop, the counter-traction being at the ischial tuberosity. After the subsidence of the acute symptoms the patient is released from confinement in bed and the bed-splint is changed to a "caliper" by cutting the end of the loop and turning in the two free ends so that they may be fastened into the heel of a shoe. The side rods must be cut of such a length that when the patient stands the sole of the foot is free of the sole of the shoe and all weight is on the splint. Another use of the Thomas splint is as an ischiatic crutch while the knee is held immobile in a plaster of Paris or leather splint. The patient wears a patten on the sound side to match the length of the splint.

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[1] Ridlon and Jones, Chronic Joint Disease (preliminary papers), p. 139.

The protective plan of treatment is to be followed until the process of repair, expected to be inaugurated and to continue under its use, is complete, and the evidences of this fact are the cessation of all local symptoms and an increase in the size of the limb and improvement of the general health. In cases that recover with motion, the increase of range of motion which has been noticed during recovery, as shown by the goniometer, ceases, the possible limit having been reached; and in cases that recover without motion, the ankylosis becomes firm and definite. In cases that do not improve under protective treatment, cases in patients with feeble resisting powers and perhaps with other tuberculous lesions, the local condition becomes progressively worse, and finally results in the development of a tuberculous abscess which is located primarily in the bone and later reaches the joint-cavity directly through the cartilage, or through the compact bony layer inside the capsular attachment, or the abscess may develop first in the infected synovial sac. The testimony of the abscess is the usual one, and, "the knee-joint being a much more accessible joint than the hip, it is not now considered advisable to continue expectant treatment for so long a time, because much can be done to shorten the disease and get a good functional result by comparatively early operation."[1] In cases where it has perforated the compact layer and affected the synovial sac locally, without immediate dissemination, causing a limited thickening, it is advisable to remove all the diseased tissues, synovial and bony, doing the operation with such technique as shall protect the rest of the sac from infection and shall insure asepsis. Then healing should be primary, and the final result may be a practically perfect joint.[2]

In cases where the abscess has not been met as it emerged from the bone, and there has developed a diffuse synovitis, the joint as a joint is already lost, its particular tissues having been in great measure destroyed, and the operation of arthrectomy should be done, especial pains being taken to remove all affected bone,[3] and care being had to avoid unnecessary mutilation of the epiphyseal cartilage, remembering that disease originally epiphyseal rarely attacks the diaphysis, and that all future growth depends on epiphyseal cartilage integrity.

This operation, though appropriate for all properly selected cases, is especially to be chosen for such work in children under twelve years of age, because of its conservation of non-infected tissues, and especially its preservation of the epiphyseal cartilages.[4] In older children a formal excision may, if necessary, be done, though here, too, if the conditions of the joint permit it, an arthrectomy is a proper procedure.

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[1] Cheyne, Tuberculous Diseases of Bones and Joints, p. 263.

[2] Ibid., p. 285.

[3] Ibid., p. 284.

[4] Willard, Journal of the American Medical Association, 1895, vol. xxv. p. 184.

The operation depends for its success, in the first place, on the thoroughness with which the tuberculous tissues are removed, plenty of time and effort being given to searching out and excising every particle of diseased ligament, membrane, and bone, while uninfected tissues are protected from the disaster of infection; and, in the second place, on the efficiency of the aseptic technique, suppuration after such a procedure being an especially unhappy event, as it jeopardizes not only the limb but the life of the patient. In cases where the knee is already septic, the abscess having opened or been opened, and infection with pyogens having taken place and a sinus remaining, the difficulties in securing a satisfactory healing, or any healing at all, are much increased. In selected cases it is still a possibility to do a successful arthrectomy, the wall of the sinus being removed before the joint is opened, the track being thoroughly sponged out with pure carbolic acid, and finally, after the operation on the joint-tissues is done, the whole surface being sponged with the same. In the suture of such a wound, drainage must be arranged for by tubes[1] or gauze.

If, on opening and inspecting the joint, the condition is found so bad as to preclude the possibility of healing and the securing of a useful limb, amputation should be done as conservative of life.

The condition that will call for this operation is not the presence of sinuses or even tuberculous abscesses in these soft parts, for these are amenable to surgical treatment, and as the operation of arthrectomy leads usually to ankylosis, the preservation of the integrity of muscles and ligaments is of minor importance, but it is the extension of the tuberculosis to the diaphysis, causing an osteomyelitis of part of the shaft, the whole of the tuberculous region being probably the site of a secondary septic infection. In later stages of the condition the state of the femur and that of the tibia is usually similar, but the extensive implication of either, which precludes the possibility of conserving enough tissue to secure a competent limb, calls for amputation.

In old cases of tuberculosis at the knee that have recovered with deformity, the knee being flexed and subluxated, there are two principal plans of treatment. An excision may be done, the sections through the bone being so arranged that a wedge is removed and extension is permitted; or the fibrous ankylosis may be broken up by an apparatus devised by Goldthwait. This consists of two bars joined above by bands and approaching each other and uniting below. Together they constitute a lever which gets a fulcrum by an anterior band on the femoral condyles in front, and applies force by a posterior band to the head of the tibia behind, and again, by another posterior band lower down on the leg. The end below the foot is the power end of the lever. The knee, having had such adhesions as could be broken by manual force, is put in the apparatus and the deformity is corrected by intermitting pulls. The leg is then put in a plaster of Paris splint to retain the reposition. Although a *brisement forcé*, in a tuberculous joint the results of the manipulation have been surprisingly good.

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[1] Cheyne, Tuberculous Diseases of Bones and Joints, p. 281.

TUBERCULOUS OSTEO-ARTHRITIS AT THE ANKLE AND IN THE TARSUS.

The disease at the ankle comprises 5.4 per cent. of all cases in the first decade and 5.9 per cent. in the second decade. That of the tarsus supplies 4.6 per cent. in the first and 5.9 per cent. in the second decade.[1] In young children the primary location in the tarsus is more often in the synovial membrane, and it is very difficult in the bone cases to decide which one of the tarsal bones was the first affected. In the ankle the primary location is more frequently in the bone. Of all the bones the ones most frequently affected are those that do the most work in transmitting weight,—the tibia, the os calcis, the head of the astragalus, the tarsal bones, and the proximal end of the first metatarsal bone.[2]

Symptoms.—In ankle disease the order of frequency of bone-locations is first the astragalus and then the inner malleolus. In tarsal disease the order of frequency is the os calcis, base of the first metatarsal, cuboid, astragalus, scaphoid, and cuneiforms. The lesion may be in the form of a granulating focus, or the necrotic form, and the unaffected bones often are osteoporotic.[3] In ankle cases with primary bone-location, limited motion, and later rigidity of the joint with the foot in the position of rest, —that is, extended,[4]—associated with thickening of the soft tissues over the lesion, pain and tenderness locally and atrophy of the calf muscles are the usual symptoms. The disability causes a peculiar use of the foot as the disease commences, the whole leg being rotated out, the line of progression being across the foot, not longitudinal to it, thus saving work for the bones and joints. When tuberculous abscess forms, it and its resultant sinuses are on the antero-lateral aspects of the joint. Recovery leaves the foot ankylosed in the extended position, a definite deformity. In children a single malleolar location may result in interference with growth at the epiphyseal line, and a lateral deformity would follow unequal development.

In the earlier stages of malleolar location, the lesion being recognized, it is proper to cut down on and trephine the bone and remove the infected tissue, avoiding interference with the epiphyseal cartilage and keeping out of the joint. Failing this, protective treatment should be tried, the foot and leg—the former in the position of work, at right angles to the latter—being put into a plaster of Paris splint. If the case does not do well, or the disease extends, an arthrectomy should be done, the joint being reached by lateral incisions on either side in front of the malleoli, and access to the cavity being made easier by section of the lateral ligaments. The removal

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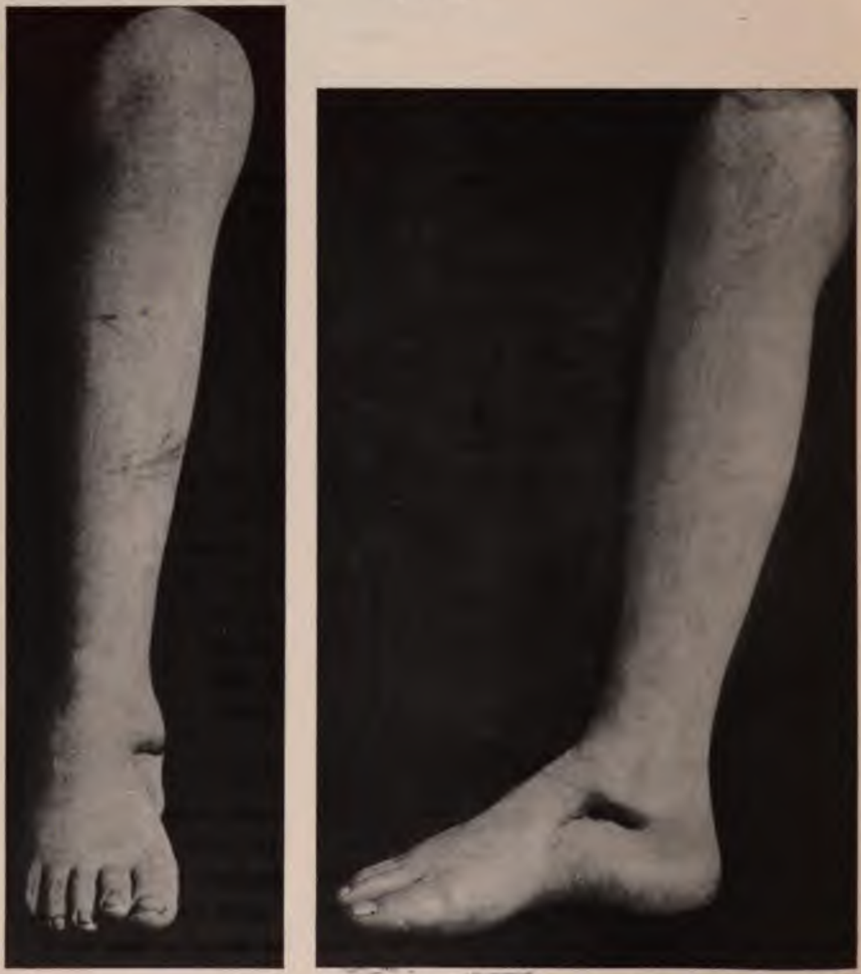
[1] Cheyne, loc. cit., p. 115.

[2] Senn, Tuberculosis of Bones and Joints, p. 473.

[3] Cheyne, loc. cit., p. 287.

[4] Cf. Ellis, The Human Foot, William Wood & Co., p. 11, Plate 8.

FIG. 15.



Front and side views of foot after removal of internal cuneiform, scaphoid, and head of astragalus.

of all infected tissues often contemplates the excision of the astragalus, it being diseased. The functional result in these cases in children may be excellent.[1]

If the lesion is too extensive to permit a satisfactory result after arthrectomy, an amputation, either a Syme's operation or one through the continuity of the tibia, above the disease, may be done; and in doing the latter it must be remembered that the chief growth of the tibia takes place at its upper epiphysis, and consequently the flaps must be cut very long to avoid the mischance later of a conical stump.

In the tarsus the early recognition of the primary location before the infection has spread through the whole part would call for the excision of the diseased tissues, and in doing these operations it is well to avoid, if possible, the use of sharp spoons, for they are not satisfactory in removing the infected soft tissues, while with scalpel and curved scissors it is easy to excise all that is necessary. The skin incisions should be planned to avoid cutting important tendons. The os calcis, if the seat of a primary focus, may be subjected to an early radical operation, or after the formation of tuberculous abscess this may be opened and its track into the bone followed and the diseased bony tissues scraped out. The dressing should be a gauze packing, the wound finally healing from the bottom. If the lesion is too extensive, the bone may be excised by a semicircular incision around the border of the sole, the latter being turned forward and easy access to the bone being had. The cuboid may be treated similarly. At the astragalo-scaphoid articulation the disease very likely originated in the head of the astragalus; this must be removed and all the synovial membrane and perhaps part of the scaphoid. (Fig. 15.) In more diffuse disease in the tarsus the foot may be split longitudinally between the first and second toes,[2] the method giving ready access to the infected bones.

The choice of lines for amputation is that of Syme.

TUBERCULOUS OSTEO-ARTHRITIS AT THE SHOULDER.

This is the least common of the tuberculous joint-affections, giving usually no cases in the first decade and but 1.6 per cent. of those of the second.[3] The location of the lesion is usually in the head or greater tuberosity of the humerus, less often in the scapula. The usual pathological changes of tuberculous osteo-arthritis are seen, and there is also seen the special form, almost peculiar to this joint, of *caries sicca*.

Symptoms.—In the usual form the initial symptom is pain, which may vary much in intensity, and may be around the joint or be felt as a neuralgia down the arm. Probably before the pain has been felt the joint-motion has been restricted by the tonic muscular contraction inhibiting motion, for this, here as elsewhere, is a constant symptom of the lesion. The arm, too, assumes a definite position, being adducted, rotated in, and held to the side, the shoulder being elevated. This position is to be distinguished from that of a synovial lesion simply when the arm is abducted, slightly flexed, and rotated out, the shoulder being drooped. With this last position is apparent lengthening, and with adduction is at first apparent and later probably real shortening.[1] Swelling may be observed about the joint, though often concealed by the coincident atrophy of the deltoid.

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[1] Cheyne, loc. cit., p. 295.

[2] Studsgaard, quoted by Mynter, Transactions of the American Orthopædic Association, 1894, p. 254.

[3] Cheyne, Tuberculous Disease of Bones and Joints, p. 115.

Joint atrophy also occurs, affecting all the tissues of the limb, sometimes even the pectoral muscles.[2] The wasting of the muscles covering the shoulder permits the bony points of the skeleton to be more clearly seen. Tuberculous abscess occurs frequently, and may appear down on the arm, having followed the tendon of the biceps from the joint, or it may appear in the axilla, having perforated the capsule below the border of the subscapularis.[3]

Tuberculosis of the shoulder may be the accompaniment of pulmonary tuberculosis.[4] If it is associated with similar lesions of other joints, it is the last joint to be affected.[5]

In *caries sicca* there are multiple granulating foci,[6] but the process is accompanied by or produces wasting and disappearance of the bone, the whole head being absorbed and the neck being made smaller. This process often does not affect the articular cartilage, which may adapt itself to the new shape of the head,[7] but the synovial membrane is implicated, and ankylosis is the usual result. Tuberculous abscess is not a common effect.

This form of bone-tuberculosis may begin in ordinary healthy individuals, sometimes apparently spontaneously, sometimes after a trauma. The first indications are pain and limited motion, but, instead of swelling, there is a progressive atrophy of all the structures around the shoulder. Atrophy of the humeral head causes the acromion to appear unduly prominent. Any forced motion is painful and causes crepitation.

There are no general symptoms; the condition persists a year or two and recedes, leaving the joint ankylosed. The rare abscesses are small and extra-articular.[8]

In the ordinary form the treatment may have to consist of traction to relieve pain, and this may be made while the patient is in bed, or an apparatus consisting of an axillary crutch and a bar extending down the arm may be used.[9] Immobilization must take into account the probable ultimate ankylosis, which in this joint, because of the mobility of the scapula, is not so serious a disability, and must retain the limb in the position of greatest future usefulness. A long wedge-shaped pad should, therefore, be put between the arm and the trunk to insure some abduction, and the arm should then be fixed, preferably by a plaster of Paris bandage enveloping arm and trunk, in a position of a little rotation out, so that the hand may be put out straight forward. This point is necessary because of the limited range of motion of the clavicle.[1]

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[1] Cheyne, loc. cit., p. 305.

[2] Townsend, Transactions of the American Orthopædic Association, 1894, p. 141.

[3] Senn, Tuberculosis of Bones and Joints, p. 397.

[4] Cheyne, Tuberculous Disease of Bones and Joints, p. 304.

[5] Townsend, Transactions of the American Orthopædic Association, 1894, p. 143.

[6] Senn, loc. cit., p. 129.

[7] Cheyne, loc. cit., p. 66.

[8] Ibid., loc. cit., p. 65.

[9] Shaffer, Transactions of the American Orthopædic Association, 1894, p. 156.

With the development of abscess will arise the question of operation. As the growth of the humerus takes place chiefly at its upper end, the epiphyseal cartilage must be respected, consequently, if operation is decided upon, an atypical erasion is preferable to any excision. Especial care must be taken not to injure the circumflex nerve or its branches. In cases where pulmonary tuberculosis exists, operation, except as a means of relieving pain, would probably seem to be useless. The possible danger of a consequent meningeal tuberculosis should not deter one from an otherwise justifiable operation, any more than possible sepsis should deter one from any ordinary operation where antiseptic technique could be practised.

TUBERCULOUS OSTEO-ARTHRITIS AT THE ELBOW.

The elbow-joint is affected in about 6.3 per cent. of the cases of bone- and joint-tuberculosis,[2] the joint supplying 6.7 per cent. of the cases of the first decade and 9.2 per cent. of those of the second decade.[3] The lesion is much more frequently primarily osseous, and the location of predilection is the olecranon, after that the humerus, and then the humerus and ulna together.

Symptoms.—The first evidence of bone-infection is rigidity, the tonic muscular contraction inhibiting motion, and gradually the joint comes to be held rigid, the forearm flexed about forty to fifty-five degrees from the position of full extension, and pronated. Pain is here, as elsewhere, a variable symptom, tenderness being elicited in handling or joint motion. The swelling is usually first seen on the outer side of the joint, over the radio-humeral articulation, and here, too, the tuberculous abscess usually forms, but it may wander some distance before perforating the skin.[4] The swelling is made to appear greater by the atrophy of the arm and forearm.

Left to itself, the disease may recede and leave the joint ankylosed, or it may go on to infection of the surrounding parts, with great swelling and many abscesses and sinuses.[5] Protective treatment, the first to be instituted in recent cases, rarely calls for traction, and it suffices to put the joint up in a suitable apparatus, preferably a plaster of Paris splint, the forearm flexed to a right angle with the arm, midway between pronation and supination. As the disease is most often in the olecranon, a superficial portion of bone, it not infrequently is possible to locate and remove the initial lesion and so save the rest of the articulation. The same may be done in primary locations in the condyles of the humerus. If this has not been done, or, having been done, has failed to act as a prophylactic against further infection, an erasion is indicated, and had best be done early, the results of the operation being good, especially in children.[1]

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[1] Cheyne, loc cit., p. 308.

[2] Ibid., Tuberculous Disease of Bones and Joints, p. 107.

[3] Ibid., loc. cit., p. 115.

[4] Senn, Tuberculosis of Bones and Joints, p. 408.

[5] Bradford and Lovett, p. 435.

The best incision is on the posterior surface of the arm, over the olecranon, and more room can be gained by a transverse cut from the middle of this out over the humero-radial joint. In this operation great care must be taken to avoid injury of the ulnar and posterior interosseous nerves. The conservation of sound periosteal and bony tissue is important, in view of future usefulness. After-treatment consists in passive motion to prevent ankylosis, which should be begun after healing of the wound, or, if ankylosis seems inevitable, the joint must be put with the forearm flexed to a right angle.

TUBERCULOUS OSTEO-ARTHRITIS AT THE WRIST.

As the disease at the wrist rarely begins in the bones, the title is hardly etiologically correct, but the result of the tuberculous synovitis is quite soon and constantly a panarthritis, with possibly accompanying teno-synovitis, and this condition follows equally the rather rare primary osseous locations.[2]

The disease at the wrist is rare in children, tuberculous lesions below the elbow being more apt to be in the metacarpal bones. The joint is credited with but 6 per cent. of the cases of the first decade and 8.4 per cent. of those of the second decade.[3]

Symptoms.—The symptoms are those usually seen in tuberculous joint-lesions. Swelling of the joint begins early, more marked on the dorsum, and at the same time there are weakness and tenderness. The forearm is atrophied. Slight flexion usually occurs, and the digits soon assume a straight position, the thumb being parallel with the others and close to the index finger.[4] Abscess is not common. There is practically no opportunity for an early radical operation, and the treatment is limited to immobilization. The best apparatus is a plaster of Paris splint extending from the knuckles to the elbow, and the results with this are usually good, the joint recovering in eight or nine months with satisfactory function.[5] Failing to secure this, the joint may be excised, and the operation will usually have to include the wrist-joint and the entire carpus, the disease spreading throughout the extensions of the synovial sac.

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[1] Senn, Tuberculosis of Bones and Joints, p. 417.

[2] Ashby and Wright, The Diseases of Children, p. 617.

[3] Cheyne, loc. cit., p. 115.

[4] Moore, Transactions of the American Orthopædic Association, 1896, p. 101.

[5] Ibid., loc cit., p. 102.

The operation is best done by a single median dorsal incision between the tendons of the extensor indicis and the extensor secundi internodii pollicis. The most difficult point to secure in an otherwise excellent result is mobility at the metacarpo-phalangeal articulation.[1]

Implication of the sheaths of the tendons complicates these cases seriously. While the immediate effect of the treatment, protective or operative, may be good, these subjects rarely live to maturity.[2]

TUBERCULOSIS OF THE VERTEBRÆ.

This is the third most common form of bone-tuberculosis, 12 per cent. of the cases of the first decade and 15.2 per cent. of those of the second being spinal cases. The primary location may be in the periosteum on the anterior surface of the bodies, or in the interior of the bodies, and the latter may be in the form of a granulating focus, or of the necrotic type with sequestrum formation. Locations in the articular, transverse, or spinous processes are rare, but occasionally seen. The rule is for more than one bone to be involved, usually three, four, or five contiguous bones being implicated. Simultaneous affection of groups of bones in different regions of the spine is sometimes observed. The usual spinal location is the middle and lower dorsal, then the upper lumbar,[3] and then the upper dorsal or cervico-dorsal. The disease usually begins before the age of ten years, and may begin in a baby of but a few months.[4] The immediate effect on the bone is the production of structural weakness and lessened power of resistance to pressure. Under the pressure of superincumbent weight and the pressure produced by tonic muscular contraction, inhibiting motion, the softened bone gradually changes shape from a cylinder to a wedge, the base of the wedge looking backward. With the bone-lesion is a similar affection of the intervertebral cartilages, and often their destruction,[5] thus permitting approach of the adjacent bodies towards each other. The result is a kyphos posteriorly, its shape and size governed by the number of bones and cartilages implicated and the extent of their destruction. The arches and processes, of harder bone, usually escape disease. In order that the erect posture may be preserved, there are developed in healthy spinal locations compensatory lordoses. There is sometimes but one of these, above or below the kyphos in the region of the spine, where there is the greater normal mobility, but usually there are two, the major being in the region of the greater mobility. The kyphos, the compensatory lordoses, and in the dorsal region the accompanying changes in the shape of the thorax, all unite in producing a complex deformity. In the cervical region this is characterized by the flexion of the

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[1] Ashby and Wright, loc. cit., p. 618.

[2] Moore, loc. cit., p. 101.

[3] Cheyne, Tuberculous Disease of Bones and Joints, p. 331.

[4] Ibid., p. 330.

[5] Ibid., p. 332.

spine at the disease location and the compensatory extension of the head on the spine, the whole being rigidly held; or there is added to this a torsion of the spine resulting in a close simulation of the torticollis attitude. In the lumbar and dorso-lumbar regions the compensation usually takes place on either side, and as, counting the tenth, eleventh, and twelfth dorsal vertebræ, the amount of natural mobility is very much the same on either side, the lordoses above and below are about even. In these regions there may be distortion of the spine at the disease location and a general postural deformity simulating an atypical scoliosis; observation shows that there is but little if any torsion.[1] In dorso-lumbar and lumbar locations the vertical dimension of the abdomen is always lessened. In the dorsal regions the accompanying deformities of the thorax are of particular moment. Flexion at the disease location would naturally tend to carry forward and downward the ribs attached to the upper segment and forward and upward those attached to the lower segment, but the downward and upward tendencies are restrained by the sternum, and the result is a forward projection of the sternum,[2] more above than below in high dorsal and more below than above in lower dorsal locations. This increases very much the antero-posterior thoracic diameter, but in the advance of the sternum the ribs are made straighter and the transverse diameter is lessened,[3] and by the loss of bone-tissue plus the effect of the kyphos the vertical dimension is made smaller. All of these changes interfere with the respiratory movements, lessening the amount of tidal air, and at the same time there is often in each respiratory act a catch due to a momentary holding of the breath for the sake of the support the inflated thorax can give the spine.

Symptoms.—The first symptom is probably what may be called the spinal limp, but this is usually not noticed, as the step is the same on the two legs, and it is really only the careful carriage of the trunk to protect the affected bone as much as possible from the pressure incidental to jars and sudden movements. In young children the first complaint is often of a pain in the chest or stomach, more particularly when they are handled, the pain being always referred to a place on a lower level than the disease, except in cases of high cervical disease. In middle cervical and cervico-dorsal locations pain may be complained of in the arms. Often, however, the pain is entirely absent. Then comes the well-known squat instead of a stoop in picking up things from the floor, an instinctive avoidance of spinal motion and work; and later the carriage of the trunk erect is too much effort, and the thighs are grasped by the hands, or extraneous support is sought. By this time there is probably a beginning kyphos, and examination finds this and also a well-marked spinal rigidity. This rigidity has existed from the earliest stages of the disease, and could have been found more or less easily by examination. It is here, as in other locations, a tonic contraction of the muscles to prevent motion between the individual vertebræ, and to hold the spine so that it shall act as a whole and avoid localized efforts or strains. It lasts until the very end of the pathologic progress, and its presence is always evidence of active disease. It is the most important symptom, from a diagnostic stand-point, as it precedes the development of the kyphos and makes an early diagnosis a possibility, and it is a condition that should always be looked for in cases where pain in the chest or stomach is complained of. Its demonstration is simple, and is done by laying the child prone and lifting the lower limbs and pelvis by the feet from the table. If the normal flexibility is present, the lumbar spine extends and the chest is not lifted. If there is rigidity, the spine lifts as a whole and without the development of curves. If in this examination the evidence is negative, the child may be turned over and the knees doubled up to the chin, a motion which is quite an impossibility if there is any vertebral tuberculosis. Both of these manipulations must be made with judgment and gentleness, for a slight amount of rough handling may be a trauma to a possibly tuberculous region.

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[1] Lovett, Transactions of the American Orthopædic Association, p. 182.

[2] Schapps, Transactions of the First Pan-American Medical Congress, p. 581.

[3] Ibid.

The tuberculous abscess of vertebral disease occurs in about one-fifth of all cases, being less often seen in cervical disease, more frequently in dorsal, and most frequently in lumbar.[1] Cervical abscess may open into the pharynx or œsophagus, or may descend into the posterior mediastinum and point in an intercostal space; or, escaping from the prevertebral fascia, it may—but very rarely—open into the trachea or bronchi, or point under the skin by the side of the sterno-mastoid muscles. Dorsal abscess, high up, points in the intercostal spaces; lower down, it is apt to pass beneath the ligamentum arcuatum internum and form a psoas abscess. Abscesses arising in the lumbar region may form in the iliac fossa or lower down in the pelvis, escaping then by the great sacro-sciatic foramen; or they may follow the psoas muscle, pointing at the saphenous opening in the thigh or behind the great trochanter; or they may appear posteriorly as lumbar abscesses. Rarely do they open into any of the hollow viscera.[2] The recognition of these collections of fluid and semi-fluid material is not usually difficult, fluctuation being generally very easily appreciated in them, even before they have appeared as superficial swellings.

The aim of treatment is the classical one of securing rest—cessation from work—for the affected bones. This may be accomplished most perfectly by recumbency, less perfectly by ambulatory methods. The latter plan uses a brace or a "jacket," the former confines the patient to a frame or a stretcher.

In all cases of vertebral tuberculosis, of whatever location, the recumbent plan is to be chosen for the acute or the deforming stages. Patients when recumbent are materially longer than when standing,—that is, there is less intervertebral pressure, and the pressure of the superincumbent weight is quite removed. There is, moreover, a direct posterior lever action from the upward pressure of the supporting surface on the kyphos,[1] and it is possible, in addition, to make direct spinal traction by grasping the head above with a sling attached to the head of the frame and the pelvis or the limbs below by a belt or by ordinary traction plasters attached to the foot of the frame, and by this means rest, both that from ordinary work and physiologic rest, may be quite surely attained. The method does not contemplate house confinement, for on these frames an out-of-door life is possible, and even long journeys may be taken, and the plan should be strictly followed until a gain in the general condition of the child, and especially a gain in body weight, give evidence of a cessation of the pathologic process and the commencement of repair.

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[1] Townsend, Transactions of the American Orthopædic Association, p. 164.

[2] Tubby, Deformities, pp. 17–19.

There are certain points which are of moment in constructing a frame to suit the needs of these cases. The material is iron pipe, three-eighths of an inch in size for the smaller cases and larger for larger children. It must be from ten to fifteen centimetres longer than the child and a little wider than the shoulders. The supporting material is canvas, No. 2 for the smaller cases and thicker for the larger children. This is to be hemmed and fitted with eyelets, and must then be a little smaller than the inside measurements of the frame, so as to allow for some stretching. It is to be laced on the frame as tightly as possible, the corners being fastened first and afterwards the sides and ends separately. A narrow oval hole, the edges hemmed and hammered flat, is provided for urination and defecation. It should be about fifteen by eight centimetres in size, longitudinally placed, and its centre for ordinary cases should be three-fifths of the distance from the top. A flap underneath is to be arranged to close this opening when it is not in use. Padded straps of webbing are arranged to pass around the shoulders and axillæ and across the chest. In cervical and cervicodorsal cases loose pads or pillows are made to lie behind the neck and support the spine. In dorsal disease firmer similar pads are sewed to the canvas so that they will press upon the kyphos, lying on either side of the spinous processes and against the transverse processes. These should be from one and a half to two centimetres thick and fifteen centimetres wide and long enough to reach the whole length of the kyphos. A softer pad may be made to support the lumbar spine, and another may be put under the lower extremities to protect the heels and relax the flexors. The pelvis and thighs are to be fastened to the frame by broad bandages. Underneath the canvas at the level of the kyphos broad straps of webbing pass from side to side, fitted with buckles to regulate tension, and by the regulation of this greater or less upward pressure may be made against the canvas, controlling to a nicety the amount of posterior leverage to be used. Pulleys may be placed at head and foot for spinal traction by weight,[1] or, instead of pulleys, simple uprights between which the patient may be definitely fixed, or traction may be made by elastics. The patient lies directly on the canvas, the anus placed just below the upper end of the oval hole, and he is to be gently rolled on one side once a day for an alcohol sponging of the back and its powdering with a talcum powder. Sufficient warm blankets are to envelop both child and frame. In certain cases it may be advisable to bend the side bars in order that the frame may be slightly convex longitudinally and the patient lie with the entire spine in a position of extension, or the side bars may be bent forward only at the region of the kyphos and in such a manner as to fit the deformity and support it.[2] In certain other cases where the maximal amount of immobilization is necessary it may be best to fit a spinal brace of the usual posterior lever type which the child shall wear when on the frame. This same method may be followed in milder cases where the use of the frame is expected to be comparatively brief and with the intention of accustoming the child to the brace before it gets up to go about.

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[1] Schapps, loc. cit.

After the acute stages have passed, the deformity having ceased to increase and the general health having improved, as evidenced chiefly by a resumption of developmental increase in weight, the child may be taken from the frame and fitted with ambulatory apparatus, a brace or a jacket.

The brace employed should be on the posterior lever principle, and of these the Taylor brace is the type, illustrated and described in the original article.[3] The apparatus has, however, its limitations, for it is only in mid-dorsal cases that its leverage principle can be efficiently applied, approach of the disease location to either end of the spine shortening that arm of the lever and lessening power; and even in mid-dorsal cases its action is limited by the ability of the skin over the transverse processes to bear pressure. That the skin cannot possibly bear the amount of forward pressure necessary to carry out the idea of the brace and transfer the weight of the superincumbent portion of the trunk from the bodies of the vertebræ to their articular processes has been shown,[4] and probably the brace is nothing more than a very perfectly adjusted splint,—that is, a restrainer of motion. There are certain forms of chest deformity which stultify the action of this brace, as do all forms which so thrust forward the sternum as to make its anterior surface look forward and upward, for a brace applied to such a case not only does not extend the spine but actually flexes it. It is still, however, for cases of moderate deformity situated between the sixth and tenth dorsal vertebræ, the preferable appliance.

For cases below the tenth dorsal vertebra the plaster of Paris jacket is the best apparatus. The jacket is a splint applied to the trunk, and, in so

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[1] Schapps, loc. cit.

[2] H. L. B. and H. W. C., The Children's Hospital, Boston, p. 143.

[3] Roberts, vol. iii. p. 1038 of this Cyclopædia.

[4] Lovett, Medical News, February 29, 1896.

far as its contents are constantly changing in shape and size, its action as a splint is far from perfect, as can be seen in any case where a fenestrum has had to be cut over some spinous processes. As a means of elongating the spine by lifting the upper segment and so taking weight off the affected bones it has been shown to be also lacking, the height of children with and without the jacket not being different.[1] But still it is by far the best means at command for ambulatory treatment of cases of dorso-lumbar and lumbar location, and grasping, as it does, the pelvis below and the thorax above, it confers the maximal amount of fixation and support.

In all cases above the fifth or sixth dorsal vertebra ambulatory treatment demands that the apparatus should carry the head, and with it the shoulders and arms. Those forms of head attachments to posterior lever-braces which try to produce extension at the disease location by pressure backward on the head are inefficient, in that the lever, the cervical portion of the spine, is flexible. For efficient traction on the head and some extension in the cervical and cervico-dorsal regions it is necessary that the apparatus have a base of support on the pelvis. This permits the use of a certain amount of lifting force, and in all cases of high dorsal or higher locations, where the pelvis is sufficiently developed to carry a jacket or a pelvic belt, it is a satisfactory apparatus. (Fig. 16.) The head is supported by a chin-collar, the chin part being made of aluminum. To get the necessary perfect fit of this, a plaster of Paris bandage is folded around the lower jaw as far back as the angle, and when it is set it gives a negative model of the chin, from which a plaster of Paris positive may be made, and on that the aluminum is fashioned into shape. The occipital part is made of sheet steel, properly padded and leathered, and there are put on either side two hook-catches to fasten into the prongs of the fork, the hooks being on a lower level than the collar, so as to correspond somewhat in position with a common axis of flexion for the head and cervical spine. The fork is made of a steel rod, strong enough not to bend when in use, but malleable, so as to permit of adjustment by wrenches. The attachment to a jacket is by a bracket, as in the well-known Sayre's jury-mast, but usually it will be more satisfactory to get a model of the hips in plaster of Paris, by the same plan as that used for the jaw, and on this to have made in sole leather a broad, firm pelvic belt, which shall extend upward to the dorso-lumbar region. To this are attached spinal uprights similar to those of the Taylor brace, with the accompanying chest-pads, and the spinal uprights carry the block which supports the fork, the location of the block being always below the kyphos. The fitting of the leather will be made easier if, when the plaster of Paris bandage negative is made, a single turn of a broad rubber bandage is put around the crests of the ilia, the ends crossing in front and being fastened below and at either side and so far in advance as not to compress the abdomen.[2] This brings more into relief the bony points. Later, when

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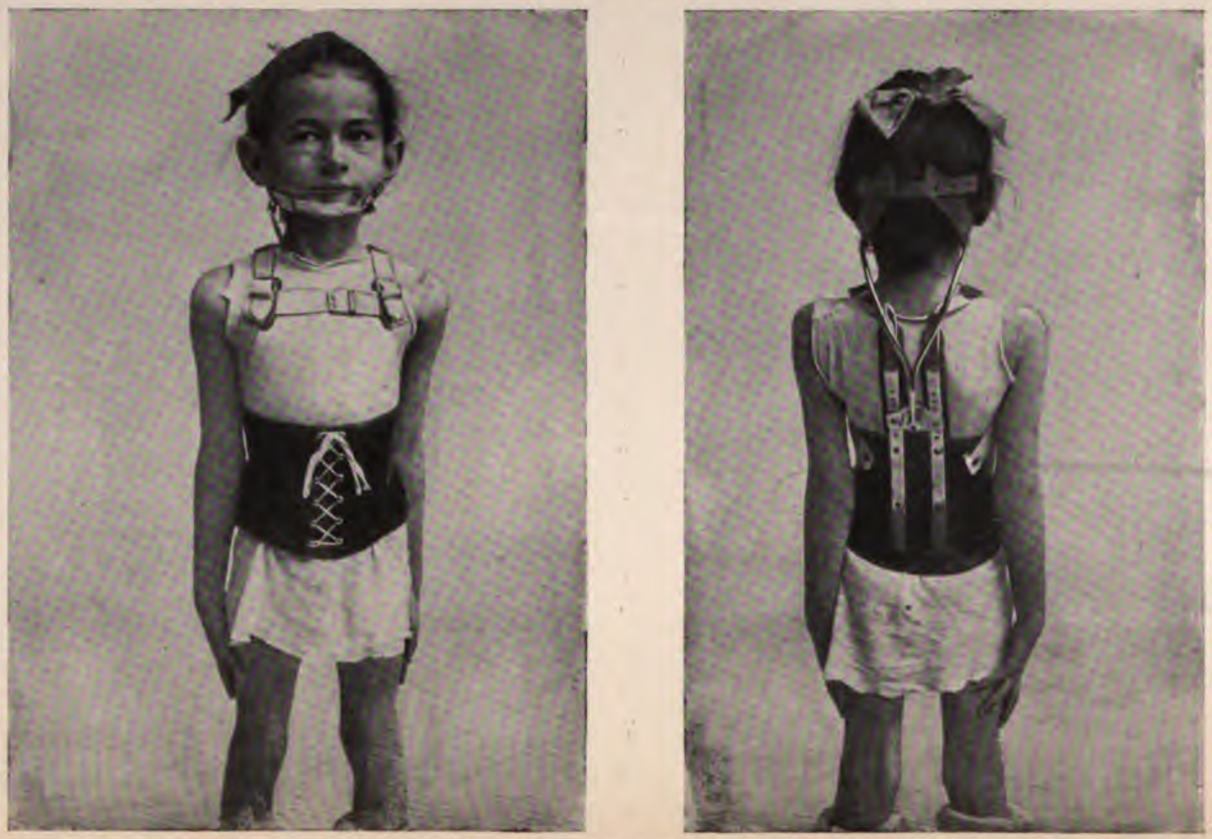
[1] Lovett, loc. cit.

[2] After Lorenz.

Fig. 16.



Front and back of combination brace, leather pelvic belt, spinal uprights and chest pads, and aluminum head-carriage apparatus.

the leather is being shrunk to the model, there must be other strips of leather fastened to the model along the iliac crests, these making recesses in the inside of the belt, into which felting is afterwards put, so that the interior of the belt is a perfectly smooth surface, but the part which is to bear the weight is soft.

The treatment of the abscess of tuberculosis of the vertebræ presents the special difficulty that it is not often possible to extend any operative measure to the bone-focus, and that, therefore, at the best, a symptom only is treated. In these unsatisfactory premises evidence that the abscesses may be disregarded, except so far as they emphasize the need of the greatest possible attention to their bony source, is welcome. It is a fact that under protective treatment for the spine and expectant for the abscess a certain number of the latter are absorbed and eliminated. Others open spontaneously, discharge, and either close definitely or contract to sinuses, which persist for a longer or shorter time[1] or for the life of the patient. Meanwhile the general condition may not be appreciably affected by the presence of the abscess; the absorption or spontaneous evacuation and the healing of the sinus are distinct evidences that retrogressive changes have taken place in the bone-focus. Therefore it is advisable to follow the protective and expectant plan so long as the general health does not suffer, and, after what has been said on the treatment of the spinal lesion, it is evident that, if the abscess appear in a case being treated by recumbency, that plan must be continued, and if the abscess appear during ambulatory treatment, the recumbent plan must be substituted for it.

But if, during this plan, the general condition begins to suffer, if the moderate evening temperature of tuberculosis changes to the higher range of a possibly beginning chronic sepsis, and if there is loss of body weight, it is time to come more actively to the assistance of nature. There is possibly a more delicate and earlier test than these two of higher temperature and loss of weight,—the development of leucocytosis, a sign of the addition of a pyogenic infection to that of tuberculosis, and an indication that the time when absorption or an innocuous spontaneous rupture may be hoped for has passed or is passing. Under these circumstances the abscess must be opened, and if it has been the result of a comparatively acute process the preferable plan is to make a simple aseptic incision[2] into it at the point best suited for drainage, leaving the evacuation of the contents and the ultimate result to natural processes. The dressings of such wounds would be merely the keeping of them covered with aseptic gauzes sufficient to absorb the discharge. The same plan is to be followed in older abscesses when the danger of sepsis cannot be practically eliminated.

If, however, the abscess is of longer standing, being the result of very chronic processes, or if the bone-affection is apparently receding while the abscess is not decreasing, and if it is probable that a practical asepsis can be secured, the abscess is to be opened at a formal operation, the contents evacuated, the wall of tuberculous granulation tissue preferably dissected out,[1] or, failing the possibility of this, scraped out by the finger or by an irrigating spoon, and then any remaining shreds rubbed off by sponges or gauze, so that the surface is left clean and in condition for satisfactory healing.[2] In the after-treatment three courses are offered. Trusting to the thoroughness of the removal of the abscess contents, and disregarding the existence of the bony focus, the incision may be stitched, the cavity having been first filled with an iodoform glycerin mixture if the operator so desires. Under this method first intention healing is usually secured, but the abscess sometimes recurs, necessitating a second operation. Second recurrences, however, do not often happen. Or a sinus may form through the cicatrix, but by drainage and irrigation this may be made to heal.[3] A second plan arranges definitely for drainage, a tube being passed to the deepest part of the cavity and kept in position until all tuberculous discharge has ceased, and through it, during healing, the cavity is at intervals washed out.[4] A third plan arranges for temporary drainage by gauze strands, which are removed after from forty-eight to seventy-two hours, and their track permitted to close if it will do so. In the washing out of these cavities proper solutions of any selected antiseptic may be used, but those of corrosive sublimate, of a strength of from 1 to 3000 to 1 to 5000, and at about the temperature of 38° C., are preferable.

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[1] Shaffer, New York Medical Journal, February 29, 1896.

[2] Cabot, statement of Gage, Boston Medical and Surgical Journal, 1896.

The secondary local effects of vertebral tuberculosis are shown through the nervous system, the spinal cord or the spinal nerves exhibiting symptoms of disturbed or inhibited function due to pressure.

The pressure is due to the occurrence within the rigid limits of the spinal canal of the usual phenomena of tuberculosis of bone, swelling and hypertrophy of the soft tissues over the tuberculous region, the growth of a tumor made up of tuberculous tissue, and the formation of a tuberculous abscess. As the result of any or all of these there is a thickening of the spinal dura, a pachymeningitis.

In cases of cervical location the pressure may be made by the bone.[5] Pressure on the nerves may cause only pain, as the pain felt in the arms in cases of cervical location, or there may be a herpes zoster along the course of the nerve pressed on.[6]

The frequency of the occurrence of pressure paraplegia may be put at from one case in six[7] to one in thirteen.[8] As regards the region, the largest number occur in the mid-dorsal, the next largest number—about a third as many as the largest—in the lower dorsal, and the dorso-lumbar and cervical follow with still fewer. The symptom usually supervenes about a year or a year and a half after the beginning of the disease, and it lasts from a few months to a year in most cases, but may last several years, or even for life. Second or third attacks are not uncommon, the patient finally, perhaps, recovering from them all. The condition may develop in the course of carefully managed cases.

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[1] Cheyne, Tuberculous Disease of Bones and Joints, p. 359.

[2] Treves, Operative Surgery, vol. ii. p. 734.

[3] Ibid., p. 736.

[4] H. L. B. and H. W. C., The Children's Hospital, Boston, p. 159.

[5] Parkin, British Medical Journal, 1894, vol. ii. p. 699.

[6] Ashby and Wright, The Diseases of Children, p. 670.

[7] Myers, Transactions of the American Orthopædic Association, 1890, p. 209.

[8] Little, quoted by Tubby, Deformities, p. 23.

The invasion is usually slow, easy fatigue and muscular weakness preceding any paralysis, and at the same time exaggerated knee jerks and ankle clonus are present. The weakness increases to complete paralysis with exaggeration of all reflexes, then spasmodic contractions occur which may result in contractures with deforming positions of the limbs and extremities, and later paralysis of the sphincters of the bladder and anus, and very likely the development of bed-sores. In the severer cases paralysis of sensation occurs, knee-jerks and other reflexes may disappear, and very rarely there may be gangrene of a portion of an extremity. Improvement shows itself by a gradual recession of all these symptoms, those last produced disappearing first.

The prognosis in general is good, the percentage of recoveries in cases that have been followed and the result definitely ascertained being more than seventy per cent.[1] The extent of the paralysis, showing the amount of the pressure on the cord, is of prognostic value, those cases where the bladder and rectum, and more especially sensation, are paralyzed not having the same expectation of recovery as the milder cases; but even in severer cases recoveries take place.

The treatment primarily is that of the vertebral lesion. The most careful and patient following of the recumbent plan is imperative if the best possible result is to be secured. This is to be persisted in so long as the general condition of health of the patient is good and there are no evidences of any inflammatory or degenerative changes in the spinal cord. If, in such a case, with the proper mechanical treatment, the general condition begins to fail, the paraplegia not lessening; or if the paraplegia persists a very long time, or increases rapidly at any time; or if a pressure myelitis threatens the integrity of the cord, it is evident that the treatment of the original lesion is not sufficing, and special treatment must be directed to the symptom, aiming to relieve the pressure by operation.

The operation of laminectomy—removal of the laminæ and spinous processes—has in the past nine years been frequently done for this purpose, with results that have been good, bad, and indifferent in the hands of all operators, and this is inevitable because of the tuberculosis which is the cause of the paraplegia; but it is always undertaken in conditions that render any chance of benefit a most desirable thing, and it has been followed by such a measure of success as to class it as a justifiable procedure in the proper cases.[1] At the same time the general good outlook for those cases, even after long-existing paraplegia, and the lack of success that has often followed the operation make it incumbent on the surgeon to resort to it only after protective and expectant methods have failed after being thoroughly tried. Of thirty-nine children thus operated on sixteen died,[2] and this may be considered as fairly representing the expectations of mortality.

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[1] Lloyd, Annals of Surgery, 1892, vol. xvi. p. 297; and Bradford and Lovett, p. 51.

The operation is done as follows. The patient lies on the table semi-prone; proper precautions are taken against shock. An incision is made down on the spinous processes and then down their sides to the laminæ. The skin and muscles and all tissues down to the laminæ are dissected or lifted from the bones by scalpels and elevators and pushed to either side. Hemorrhage is controlled by pressure. The spinous processes are removed by bone forceps. The laminæ are divided at their outer ends by a Hey's saw or by laminæ forceps. With a scalpel the fibrous and ligamentous tissues are cut, beginning below, and the plate of laminæ lifted and turned up, the upper attachment being left. This exposes the canal, and any pathological condition found must be properly dealt with, abscesses opened, tuberculous granulomata removed, and sequestra and other detritus taken away. It will but very rarely be necessary to open the dura. The plate of laminæ may be detached or replaced and covered in. Drainage by tube is to be provided and the muscles and skin sutured. If the originally planned operation does not cause return of pulsation to the compressed cord, it is right to remove more laminæ above or below until pulsating cord is reached.

After the operation the recumbent treatment must be continued until all the paraplegic symptoms have disappeared and the spine has become strong enough to support the weight of the patient. When the erect posture is assumed, the spine must be supported by a proper brace, which should be worn a long time. Even in cases where the laminæ have been removed the strength of the spine is not seriously impaired, provided the patient recover from the tuberculosis.

THE FORCIBLE CORRECTION OF THE DEFORMITY OF VERTEBRAL TUBERCULOSIS.

During the past two years there has been a return to the methods of Hippocrates and Ambroise Paré in the treatment of the kyphos, which is the most obvious evidence of tuberculosis of the vertebræ. The practice was reinaugurated by Dr. Calot, of Berck-sur-Mer,[3] who reported thirty-seven cases with no deaths, immediate or remote, no paralyses, and but two abscesses sequent to the operation; furthermore, three abscesses existent at the time of the operation became absorbed. His technique was to have strong traction made on the head and feet by assistants, the patient being prone, and then to himself press on the kyphos with sufficient force to extend it at the point of pathologic flexion; and he not only corrected, but over-corrected, the act being accompanied in the bones implicated by crackling and breaking, which could be felt by the operator and heard by the bystanders. The patient was then put in a plaster of Paris jacket or splint, which firmly grasped the head, trunk, and pelvis and maintained the corrected position. This first apparatus remained in place three or four months, and was then followed by a second, and perhaps a third, each remaining a similar time. Recumbency was enforced for three months, after which the patient got up and walked. By the ninth month Dr. Calot considered that repair and consolidation were complete. In cases in which the deformity was very rigid, from consolidation of the affected bones, Dr. Calot did a cuneiform osteectomy, removing spinous and articular processes and the corresponding transverse processes and portions of the ribs, and then did an osteotomy of the bodies at the apex of the kyphos; although he considers that in old cases this will be rarely indispensable.

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[1] Lane, British Medical Journal, 1891, vol. ii. p. 949; and 1892, vol. ii p. 1423; Parkin, British Medical Journal, 1894, vol. ii. p. 699.

[2] Lloyd, loc. cit.

[3] Annales de Chirurgie et d'Orthopédie, December, 1896.

The plan had been foreshadowed by Dr. B. E. Hadra, of Galveston, Texas, who had straightened a fractured and kyphotic spine, and had wired the spinous processes to each other in 1890, and mentioned the possible doing of the same thing for the deformity of vertebral tuberculosis.

Since Dr. Calot's memoir, other surgeons, American and European, have practised the method, with the result that its applicability and limitations are fairly well made out.

The treatment is plainly addressed to the symptom-deformity, just as are other forcible corrections applied to deformities and malpositions following tuberculosis in different parts of the body. In the spine, too, as in other regions, the deformity is the result of a functional malposition which has become permanent, plus a changed mutual relation of the implicated bones due to pathologic changes in their shape and consequently in their mechanical action. It should, therefore, be expected that the forcible correction would be of benefit in so far as it removed a deformity due originally to a functional malposition which had become permanent, but that it would be valueless in cases where bone disease and destruction had so changed shapes and relations that new mechanical conditions existed. On the pathologic process itself the result is not definitely known; all that can be said is that in other parts of the body trauma aggravates tuberculous osteo-arthritis.[1] It is, however, conceivable that early in a case, before there has been much bone-destruction, and before consolidation has taken place, the gentle straightening of a flexed spine may relieve the intervertebral pressure, secure more perfect rest for the bones, and put them in the best position for recovery, just as a similar plan in the management of hip-joint tuberculosis results in a betterment of conditions. Later in the course of a case where the markedly prominent deformity is the outward sign of bone-destruction the obliteration of the curve opens up spaces between the remnants of the vertebral bodies, and these spaces, following the general rule of bone tuberculosis, are not filled with bone, but with fibrous tissue; or there may be no reparative act at all, and the spaces may remain as false joints bounded by caseous and diseased tissues.[1] That abscesses may follow the operation is obviously true, but they are such constant accompaniments of the disease that it is not possible to credit them to the procedure;[2] at the same time, experience in forcible corrections in other locations leads to the expectation that abscess would be a more common complication in cases submitted to this operation than in others. Paraplegia seems not to be caused by the operation nor to follow it; and of five cases with existing paraplegia, it was recovered from in three cases and lessened in one, and this apparently because of the straightening.[3]

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[1] Ménard, La Presse Méd., No. 57, 1897.

The cases that are amenable to this plan are limited in number, and are those in which the disease is recent, the angle of curvature a changing one, the patient under twenty, the general health fair, and the disease not so active as to give rise to general constitutional disturbance. Lower dorsal and lumbar curves are, *cæteris paribus*, the most suitable for reduction. Patients who have other tuberculous foci, or much wasting, or cough or respiratory trouble, or abscesses, or in whom firm ankylosis has taken place or considerable alteration in the shape of the thorax, or the curve is in the cervical region, or who are over twenty years of age, are entirely unsuited for the operation.[4]

In the technique the plan of Calot has been variously modified. Some operations have been done without an anæsthetic,[5] but usually one is used. Plaster jackets are best applied with the subject in the prone horizontal position, especially in young children with the deformity below the ninth of tenth dorsal vertebra. In all other cases it is best to suspend the patient by the feet or knees.[6] Or the child may be kept prone during the application of the jacket and suspended by the feet for the extension of the apparatus to and about the head. In place of the plaster of Paris apparatus, a brace of iron, like a double Thomas hip-joint splint, but made to extend up to and support the head, may be used.[7]

In spite of the optimism of Dr. Calot, in no case should the treatment be shorter than it has been hitherto under the ordinary plan of immobilization and rest, for the manipulation is directed entirely against the symptom-deformity, and in no way controls the progress of the tuberculosis. Nor is the operation entirely devoid of danger, for a case is reported to have died during it,[1] and others have died afterwards of generalized or meningeal tuberculosis, and thus cast on the operation the suspicion of being the occasion, at any rate, of the extension of the disease.

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[1] Murray, British Medical Journal, December 4, 1897.

[2] Jones, Liverpool Medico-Chirurgical Journal, January, 1898.

[3] Jones, loc. cit.

[4] Tubby, Practitioner, January, 1898.

[5] Jones, loc. cit.

[6] Ridlon, Journal of the American Medical Association, March 26, 1898.

[7] Jones, loc. cit.

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[1] Vulpius, Cent. f. Chir., December 11, 1897.

FRACTURES AND DISLOCATIONS.

By JOHN H. PACKARD, M.D.

THE views presented in my former article on this subject, in vol. iii. of this work, do not seem to me to need emendation, and in the nine years that have passed since that chapter was prepared the additions to the literature of this branch of surgery have been merely records of observed cases. The material of this supplementary paper will therefore consist of a *résumé* of these reports.

A matter of much interest, but as yet in its infancy, is the use of the Röntgen rays, or skiagraphy, in the diagnosis of injuries of the bones and joints. Withington[1] has recently reported a case of fracture of the humerus by muscular action in a boy fifteen years old, with a skiagraph, which shows not only the lesion, but the line of junction of the epiphysis with the shaft. The graphic description of Professor Keen in the present volume is a correct exposition of our knowledge of the subject at this date.

FRACTURES.

FRAGILITAS OSSIUM.

A curious case of *fragility of the bones* has been reported by Plicque,[2] the child sustaining fourteen fractures within the first three and a half years of its life. It is stated that the mother and the grandmother had also had numerous fractures, and an older brother as well as a younger sister was rachitic. This child at the time of birth had a double fracture of each humerus. When two months old the right elbow was broken, and again two months later. Four months afterwards there was fracture of the right humerus, and the same bone gave way six months later, and again after the lapse of fourteen months, the child being then two years and four months of age. Two months later the right leg was broken, and two months after that the right thigh. At thirty-nine months there was fracture of the sternum as well as of the right humerus, and two months after that the latter bone gave way again. It will be noted that the fractures were nearly all on the right side, a fact which seems to favor the idea of some morbid condition of the central nervous system.

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[1] Boston Medical and Surgical Journal, May 13, 1897.

[2] Journal de Conn. Médicales, October 16, 1890.

A child two years old was recently shown by Guthrie to the Medical
ciety of London,[1] who had presented for about four months bluish swelli
in the femora, clavicles, and left humerus, which by skiagraphy were sho
to correspond to fractures. These lesions were supposed to be due to sco
butic taint.

In connection with this case, Morgan mentioned that of a child age
seven, seen by him, who had eighteen fractures. He thought that the fac
that the bones had given way in the shafts, and not at the epiphyseal junctions, was against the idea of scurvy.

INTRA-UTERINE FRACTURES.

On *intra-uterine* fractures there have been published two interesting theses, one by Vilcoq in 1888, the other by Hollerbusch in 1893. The latter author notes that the right leg is the part most frequently affected, and dwells upon the fact that such lesions are often accompanied by malformations or defects of development of the corresponding feet.

Janicke[2] has recorded the case of a girl seen by him when she was a year old, who had been born with the left leg bent at an angle salient forward at about the junction of the middle and lower thirds. At the outer edge of the sole of the right foot there was a furrow, due to the position of the two legs when the child was within the womb. The cause of the fracture of the left leg does not seem to have been determined. Section of the tendo Achillis and excision of a wedge of bone had been performed, but with very little benefit.

FRACTURES DURING BIRTH.

Wyeth[3] records a case in which the femur gave way at the trochanters under traction in a case of breech presentation. A plaster dressing was applied, and a good result obtained.

Milliken[4] has reported an instance in which a child born after a tedious labor was noticed on the second day to have the left arm hanging limp. On careful examination, the diagnosis of separation of both the upper and lower epiphyses of the humerus was made. Anterò-posterior splints of pasteboard and a starch bandage were applied, and by the thirtieth day union was firm, with some excess of callus. Extension of the elbow was somewhat limited.

NON-UNION.

Reference may be made to a paper by Mr. D'Arcy Power[5] on ununited
ractures in children, with an analysis of seventy-two cases of the kind.
f these, the clavicle was affected in six, the humerus in seven, the radius

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[1] British Medical Journal, April 17, 1897.

[2] Breslauer Aerztliche Zeitschrift, March 30, 1889.

[3] New York Medical Journal, July 4, 1891.

[4] Archives of Pediatrics, 1894.

[5] Medico-Chirurgical Transactions, vol. lxxv., 1892.

in one, the femur in twelve, the bones of the leg in forty-five.[1] Forty of the children were males, twenty-nine females, and in three instances the sex was not given. Mr. Power thinks that the increasing frequency of such cases is due to want of rest of the parts, and would ascribe this to inefficiency in methods of treatment of the original lesion. As to the results in the tabulated cases, bony union was obtained in twenty-one, improvement in four, and in forty-five the condition was unchanged for the better.

Sir James Paget[2] gives three cases of non-union in children, all finally resulting in amputations. In one, a girl was born with a bent leg, which was broken by a bone-setter who attempted to straighten it when she was three years old; union failed to occur, and five years afterwards the limb was removed.

A similar case is recorded by Southam,[3] in which a boy aged six years had had osteoclasis performed four years previously for bowing outward of the leg; the limb was wasted, and union failed to occur in spite of two osteoplastic operations and two graftings of bone. Amputation was therefore performed.

FRACTURES OF SPECIAL BONES.

Clavicle.—A somewhat curious case is recorded by Haley.[4] A child three months old fell out of bed just at the onset of an attack of scarlatina. Shortly after recovery from the fever two sinuses appeared over the left clavicle. Nearly two years later Haley saw the child, laid open the sinuses, and removed a piece of detached bone as well as the necrosed outer two-thirds of the clavicle; the wound healed promptly.

A case of separation of the sternal epiphysis of the clavicle by muscular action has been recorded by F. Le Gros Clark.[5]

Erdman[6] reports four instances of fractures of the clavicle in young children.

Scapula.—Wagner[7] has reported a case in which a child was delivered with instruments, and five weeks afterwards the mother noticed a peculiar crackling with the movements of the infant's arms. There had been no fall or other known injury. The diagnosis was made of fracture of both scapulæ, "the fracture extending from the supra-scapular notch through the spine and infra-spinous fossa." Crepitus is said to have been very marked. This must certainly be regarded as an extraordinary case.

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[1] In my former article I spoke of the humerus as more frequently the seat of non-union than any other bone, and this is true of fractures taken at all ages, according to the statistics given by Agnew and others. But in children the proportion is different, and I believe the statement of Mr. Power, cited in the text, to be correct.

[2] Studies of Old Case-Books, London, 1891.

[3] Lancet, June 20, 1891.

[4] Therapeutic Gazette, October 15, 1895.

[5] St. Thomas's Hospital Reports, 1889.

[6] Medical Record, October 26, 1895.

[7] University Medical Magazine, April, 1894.

Humerus.—Separations of the *upper* epiphysis of this bone have been recorded by Erdman[1] (five cases), by H. R. Wharton,[2] by Clark,[3] and by Rollet.[4] It seems probable that this lesion occurs much more frequently than is generally supposed.

A fracture of this bone at the junction of the middle and upper thirds in a male child one day old is recorded by Gerber.[5] No explanation of the injury was apparent, and it was thought to have been inadvertently produced by the nurse in bathing the infant. Recovery took place in about two weeks, with a thick callus.

Erdman reports also five cases of separation of the *lower* epiphysis of the humerus. An instance of this kind in which excision was performed with good results is given by MacDougall.[6] Two cases are reported by Deanesly[7] in which such a lesion was followed by thickening of the bone and consequent paralysis of the musculo-spiral nerve. The patients were boys eight years of age, and in each recovery was brought about by massage and electricity.

Elbow.—As stated in my former article, there has existed some difference of opinion among surgeons as to the best way of dealing with fractures in the neighborhood of this joint. By Allis, Roberts, and others it is claimed that coaptation of the fragments is best insured by placing the forearm in a nearly straight posture with relation to the upper arm and in supination. They assert also that the ultimate usefulness of limbs so treated is satisfactory.

A method which combines these two ideas has been advocated by Lartet[8] and Guedeney.[9] It consists in keeping the elbow in extension until the sixth or seventh (sometimes as late as the tenth) day, after which the joint is flexed, and so retained until the end of the treatment.

By Leblois[10] it is claimed that the best results are secured by massage, the joint being kept betweentimes in flexion. This method does not differ very materially from that of early passive motion, which I have always employed and advocated.

Forearm.—A case is reported by Deanesly[11] in which a girl aged six years had, in consequence of a fracture of both bones of the forearm, paralysis of the median nerve. The nerve was exposed and stretched, and recovery gradually ensued, being complete in six months.

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[1] Loc. cit.

[2] University Medical Magazine, January, 1889.

[3] St. Thomas's Hospital Reports, 1889.

[4] Lyon Médical, 1891.

[5] Pesther Med.-Chir. Presse, April 9, 1893.

[6] Edinburgh Medical Journal, March, 1891.

[7] British Medical Journal, June 17, 1893.

[8] Fractures de l'extrémité inférieure de l'humérus chez les enfants; traitement par l'extension et la supination combinées à la flexion, Paris, 1889.

[9] Du traitement des Fractures du Coude chez l'enfant par l'immobilisation en extension et en flexion alternatives avec supination, Lyon, 1893.

[10] Contribution à l'étude des Fractures chez les enfants et de leur traitement, Paris, 1894.

[11] British Medical Journal, June 17, 1893.

Separation of the *lower* epiphysis of the radius has been noted in two instances: by Clark,[1] in a boy aged fifteen, and by Shattock.[2] In the latter case the line of division was quite clean, and there was detachment also of the styloid process of the ulna; the patient, a boy eight years old, had fallen a distance of twelve feet. In one of Erdman's cases, before referred to, a girl seven years old, who had fallen from a fence, had a separation not only of the lower epiphysis of the humerus, but of the lower ends of the radius and ulna of the same arm.

Power[3] found in an analysis of 988 collected cases of Colles's fracture that 48, or nearly five per cent., were in children between the first and the tenth year of life, 37 of the subjects being males and only 11 females. Between the eleventh and the twentieth year of life there were 214 cases, or over twenty-one per cent. of the whole number; of these 201 were in males and only 13 in females. Very probably a considerable number of both these sets of cases were in reality epiphyseal separations.

Reference may be made here to a good article by Curtis[4] on neglected fractures in children. The cases described by him concern the clavicle, the inner condyle of the humerus (with luxation of the head of the radius), and the bones of the forearm.

Pelvis.—R. Winslow[5] has reported a case of severe compound crush of the pubic bone, with great laceration of the soft parts of the pelvic region, in a boy aged twelve. Recovery was complete.

Femur.—The following case has been reported by Whitman.[6] A boy eight years old fell a distance of eighteen feet. He was unable to stand after the accident, and his right knee was much swollen. A physician who saw him three days afterwards gave his attention wholly to this part, and in four weeks, the swelling having subsided, considered the child to be well. But as the child could not walk, he was taken two weeks later to the Hospital for the Ruptured and Crippled. Here it was found that the limb was shortened an inch and a half, the foot everted, and the trochanter, which was broadened and tender on pressure, was situated above Nélaton's line. Motion, although painful, was but slightly limited, the inward rotation of the foot being considerably resisted. Recovery took place, but the child limped in walking.

This case is reported as one of fracture of the cervix femoris.

In a later article[7] the author states that the child above mentioned had recovered free motion of the joint in all directions. He gives also four other instances in which he claims to have seen the same lesion in children: a boy aged six, who eighteen weeks previously had fallen a distance of fourteen feet; a boy aged five, who nine weeks before had been "knocked down or run over" by a heavy carriage; a boy aged eight, who six months before had fallen a distance of fifteen feet; and a child two and a half years old, who one month previously had fallen from a height of four stories.

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[1] St. Thomas's Hospital Reports, 1889.

[2] Transactions of the Pathological Society of London, 1890.

[3] Medical News, March 9, 1895.

[4] Medical Record, May 20, 1895.

[5] Maryland Medical Journal, March 11, 1893.

[6] Medical Record, New York, February 7, 1891.

[7] Ibid., February 25, 1893.

From the accounts given of all these five cases there can be no reasonable doubt that the neck of the bone had suffered in each. But the lesion was only part of a general smash of the whole of the upper portion of the femur by great direct violence applied through the trochanter. Such an injury, it seems to me, differs materially from the fracture of the femoral neck to which persons advanced in life are so liable.

Dr. Whitman refers to cases recorded by Rousseau, Hoffa, and Hamilton,[1] in regard to all of which the same may be said. He speaks also of having met with several instances in which the neck of the bone gave way during attempts at the reduction of old luxations of the hip in children. In these latter, the head of the bone being fixed, the leverage exerted through the shaft would, of course, act as in the case of old people. But it seems to me that the fact remains that fracture of the cervix femoris does not occur as an ordinary uncomplicated accident in early life.

Separation of the *upper epiphysis* of the femur has been observed in three instances by Bradford.[2] In one, a boy sixteen years old, it was due to muscular action; eversion of the foot was not noted in this case, but was present in the other two, both of which were caused by falls from heights.

McKenzie[3] reports a case in which the upper epiphyses of both femora became separated during an attack of scarlet fever in a boy four years of age; the fragments seemed to have been absorbed.

Fractures of the *shaft* of the femur have been reported: by Cowan,[4] in a child aged six months and twenty-three days; by Kintzing,[5] in a girl aged nine months; and by Adams,[6] in a girl aged four years and eight months. All these cases ended favorably.

Lorenz[7] has recorded an instance in which a boy four and a half years old fractured his left femur in the upper third; union took place with bowing outward, and the deformity was only relieved by a third attempt when he was ten years old, extensive section of the muscles being then performed along with linear osteotomy.

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[1] In this case, which was under the care of Dr. J. R. Wood, a healthy girl sixteen years of age was caught between two carriages. In spite of careful treatment, no union occurred. She died of an acute disease three years afterwards, and an autopsy showed transverse fracture of the bone just at the junction of the head and neck; the head was partially absorbed, and the other fragment was rounded off and covered with fibrous deposit.

[2] Boston Medical and Surgical Journal, March 3, 1892.

[3] University Medical Magazine, November, 1892.

[4] Dominion Medical Monthly, August, 1893.

[5] Maryland Medical Journal, August 23, 1890.

[6] University Medical Magazine, February, 1890.

[7] Wiener Med. Wochenschrift, January 3 and 10, 1891.

A case of compound comminuted fracture of the upper third of the shaft in a boy ten years old, who made a good recovery, is given by Winslow.[1]

Townsend[2] has reported a case in which union failed to occur in the femur of a boy aged nine until he was fitted with a hip splint, a high-heeled shoe being put on the foot of the sound limb; the child was thus enabled to move about.

Further experience has convinced me of the value of vertical extension in fractures of the femur in children, especially in those affecting the upper part of the bone. Here the very small purchase afforded by the upper fragment makes retention in any other way difficult. A silicate or plaster bandage, with an anterior splint of sheet zinc, answers perfectly when the lesion is seated lower down in the shaft.

Separations of the *lower epiphysis* of the femur have been the subject of several articles. My own case, briefly mentioned in vol. iii. of this work, has been published at greater length, with references to all the cases previously recorded.[3]

Five additional instances are given by Harte[4] in connection with an interesting discussion of the nature and treatment of these injuries.

In three of Harte's cases amputation was performed, while in two reduction was effected with good result. Robson,[5] Owings,[6] and Winslow[7] record instances in which the limbs were saved. MacDougall[8] gives two cases and Clutton[9] one in which amputation became necessary. Clutton's case ended fatally from gangrene and pyæmia.

Cases in which this lesion occurred from pathologic causes have been reported by Meisenbach[10] and Iscovesco.[11] The latter surgeon, treating of epiphyseal separations produced during attempts at correction of deformities resulting from white swellings (tuberculous arthritis) of the hip and knee, gives two instances of lesions of this kind thus caused in each of the joints mentioned.

Bones of the Leg.—Winslow[12] reports three instances of severe compound fractures in this region; two of them, in boys aged respectively seven and twelve years, did well; in the third, the patient a boy aged sixteen, both whose legs were crushed under a railroad car, recovered after a double amputation.

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[1] Maryland Medical Journal, March 11, 1893.

[2] New York Medical Journal, April 19, 1890.

[3] Annals of Gynæcology and Pediatrics, November, 1890.

[4] American Journal of the Medical Sciences, June, 1896.

[5] Annals of Surgery, 1889.

[6] Medical Record, January 3, 1891.

[7] Loc. cit.

[8] Edinburgh Medical Journal, March, 1891.

[9] St. Thomas's Hospital Reports, 1894.

[10] Annals of Surgery, 1895.

[11] Cong. franç. de Chir., Procès verb., etc., Paris, 1891.

[12] Loc. cit.

Romniciano[1] has reported the case of a rachitic girl who at eleven months of age had fractures of both bones of the leg. Union failed to occur, and when she was two years old a resection was performed with good result.

In a case recorded by Chapin,[2] a boy six years old sustained a fracture at the upper part of the fibula by muscular contraction in turning suddenly on being sharply spoken to by his mother.

An instance of separation of the *lower* epiphysis of the tibia, with atrophy of that bone and consequent bowing outward of the leg and inversion of the foot by the growth of the fibula, has been reported by Owen.[3] The patient was a girl nine years old.

DISLOCATIONS.

Shoulder.—Congenital displacements of the head of the humerus have been observed by Scudder[4] (two cases), by H. B. Robinson[5] (two cases), and by J. B. Roberts.[6] In all the dislocation was of the backward or subspinous variety. In one of Robinson's both shoulders were affected.

Acquired luxations of this joint have been reported by Chambers[7] in a boy four years old (subspinous), and by Brackett[8] in a boy aged two (into the axilla). Both were easily reduced by manipulation.

Elbow.—A curious case is recorded by Kelly.[9] A man who had accidental luxation outward and backward of the ulna stated that his father, when a young boy, had had his right elbow dislocated, and a year afterwards the left; reduction was not effected in either case. Two of the patient's brothers had also had luxations of the elbow.

Abbott[10] reported nine instances of congenital dislocation of the radius, seven of them (all forward) occurring in one family. These cases were scattered over four generations; five of them, belonging in three generations, having been personally examined by Abbott. In all flexion and extension were free, but there was no pronation or supination. The lesion seemed to have been determined by a mass of bone from the outer side of the coronoid process of the ulna.

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[1] Revue de Chirurgie, April 10, 1893.

[2] New York Medical Journal, September 12, 1891.

[3] Lancet, October 3, 1891.

[4] Archives of Pediatrics, 1890.

[5] Lancet, March 4, 1893.

[6] Transactions of American Surgical Association, 1895.

[7] Nashville Journal of Medicine and Surgery, July, 1889.

[8] Medical Record, September 27, 1890.

[9] Lancet, February 25, 1893.

[10] Ibid., April 9, 1892.

In the other two joints (in the body of one child two years old), the right one was a luxation of both bones backward, the left being upward, backward, and inward, due seemingly to a defective formation of the trochlea.

Wrist.—Moriarty[1] reports a luxation forward of this joint, in a boy fifteen years old, from a fall on the palm of the hand.

In an instance recorded by Guépin,[2] a whole family, of three generations, had laxity of the ligaments of the wrist, so that the head of the ulna was markedly prominent and mobile. The lesion seemed to be due to the carrying of heavy loads.

Hip.—In the summer of 1890 I saw within ten days two cases of dorsal dislocation of the hip, one in private practice in a girl aged ten years, the other at the Pennsylvania Hospital in a girl aged nine. Both were easily reduced by manipulation under ether, and both did well.

Other instances of dorsal dislocations have been recorded by Dixon,[3] in a girl aged six, reduced by extension outward; by Douglas,[4] in a boy seven and a half years old, reduced by manipulation; and by Clark,[5] in a boy aged four, reduced by manipulation on the third day.

Sciatic luxations (dorsal below the tendon) have been reported by Turner,[6] in a girl aged five, and by Davidson,[7] in a Hindoo boy three years old. Both these were reduced by manipulation, the latter at the end of the second week.

A dorsal dislocation requiring arthrotomy, in a boy aged four years, has been recorded by Helferich.[8] The operation was successful.

In a case reported by Lange,[9] a girl seventeen months old had had six weeks after birth "pneumonia and convulsions," after which her left leg was rotated outward and abducted; the diagnosis was luxation into the thyroid foramen. The course to be pursued had not been determined at the time of the report.

A dorsal luxation, the result of chorea, in a girl seven years old, is recorded by Nichol.[10] Reduction was easily effected.

Knee.—Robinson[11] states that in two years he met with three cases of subluxation of this joint, due apparently to impairment of the general health. The patients were all female children about one year old, and all recovered under tonics and massage.

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[1] British Medical Journal, April 27, 1890.

[2] Comptes rendus de la Société de Biologie, 1892.

[3] Lancet, November 2, 1889.

[4] Ibid.

[5] British Medical Journal, March 28, 1896.

[6] Australasian Medical Gazette, August, 1892.

[7] Lancet, April 29, 1893.

[8] Deutsche Medicinische Wochenschrift, August 10, 1893.

[9] New York Medical Journal, February 16, 1889.

[10] Lancet, March 11, 1896.

[11] British Medical Journal, July 27, 1895.

Congenital dislocation of the knee has been noted by McGillicuddy,[1] Karewski,[2] and Bloch[3] (two cases). Karewski refers, in connection with the case observed by him, to thirteen other reported instances. The lesion would seem to be the result of pressure by the uterine wall by reason of deficiency in the quantity of liquor amnii, and the affected limbs are sometimes imperfectly developed.

Ankle.—Hardyman[4] has reported the case of a girl seven years of age, whose foot was run over by a wagon, and the first, third, and fourth metatarsal bones were dislocated upward on the tarsus. Reduction was effected under chloroform.

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[1] Journal of the American Medical Association, July 23, 1892.

[2] Archiv für Kinderheilkunde, 1891.

[3] Prager Medicinische Wochenschrift, December 21, 1892.

[4] British Medical Journal, February 6, 1892.

EPIPHYSEAL SEPARATIONS.

By RICHARD H. HARTE, M.D.

TRAUMATIC injury, or separation of the epiphysis, is regarded by the majority of surgical writers as a rare injury, but careful observation among patients where the accident is possible leads one to believe that many cases are not recognized as such in the proper sense, and are regarded as fractures about or into a joint. The importance of careful recognition makes this accident worthy of special study.

Separation of the epiphysis is of even more consequence than fracture. Its occurrence in early life and the important effect which may ensue call for special consideration. The frequent error in diagnosis which separation of certain epiphyses has caused may be attributed possibly to insufficient and inexact teaching on the subject by many of the text-books on surgery.

Complete separation of an epiphysis is usually accompanied by a much greater stripping off of the periosteum from the shaft of the bone than is ordinarily found in a simple fracture; hence the greater tendency of such injuries to be followed by suppuration and necrosis and, during repair, an excess of callus, which may in itself interfere greatly with the function of the joint.

In many instances a detachment of the epiphysis implies an injury to the joint which it helps to form,—*e.g.*, in the lower end of the humerus.

Much difficulty is experienced in replacing and retaining certain epiphyses in position after separation,—a difficulty which is rare in dealing with fractures of the shafts of the bones.

Careful study of a bone taken from a young subject is both interesting and instructive. The knowledge of its growth and development makes clear what otherwise might be imperfectly understood. It will be noticed that the periosteum at the epiphyseal attachment is, as a rule, much thicker where it is attached to the cartilaginous disk and partakes more of the character of a ligament. This anatomical fact will explain why so much more bone is often denuded of its periosteum than occurs in simple fracture,—the thicker and denser the periosteum the more likely it is to strip off *en masse*, as we have seen it do in compound separations of the lower end of the femur, where almost one-half of the shaft of the femur was divested of its periosteum. The ligamentous character of the periosteum is often the strongest bond of union between the diaphysis and the epiphysis. It has been shown in the small epiphysis of the head of the femur, where the periosteum was divided circularly, that one-fifth of the amount of force will suffice to detach the epiphysis which would be required under other circumstances. It has been observed in the great majority of cases of separation that the cartilaginous disk is adherent to the epiphysis. This is again verified, first, by the experiments of Hutchinson on animals, where, upon examining and making sections through the epiphysis, it was noticed that the trabeculæ were arranged in a much stronger and better manner on the epiphyseal side of the disk, so that on separation it will invariably be found adhering to the epiphysis.

The delicate attachment of the epiphyseal disk would lead one to expect that either the violence necessary to detach or the reaction attending subsequent bony union would be sufficient to obliterate it and check its further development. Fortunately, this is not always the case, as the disks show a remarkable power of resistance. If this were the case, Ogston's operation for the correction of genu valgum would have fallen into disuse much sooner than it has. On the other hand, it must be remembered that arrest of growth is a fairly frequent result of epiphyseal separation, and particularly so if the latter remains displaced and connected only by the periosteal new bone to the diaphysis. The arrest of development after epiphyseal separation is most disastrous when it affects only one bone of either forearm or leg. Usually the ulna or fibula will continue to grow, pushing either hand or foot over to the opposite side and distorting the ankle- or wrist-joint, which will demand for its correction subsequent surgical interference. The main difficulty in the reduction of the epiphysis after separation is due principally to the position of the soft parts, especially the sleeve of the periosteum, through which the diaphysis has been forced. In the lower end of the radius one or more tendons may become entangled with the fragment, thus preventing reduction.

It is most important to effect reduction as soon as possible, as the rapid occurrence of swelling will make the diagnosis and the reduction more difficult. The connecting bridge of the periosteum soon thickens and shortens, in a few days rendering replacement impossible; therefore the more prompt the reduction the less will be the risk of exuberant callus or of interference with the growth, and possibly of suppuration or necrosis. From what has thus far been stated, early and accurate replacement of the bone, with careful avoidance of rough manipulation and with the application of cold to check all inflammation, is incumbent on the surgeon in dealing with these injuries. I feel that the friends of the patient should be advised by the surgeon of the character of the injury and the possible danger of progressive deformity due to an arrest of development, and of the danger of suppuration and necrosis. The stripping off of the periosteum from the shaft which occurs with some displacements is extraordinary in extent, and by some much importance has been attached to this periosteal stripping as the chief factor in producing suppuration.

The tendency of some epiphyses after separation to undergo suppuration is very marked, and it would appear, from analyzing a large number of reported cases, that those epiphyses which are most active in carrying the nutritive forces—*e.g.*, the epiphyses in relation to the shoulder, wrist, and knee—are most liable to be thus affected, because we find that the principal growth or development takes place at the shoulder and wrist in the upper extremity and at the knee in the lower extremity. It has been shown that the exact dates given for the fusion of the various epiphyses differ considerably. As expressed by Mr. Hutchinson, "the chronometer of life may be sometimes fast and at other times slow."

I pass now to the consideration of individual epiphyses, beginning with the *sternal end of the clavicle*, which is one of the most anomalous in the body. The epiphysis begins to ossify at eighteen and is joined to the shaft at twenty-five years, and forms but a thin lamina of bone.

These injuries are rare and the authenticated cases few in number. Tubby[1] has collected three cases, one of which is compound and the other two seem so clear that the diagnosis cannot be questioned. This injury is most likely to be mistaken for a dislocation of the sternal end of the clavicle. The undue prominence of the inner end of the clavicle and the presence of a sharp edge beneath the skin are so unlike the smooth end of bone covered with cartilage that the absence of swelling and the knowledge that such an injury is possible leave little doubt in the diagnosis. The cause in two of these cases was assigned to muscular action and in one to the passage of a wheel over the end of the bone.

Treatment.—Reduction, rest on the back in bed, and a figure-of-8 bandage will usually be effectual.

Separation of the Acromion Epiphysis of the Scapula.—So far as I have been able to learn, this injury has not been recognized in children. This process is developed from two nuclei appearing about the fifteenth or sixteenth year and consolidating about the twenty-fifth year. Nearly all museums possess specimens which exhibit ununited fractures of this process. I agree with Hamilton in regarding them as epiphyseal separations which have failed to unite; in fact, the epiphyseal tips may remain separate throughout the life of the individual, as the acromion is often found separate in macerated specimens.[2]

The treatment should be similar to that directed for fracture of the acromion process.

Separation of the Upper Epiphysis of the Humerus.—This epiphysis is ossified from three centres, these centres fusing together at the fifth year, and the whole mass uniting about the twentieth year, although occasionally this is prolonged until the twenty-fifth year. Uffelmann asserts that it may be postponed to the thirtieth year. This epiphyseal line is a very irregular one, and follows somewhat the contour of the rounded head of the humerus. In early life, before the fifth year of age, the bony nucleus of the head is within the capsule of the shoulder-joint; after that age almost the whole of the capsule is attached to the epiphysis and all the rotators of the arm, and consequently the number of vessels they carry enables us to understand how frequently firm union follows after separation of this epiphysis, differing from that obtained in cases of similar injury to its analogue, the head of the femur. As this epiphysis is mainly instrumental to the growth of the humerus, it follows that injuries to this epiphyseal line are sometimes followed by more or less shortening of the limb. In most cases of separation the capsule of the shoulder is not opened, and the younger the patient the less likely is it to occur. Mr. Hutchinson regards it as a point of practical importance that separation of the epiphysis before the age of twenty takes the place of dislocation of the humerus, only about one per cent. occurring in persons under twenty years of age. This is verified by my observations at the Pennsylvania Hospital. Out of two hundred and twenty-five dislocations of the shoulder treated there during the last ten years, two occurred in boys aged fourteen and seventeen respectively, and four in persons between nineteen and twenty years of age. The younger the subject the more common are these cases of detachment without displacement. Whether muscular action is responsible for epiphyseal detachment I am unable to say; it can, however, cause displacement of the humeral head in adults.

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[1] Guy's Hospital Reports, 1889.

[2] Morris, Human Anatomy, p. 134.

Diagnosis.—The age of the patient is under twenty years. The arm hangs helplessly and the elbow projects slightly from the side. Below the head of the humerus, in the line of the acromion process, is often a slight depression, particularly if the elbow is carried away from the side of the body. On the inner side of the arm, below the acromion process, will be noticed an abrupt projection caused by the upper extremity of the diaphysis being drawn in by the muscles, forming the anterior and posterior axillary fold.

By directing the elbow inward and making extension and counter-extension, crepitus of a soft, moist character will be elicited, and the deformity can be reduced without much difficulty, but will soon recur when the arm is released. The natural roundness of the shoulder is not altered, and, as a rule, there is but little shortening. One valuable guide which is useful in determining the injury is the fixation of the head of the humerus with one hand and making the shaft rotate by grasping the elbow with the other hand. It will be noticed that the movements are angular in character and the head is stationary. If the head is not fixed, owing to its concavity and the lower fragments fitting into it, rotation will take place in the entire length of the bone.

The most common form of injury about the shoulder for which the displacement of the epiphysis will be mistaken is dislocation of the head of the humerus, either under the coracoid or clavicular. As before stated, dislocation under the twentieth year is very rare, and when reported as such is found almost universally to be an epiphyseal separation which has not been recognized. In some points the two injuries resemble each other, but the most reliable guides are the reappearance of the deformity after reduction, the presence or absence of crepitus, and the ability to bring the elbow in contact with the side. In a severe case, with much swelling and bruising, it is difficult to make an immediate or precise diagnosis.

A very important question is how much cases will be benefited by operation when the injury has been neglected, or not recognized, for weeks or months after the accident. The bad results that may be expected are deformity, arrest of growth, and ankylosis. Much will depend upon the individual case and the operative skill of the surgeon. Judging from the experience of cases that have been operated on, the results are most favorable.

If the separation is compound, with laceration of the vessels, amputation will be demanded. Otherwise, the replacing of the parts or resection may be called for, depending on the character of the injury.

Treatment.—When perfect reduction is secured the bones are best retained in position after the manner of dealing with fractures of the neck of the humerus: *e.g.*, a properly adjusted pad in the axilla and a well-fitting shoulder-cap, the arm returned to the side and the hand supported in a sling, will fulfil all indications in treatment.

Separation of the Lower Epiphysis of the Humerus.—The development of this epiphysis is rather complex, consisting of four nuclei, two of which are on the articular surface, the one for the capitulum appearing about the third year, the nucleus for the trochlear surface appearing about the tenth year. These epiphyses for the first few years increase steadily in width but comparatively little in depth. A centre appears for the epicondyle about the fifth year, and as development progresses it becomes more distinct from the lower epiphysis. From this it would seem that the lower epiphysis of the humerus would be more liable to be detached *en masse*, taking with it both condyles. These displacements are uncommon before the sixth year of age, but may occur years later.

The deformity resembles a posterior dislocation, and is often mistaken for it. If seen soon after the injury, before the joint is masked by swelling, the end of the diaphysis has not the characteristic outline of the trochlea and capitulum, and the elbow can be over-extended beyond a straight line. There is slight shortening, and the deformity can readily be corrected, but immediately recurs. It must be remembered that dislocation of the elbow is not an uncommon accident in children; in fact, the majority of dislocations occur before the fifteenth year, differing entirely from what is found in the shoulder.

The complication arising from these injuries is impairment of function due mostly to excessive callus, sometimes extending up the bone and invading other vessels or nerves. In obscure cases, where the landmarks are masked by inflammatory swelling, the use of the Röntgen rays will assist in arriving at the exact character of the injury.

Treatment.—The indications are for the correction of any existing deformity and to maintain the fragments in position. The smallness of the lower fragment always makes it difficult to deal with. Inflammation should be controlled as far as possible by cold or evaporating lotions, the arm flexed or placed on an angular splint and laid on a pillow, resting in bed for at least a week. A pasteboard or plaster of Paris splint, carefully moulded to the arm, has some advantage over the wooden splint. In all cases the splint should extend to the ends of the fingers. To depend simply on a sling, keeping up constant passive motion, is not advisable. It is advocated by some to treat the limb in the extended position with straight splints; when stiffness follows, as it at times does, it leaves the arm at great disadvantage.

Separation of the Internal Epicondyle of the Humerus.—These displacements are usually regarded as due to muscular violence, as stated by Granger,[1] but the evidence of the four reported cases is strongly in favor of direct violence, as evinced by the amount of ecchymosis and the injury done to the ulnar nerve. The symptoms and diagnosis are very evident if seen before much swelling has set in.

Separation of the Upper Epiphysis of the Ulna.—This small epiphysis is not deposited until about the tenth year, and unites about the sixteenth year. It occupies but a small portion of the end of the olecranon. It may become displaced as the result either of direct violence or possibly of muscular action. Hamilton produced this displacement in the reduction of a dislocation in a child seven years old. The symptoms of this injury are nearly identical with those of fracture of the olecranon, the points of difference being the age of the patient and the absence of bony crepitus if the fragment can be sufficiently reduced.

Separation of the Lower Epiphysis of the Ulna.—It is possible that this epiphysis may become detached as the result of direct violence. In a case reported by Mr. Hutchinson,[2] in a boy aged fourteen who had a compound fracture demanding amputation, the lower epiphysis was found on dissection clearly separated and wedged in between the shaft and the lower end of the radius. The wrist-joint was not opened.

The age of the patient and the size of the epiphysis and its superficial character aid in making a diagnosis. The treatment should be similar to that advised for fracture in proximity to the wrist-joint,—*e.g.*, a straight splint which fixes the hand and forearm. If any lateral displacement recurs, it is best corrected by a short lateral splint on the outer side of the forearm along the ulna. The non-recognition of these separations apparently has led to permanent deformity.

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[1] Edinburgh Medical and Surgical Journal, vol. xiv. p. 196.

[2] British Medical Journal, March 31, 1894.

Separation of the Upper Epiphysis of the Radius.—This small epiphysis is laid down about the fifth year and fuses with the shaft at the seventeenth year. It differs from other epiphyses, being entirely within the capsule of the elbow-joint and surrounded by the orbicular ligament, and having no muscular or ligamentous attachments. Many cases of this displacement are recorded, but a more careful analysis will prove them to be dislocation of the radial head rather than separation of the epiphysis, which can only be displaced by direct violence. In obscure cases the use of the Röntgen rays will aid materially in verifying the diagnosis.

Separation of the Lower Epiphysis of the Radius.—This epiphysis, with the corresponding one at the end of the ulna, is largely instrumental in the growth of the forearm. Appearing at the second year and fusing at the twentieth year, the line of the epiphyseal disk runs transversely across the lower end of both bones. From its position it is particularly liable to displacement, resulting in great deformity and serious impairment of function in the hand in after-life.

The great strength of the ligament of the wrist-joint connecting the epiphysis to the carpus would lead one to suppose that fracture would be an impossible accident under twenty years of age. This statement is not entirely accurate, as I have seen Colles's fracture in children which was verified by post-mortem. Still, it is a safe statement that three-fourths of the cases diagnosed as Colles's fracture are true epiphyseal separations.

Little difficulty should be experienced in determining the character of the separation, especially if the age of the patient is considered. In a true separation the abrupt end of the diaphysis can be distinctly felt, the epiphysis on the ulna going with the radial epiphysis, and may be mistaken for dislocation of the wrist-joint. Where this takes place the joint relations are unaltered, but the deformity above the joint is very great, characterized by the silver-fork displacement. The crepitus elicited by bringing the surfaces together differs from fracture, being moist in character.

In dealing with these cases successfully everything depends, as in a Colles's fracture, on thorough reduction of the deformity, which should be done under an anæsthetic. When thoroughly reduced there is no disposition of the deformity to reappear. The wrist should be placed on the same splint as is used for the treatment of a Colles's fracture (Bond's). Passive motion of the fingers should begin on the third day and gentle motion in the wrist-joint on the sixth day. If this is systematically pursued, there will be but little stiffness of the fingers and wrist, which, however, is sure to follow if the treatment indicated above is omitted.

Other complications may follow, as arrest of growth, abscesses, and necrosis.

Separations of the Metacarpus and Phalanges.—These injuries are rare. Sir Astley Cooper says that the appearance resembles dislocation of the phalanges. Lucas reports such an accident[1] in which reduction was difficult.

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[1] Lancet, vol. ii., 1885, p. 801.

with a tendency to recur. In subjects under eighteen years the possibility of this injury should be carefully considered.

Separation of the Upper Epiphysis of the Femur.—Up to the twelfth or eighteenth month the head of the femur is cartilaginous. In this is deposited an ossific centre, this cartilaginous disk corresponding to the margin of the acetabulum. The attachment of the capsule is a short distance below the epiphyseal line, differing from what is found in the upper end of the humerus. True separation has occurred, as verified by post-mortem examination.[1] The patient was run over by a vehicle and died of shock. The capsule of the joint was opened and the epiphysis for the head separated through the line of the disk. It is an injury well recognized by veterinarians, and many specimens abound in museums where both epiphyses have been separated during coition, especially in pigs. Tubby[2] has analyzed nine out of fourteen collected cases. The evidence is strongly in favor of separation of the epiphysis rather than fracture.

The average age is about fourteen years, the epiphysis consisting mainly of bone. It is therefore capable, to a large extent, of accommodating itself to sudden shocks. The thin epiphyseal line is the weakest part of the neck, and, consequently, indirect violence spends itself at that point.

The diagnosis is often difficult; the symptoms simulate very much those of fracture of the neck of the femur in adults, though eversion is less marked. In a case under our care in the Episcopal Hospital we found the following points of assistance in determining the character of the injury:

The age sixteen years. Pain less marked than would be expected in fracture of the neck. Crepitus of a peculiar dull variety and not always elicited. Undue mobility of the upper portion of the femur. Shortening from one-half to three-fourths of an inch, which was readily overcome by slight extension of the leg. Elevation of the trochanter above Nélaton's line. Two important factors in determining the diagnosis are, first, the age of the patient, under twenty years, and, second, the weak spot of the neck in the epiphyseal line, it being more reasonable to expect the bone to give way there than in the bony tissues on either side. Further, there are increased mobility, absence of impaction, and, apparently, less muscular rigidity. It can hardly be mistaken for dislocation, as the range of motion is limited and the joint relations are decidedly changed. It is hardly necessary to say that an anæsthetic should be administered before an examination is made. In our case we failed to derive any assistance from the use of the Röntgen rays, owing to the depth of the pelvis.

Treatment consists of extension and lateral support, either with sand-bags or, what is preferable, the long Liston splint, with junk-bags, which give better support, besides keeping the joint quiet. Progressive shortening due to impairment of growth here is not so important a factor in the case, as the principal growth of the limb takes place from the low epiphysis in relation with the knee-joint.

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[1] M. Bousseau, Les Bull. de la Soc. Anat., 1867, p. 283.

[2] Annals of Surgery, 1894, p. 289.

Separation of the Epiphysis of the Great Trochanter.—This process is occasionally separated by muscular action or by direct violence, the latter being the most common cause. This injury is illustrated by a specimen in the cabinet of Professor Ashhurst in the University of Pennsylvania. Hutchinson[1] states that five out of eleven of these separations were followed by suppuration, and five terminated fatally. The diagnosis is difficult and can be determined only by the mobility of the trochanter, which, if its periosteum is attached, forms a hinge-like movement on direct pressure.

The treatment demanded here would be an attempt to retain the trochanter in place by means of strips of adhesive plaster and the limb immobilized by means of a long Liston's splint.

Separation of the Epiphysis of the Lesser Trochanter.—This is a rare accident. The life of this epiphysis is short, its ossific centre appearing about the fourteenth year and uniting to the shaft about the seventeenth year. So far as I have been able to learn, but one case is reported,—that due to muscular violence in a lad, caused by jumping over a fence. The specimen is now in the museum of McGill University, the patient dying of septicæmia.

Separation of the Lower Epiphysis of the Femur.—This large epiphysis is the first laid down in the skeleton, appearing at the ninth month of fœtal life and uniting to the shaft about the twentieth year. This epiphysis includes all of the articular surface of the femur, and its detachment must mean injury to the synovial membrane. The adductor tubercle is on a line with its upper margin, and in the majority of cases the two heads of the gastrocnemius muscle are attached to the upper margin of the epiphysis; hence the marked tendency to posterior rotation of the epiphysis after separation. The great breadth of the femur at the epiphyseal line and the unusual strength of the periosteum are factors which lessen the frequency of this accident. Yet, on the other hand, when we note the strong ligamentous and muscular attachments, with the leverage of the leg, it can readily be seen why this accident is more common than has been supposed by many surgeons. When separation occurs the cartilaginous disk will invariably remain with the epiphysis. Such was my experience in three cases which terminated in amputation. I am persuaded that the accident is much more common than the text-books on surgery lead us to believe. Hamilton cites only six cases, one of which came under his own observation, and that three years after the receipt of the injury. I have observed seven cases, five of which are reported.[2] Three demanded amputation, one died of hemorrhage, two recovered with useful limbs, and one with ankylosis. The result in the last case was to be expected, as the knee was stiff before the receipt of the injury,—certainly a gloomy record. This accident, in the vast majority of cases, is due to direct violence. Out of twenty-six cases analyzed by Tubby,[1] fifty per cent. occurred by entanglement of the leg or foot in the spokes of a revolving wheel. The examination of over sixty cases shows that all except nine were boys, whose more restless and adventurous spirit naturally exposes them to greater dangers.

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[1] British Medical Journal, March 31, 1894.

[2] American Journal of the Medical Sciences, June, 1896.

Symptoms.—Pain, if very severe, may be due to stretching or pressure on the external popliteal nerve. As a rule, there is less pain than in simple fracture. Crepitus may or may not be elicited. If present, it is obscure or indistinct, although mobility may be very marked.

Deformity.—This symptom is always to some extent present, but is often misleading, owing to the rapid effusion which takes place in the knee-joint. The knee is usually semiflexed and the foot everted. The epiphysis will often bear its normal relation to the knee, especially if the knee be held in position by the strong ligaments of the joint, the displacement consisting of the lower end of the diaphysis with its sharp margin pressing against the vastus internus muscle, giving the appearance of genu valgum, if the joint is flexed, causing the joint to appear to be too high. Rotary displacement of the epiphysis by the gastrocnemius has been mentioned by Packard and others.[2] This displacement is sometimes responsible for injury to the popliteal vessels, and must be guarded against.

Diagnosis.—There are two forms of injury with which it is likely to be confounded,—*e.g.*, dislocation of the knee and fracture above the condyles. The former is very rare in adults and practically impossible in young subjects, as in attempts to artificially dislocate the knee in this class of patients separation of the epiphysis is always the result. The rapid swelling which follows these injuries often masks the character of the injury. In questionable cases the age of the individual and the character of the crepitus, if elicited, will aid in making a diagnosis. In extreme cases the Röntgen rays can be relied upon to verify the diagnosis.

Treatment.—This consists of perfect reduction of the fragment. If rotation has taken place and the gastrocnemius offers much resistance, flexion of the knee or severing of the tendo Achillis will be necessary. After reduction, extension by means of weights, as in simple fracture of the thigh, and lateral support by means of sand-bags. I prefer junk-bags and long lateral splints retained by means of a splint cloth. Extreme swelling should be combated by ice-bags, lead water, and laudanum. Compound cases must be treated according to the merits of the case. If much of the periosteum has been stripped off all sides of the bone, amputation will be the safest procedure. If the wound is small and there is not much destruction of the periosteum, thorough cleansing and closing of the wound, with the treatment for compound fracture of the thigh, will sible. If the end of the diaphysis projects through a small wound and reduction is impossible, resection of such an amount of the projecting bone as will admit of perfect reduction will be demanded. If the diaphysis is denuded of much periosteum, amputation will be the safer procedure. The time required in simple separation is usually two weeks longer than is demanded in simple fracture.

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[1] Annals of Surgery, 1894, p. 310.

[2] Annals of Gynæcology and Pediatry, November, 1890.

In simple uncomplicated cases, where reduction can be readily accomplished, I am disposed to believe that the future growth of the limb will not be impaired.

Separation of the Upper Epiphysis of the Tibia.—This large epiphysis is the second one deposited in the body, appearing at birth. A strong resemblance exists between it and the lower epiphysis of the femur. Its long unattached lip might lead to the supposition that it would, like its fellow, be liable to displacement. On the contrary, it is a comparatively rare accident. This is partly due to the attachment of the ligamentum patellæ, the internal lateral ligament, and the semi-membranous tendon to both the diaphysis and epiphysis, thus assisting in binding them together. The anterior portion of the epiphysis has a downward projection in front of the tubercle of the tibia. This is occasionally developed from a separate centre. Müller[1] has collected five cases of separation of the tubercle at this point.

The diagnosis will be aided by the character of the injury and the age of the patient,—*e.g.*, under twenty years. It may be greatly obscured by joint swelling and effusion, and some days may elapse before a positive diagnosis is determined. In obscure cases the Röntgen rays will demonstrate if any displacement exists.

Treatment.—After perfect reduction there is not much tendency to displacement. The methods employed for the treatment of fracture in the region of the knee will fulfil all indications,—*e.g.*, the long fracture-box, to be followed after the subsidence of swelling by a plaster bandage. The three cases of Mr. Hutchinson's[2] were followed by no bad results. If suppuration should follow and invade the knee-joint, amputation will be demanded.

Separation of the Lower Epiphysis of the Tibia and Fibula.—There is some resemblance between these and the corresponding ones in the lower end of the radius and ulna. They remain cartilaginous up to the second year, and unite to the shaft at the eighteenth and twentieth years respectively. The line of the disk is almost straight, slightly lower in the fibula. If both are separated, the lower end of the diaphysis of the fibula will present below the line of the tibia. The separation of both epiphyses is a more common injury than that of the tibia by itself. This displacement may be said to take the place of Pott's fracture in young subjects, and may be produced by either extreme eversion or extreme inversion.

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[1] Bruns's Beiträge, 1887.

[2] British Medical Journal, March 31, 1894.

The arrest of development, when only one of these double epiphyses is detached, is liable, as in the radius or ulna, to cause marked progressive deformity. This is also possible in the ankle, but my attention has not been called to it as it has in the wrist-joint.

When these displacements at the lower end of the tibia and fibula are compound, they, as a rule, can be treated more conservatively than injuries near other articulations. Perfect reduction is important, and if the diaphysis offers much resistance its ends should be resected.

Diagnosis.—This injury may be confounded with partial dislocation of the ankle. The character of the crepitus and the age of the patient will assist in making a correct diagnosis.

Treatment.—The treatment after reduction will correspond to that of fracture of both bones of the leg: a fracture-box until the swelling subsides sufficiently for the application of a plaster bandage.

DISEASES OF THE SKIN.[1]

BY HENRY W. STELWAGON, M.D., PH.D.

ANGIOMA SERPIGINOSUM.

THIS disease is rare, and is also known under the names of infective angioma and nævus lupus. It is characterized by groups of apparently hemorrhagic puncta, arranged in ovals or circles, usually dime- to quarter-dollar-sized. The extension occurs at the border, the centre remaining, as a rule, unchanged. It is slow in its evolution, and is not accompanied by any subjective symptoms. It may appear upon any part of the surface, but is most commonly seen about the shoulder, chest, arm, and leg. The reddish puncta are made up of tufts of capillaries. The cause of the affection is not known. Late investigations point to the disease as an angiosarcoma. Treatment consists of excision or cauterization.

DERMATITIS EXFOLIATIVA NEONATORUM.

This is an extremely rare disease, manifesting itself in the first few weeks of life. It is characterized by more or less generalized redness, with branny or flake-like scaliness. It may be either dry or undermined with serous exudation, as in pemphigus foliaceus. Blebs are occasionally present. About half these patients die, usually from marasmus. If recovery is to take place, the skin becomes paler and the scaliness gradually ceases. It is looked upon as of septic nature. Treatment is symptomatic.

DERMATITIS GANGRÆNOSA INFANTUM.

This rare disease is also called varicella gangrænosa, pemphigus gangrænosus, rupia escharotica, gangrenous infantile ecthyma, and multiple disseminated gangrene of the skin. It is not at all common, and is observed to follow vesico-pustular and pustular eruptions, such as varicella and vaccinia. The disease is seen in young children, rarely above the age of three, and more especially in girls. It usually begins while the varicella or other lesions are still existing, the changes taking place beneath the crusts, both downward and peripherally. The slough falls off, disclosing an ulcer of some depth and varying in diameter from one-fourth to three-fourths of an inch or more. When seated closely together, several may become confluent, and an irregular, more or less deep, ulcerated or sloughing area results. The disease sometimes follows after the varicella lesions have disappeared, and sometimes the disease arises spontaneously, without a preceding vaccinal or varicellar eruption. There may be only ten or twenty such sloughing ulcers, or they may be scattered in great numbers over the entire surface. In the more severe cases the constitutional disturbance is necessarily marked, and death results. The diagnosis is not difficult, as the symptoms are characteristic.

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[1] This article is written as a supplement to Part I., vol. ii.

The external treatment is based upon general principles, a mild antiseptic being applied and perfect cleanliness maintained. The constitutional treatment is to be of an invigorating and tonic character.

DERMATITIS VENENATA.

By the term dermatitis venenata are meant all those inflammatory conditions of the skin due to contact with deleterious substances. The most conspicuous cases to which this term is applied, however, are those in which the action is due to various plants, especially the rhus plants,—poison-ivy or oak and poison-sumach or dogwood. The symptoms of this variety of dermatitis venenata—rhus or ivy poisoning—vary from a mild erythema to a violent erysipelatous pustular and bullous inflammation. The inflammation presents itself, as a rule, shortly after exposures, usually from a few hours to a day. The parts most commonly affected are the hands, forearms, face, and genitalia. Some parts are affected secondarily through contact with affected parts. Well-authenticated cases of the disease being conveyed from one individual to another are rare. In the course of from one or two days to several weeks, depending upon the severity and character of the attack, the symptoms begin to subside. An average case runs its course in from ten days to three weeks. In mild cases of the erythematous type it may disappear in from one to several days. As a rule, there is considerable itching or burning present. In those having a predisposition to eczema, this latter disease may be provoked and continue for some time. The diagnosis of rhus poisoning is rarely difficult, the acuteness of the attack, the character of the eruption, and the history of exposure usually sufficing.

There are many vaunted remedies for this affection, but the fact to be remembered is that soothing and slightly astringent treatment is indicated. Boric acid solution, with from five to twenty grains of borax to the pint; the calamine and zinc lotion employed in acute eczema; the fluid extract of grindelia robusta, one or two drachms to four ounces of water; dilute lead water; lotio nigra; a solution of zinc sulphate, from one-half to two grains to the ounce, with or without a few minims of carbolic acid to each ounce; plain carbolic acid lotion of the strength just indicated; weak alkaline lotions; mild ointments, such as zinc oxide, cold cream, petrolatum, and the like, are all useful.

ERYTHEMA INDURATUM.

Erythema induratum, the *érythème induré scrofuleux* of Bazin, is sometimes seen in young boys and girls. The calf is the favorite region, although it is exceptionally observed upon the forearms. It begins as one or several —rarely many—deep-seated, hard, pea- to cherry-sized nodules, which in the course of one or two weeks have become somewhat larger, and the overlying skin of a pinkish, dull-red, or violaceous color. The nodules later are or may be from the beginning ill defined, feeling like an irregular, flattened, subcutaneous infiltration, at times not unlike deep-seated erysipelatous inflammation. The lesions may gradually undergo absorption or may slough and ulcerate; this latter is the more common. The resulting ulcer is deep, with a decided punched-out appearance strongly suggestive of a syphilitic ulcer, with which the disease is, however, in no way related. The disease is slow in its progress; the older ulcerations may remain, varying somewhat in character, or they may heal, new nodules appearing from time to time and undergoing the same changes. It may last almost indefinitely. Experience lends strength to the belief that the disease is of a scrofulous nature.

Treatment is not always satisfactory, especially without the full co-operation of the patient. Rest, relative or absolute, in the recumbent posture is of greatest benefit. Cod-liver oil, iron, strychnine, and phosphorus are the most useful internal remedies. Locally, mild antiseptics are valuable: a lotion of a saturated solution of boric acid, with one or two drachms of resorcin to the ounce, resorcin ointment, from one-half to one drachm to the ounce, and a five to ten per cent. oleate of mercury ointment may be mentioned as being the most serviceable.

HERPES SIMPLEX.

Herpes, herpes facialis, or fever blisters, may be described as an acute disease of an inflammatory type, occurring most commonly about the lips, and characterized by the formation of pin-head to pea-sized vesicles, arranged in one or several groups. If the case is severe, mild pyrexic symptoms with more or less malaise may precede the eruption. Usually, however, only a feeling of heat or burning is felt at the site at which the vesicles make their appearance. They are usually closely grouped or bunched on a hyperæmic or inflammatory base. At first their contents are clear, later becoming milky in appearance. There is no tendency to spontaneous rupture; if broken accidentally, an abrasion or excoriation of a superficial nature results. In the course of a few days the eruption dries to crusts, which in a short time drop off. No permanent trace of the disease is left. The disease is often seen in association with febrile and pulmonic diseases. It is also not infrequently observed in association with malarial and digestive disturbances.

Very little, if any, treatment is needed for the eruption itself. Predisposing conditions should be looked after. Locally, a camphorated cold cream or a boric acid ointment may be used. In the beginning of an attack the frequent application of spirits of camphor or Cologne water will sometimes serve to shorten the duration of the outbreak.

HYDROA VACCINIFORME SEU ÆSTIVALE.

This disease is also designated hydroa puerorum and recurrent summer eruption. All recorded cases have been boys. The disease begins in early childhood or infancy, and, as a rule, on uncovered parts. Its appearance is usual after exposure to wind or sun. The beginning lesions are red spots, upon which vesiculation takes place. In a day or two the vesicle shows distinct umbilication, the contents becoming milky. The lesions gradually dry up and the crusts fall off, leaving distinct pit-like scars. Successive outbreaks may present themselves and the attack be prolonged almost indefinitely. The vesicles develop either singly and discrete or in groups like zoster. In the winter months the patient is generally free. Recurrences usually are noted up to the period of manhood, when the disease gradually disappears. In severe attacks there may be arthritic symptoms, anorexia, and similar disturbance. The disease is probably a vaso-motor neurosis. So far treatment has seemed simply palliative. The patient should be protected against sun exposure, excessive artificial heat, and heavy winds. In other respects the measures of treatment are based upon general principles.

IMPETIGO CONTAGIOSA.

Impetigo contagiosa, known also as porrigo contagiosa, impetigo, impetigo simplex, is an acute, contagious, inflammatory disease, characterized by the formation of discrete, superficial, flat, rounded, or oval-like vesicles or blebs, soon becoming vesico-pustular, and drying to thin, wafer-like, yellowish crusts.

While the etiology is not positively known, the disease is now looked upon as one of pus cocci infection,—staphylococcus aureus and albus. For this reason it is occasionally seen in connection with vaccination and pediculosis capitis and other diseases in which pus may be present. It is essentially a disease of childhood, the vast majority of cases occurring in those under the age of ten or twelve. In some instances of the more or less general eruption, with slight preceding and sometimes accompanying febrile action, it bears close resemblance to the eruptive fevers. It is possible that a condition of cachexia may predispose to it. The disease is contagious, and the lesions inoculable and auto-inoculable.

The disease usually appears as several or more pin-head-sized papulo-vesicles or vesicles, most commonly upon the face and hands; these enlarge by peripheral extension, flatten out, and may become umbilicated. They may reach the size of a dime, or may become even larger. When several are close together, they may coalesce and form an irregularly-sized patch. In some cases distinct blebs result. In others the lesions are pustules from the start, somewhat rounded and with little tendency to flatten. As a rule, the lesions are without conspicuous or distinct areola. During several days or a week or more new lesions may continue to appear. Sooner or later they have all dried to thin, wafer-like, yellowish or yellowish-brown crusts, but slightly adherent, and having the appearance of being pasted or stuck on. Finally the crusts drop off, leaving slight or faint reddish or reddish-brown spots which gradually fade away. In some cases there are precursory symptoms of slight febrile action and malaise. Itching may or may not be present.

The disease is usually so striking in its symptomatology that a mistake in diagnosis is scarcely likely to occur. It is not to be confounded with pustular eczema or with ecthyma. The character of the lesions, their growth, and the lack of conspicuous infiltration or other inflammatory symptoms will serve to differentiate it. The disease usually runs its course in from one to several weeks. Exceptionally, from the constant scratching of the parts, new points are inoculated and the disease thus kept up.

The essential part of the treatment is to destroy the auto-inoculable properties of the lesions. This may be effected by removing the crusts by repeated warm water and soap washings, with the conjoint use of an application of ammoniated mercury ointment, from ten to twenty grains to the ounce. It is advisable, especially in extensive or itching cases, to apply, as a general application, a lotion of boric acid containing a half-drachm of carbolic acid or resorcin to the pint; this will serve to prevent the inoculation of new points.

LYMPHANGIECTODES.

This disease is also known as lymphangioma circumscriptum and lupus lymphaticus. It is a rare disease, characterized by pin-head to millet-seed-sized, deep-seated vesicles. The lesions are connected with the lymphatics, somewhat of the nature of lymphatic dilatations. One patch, generally from one to two or three inches in area, is usually observed, and on almost any part of the body. The lesions are closely crowded; the area is apt to be broken up into irregular groupings. The vesicles are wart-like in appearance and deep-seated, either without color or of a faint pink or light straw color. In some of the vesicles the overlying skin shows vascular tufts or striæ. It is unaccompanied by any inflammatory symptoms. Its progress is extremely slow. The disease begins in early childhood or infancy. In several cases venous nævus has been associated. Spontaneous disappearance, so far as the records show, has not been noted. Treatment consists in operative measures and in the use of caustics; recurrence has frequently followed. Electrolysis is advisable, if any treatment is deemed necessary.

MILIARIA.

This disease is also variously designated prickly heat, heat rash, lichen tropicus, red gum, strophulus, miliaria rubra, miliaria alba.

Excessive heat is the potential cause, so that the disease is commonly seen in torrid weather. Too much clothing is also provocative. A debilitated condition has a predisposing influence. The lesion is usually formed by the sweat-duct becoming obstructed, the sweat-secretion breaking through it and forming a vesicle between the deeper corneous layers.

The eruption consists of pin-point- to millet-seed-sized elevations, which may be papular, vesico-papular, or vesicular. Usually the eruption is mixed. The lesions are discrete, but are apt to be numerous and closely crowded, involving one or more regions or the entire surface. The trunk, neck, and genito-crural regions are favorite localities. The color may be pinkish or reddish,—miliaria rubra; later the areolar redness fades, the vesicular contents become somewhat opaque and yellowish white,—miliaria alba. It is not unusual in long-continued cases, especially in children, for boils to develop, and in neglected cases a veritable eczema may result.

Miliaria is to be distinguished from eczema and from sudamina. The lesions are smaller, show no tendency to rupture, there is no underlying thickening of the skin, and very little, if any, tendency to become confluent, characters which serve to differentiate it from eczema. There are no inflammatory symptoms in sudamina, and this latter is usually seen in connection with some low fever or acute rheumatism.

In the management of the case the cause of the disease—excessive heat—is to be removed if possible. Too much clothing is to be avoided. An application of a cooling and slightly astringent lotion or a mild dusting powder will be found to exert a favorable influence. A lotion of saturated solution of boric acid, with from two to four drachms of calamine and zinc oxide and from ten to twenty grains of carbolic acid to the pint, is one of the most useful; to make it more cooling, from a half-ounce to two ounces of alcohol may be added. Simple cooling lotions, such as equal parts of alcohol and water or vinegar and water, will often be sufficient. Of the dusting powders, boric acid, starch, lycopodium, talc, and zinc oxide may be prescribed, individually or in combination.

PITYRIASIS ROSEA.

The disease is also known under the names of pityriasis maculata et circinata and herpes tonsurans maculosus. It is a disease of a mildly inflammatory nature, usually seated upon the upper trunk. It has, however, been observed on the neck, invading the lower face, on the arms, especially in the flexor of the elbow, and about the genito-crural and popliteal regions. It is characterized by discrete and confluent pinkish or rosy-red or salmon-tinted, variously-sized, raised, scaly macules. The scaliness is apt to be more or less branny in nature, although in some instances it is quite flaky. There is rarely any infiltration of the underlying skin, and the inflammatory symptoms are not striking. The eruption appears, as a rule, rapidly, reaching its full development in one or two weeks, and gradually subsiding and disappearing in from four to twelve weeks. Exceptionally it is somewhat persistent. It is variously viewed as of parasitic nature, and as being a mildly inflammatory affection allied to psoriasis. It is also looked upon as analogous to seborrhœic eczema. The eruption resembles, to some extent, ringworm, mild psoriasis, and the squamous syphiloderm.

Treatment is purely expectant or symptomatic. Mild ointments of sulphur, from twenty to sixty grains to the ounce, together with an occasional saline aperient and tonic treatment, if indicated, seem to exert a favorable influence.

VERRUCA.

Verruca, or wart, is not an uncommon occurrence in childhood. It may be rounded, flat, or filiform. The ordinary flat variety is most common. It is usually rounded at the base, hard, and somewhat horny, sometimes with a digitate or papillary surface. There is no limit to the number that may be present; as a rule, there are from several to twenty or more. The cause of warts is not positively known. There is, however, a growing belief in its parasitic, and therefore contagious, nature. It is essentially a circumscribed hypertrophy of the epidermic and papillary layers of the skin, the interior of the growth containing the loop of a blood-vessel.

There are several means of treating these blemishes. Of the mild and doubtful methods may be mentioned sulphur ointment, white precipitate ointment, and a saturated solution of boric acid. If the lesions are small and numerous and close together, one of these drugs might be first tried. Another plan of treatment is by means of caustics. The application of salicylic acid—a saturated solution in alcohol—or salicylic acid in collodion, from thirty to sixty grains to the ounce, is useful at times; also paring off the hardened or horny top and cauterizing with the stick of silver nitrate. Touching with a solution of chromic acid, one to five or ten parts of water, is a more active remedy. Any of the other well-known caustics may be used. Excision with the curved scissors is the quickest method.

XERODERMA PIGMENTOSUM.

This rare disease is also designated parchment skin and angioma pigmentosum et atrophicum. The beginning symptoms consist of the appearance of numerous freckle-like spots and telangiectases. Later atrophic changes in the muscles and more or less shrinking and contraction of the integument are observed. Still later in the course of the disease, and exceptionally at an earlier period, epitheliomatous tumors and ulcerations take place. Death, usually after years, finally results. The true nature of the disease is not known. It begins in early childhood and is extremely slow in its course. It has been observed to occur in several children in the same family. Treatment is essentially palliative and expectant. Tonics and alteratives and nutrients are to be prescribed if indicated. Mild antiseptic protective applications are to be made to the ulcerated surfaces or tumors. Ablation of the tumors may be practised.

CONGENITAL SKIN DISEASES.

BY J. W. BALLANTYNE, M.D., F.R.C.P.E., F.R.S.E.

INTRODUCTION.

THE group of morbid conditions to which the name "congenital skin diseases" (or, shortly, "congenital skins") has been given may be regarded as including only those states which are commonly looked upon as cutaneous *deformities*, as faults in the embryology of the skin and subcutaneous tissue, or as embracing also the cutaneous *diseases* which may affect the fœtus and be present at birth. It is in this second and wider sense that I have accepted the designation and have constructed a classification to meet its requirements.[1] I have done so for various reasons, but chiefly because it is in the present state of our knowledge practically impossible to draw a hard and fast line between the deformities properly so called and the diseases of the skin. Theoretically it may be affirmed that the deformities are produced by morbid causes (whose nature can only be guessed at) acting during the *embryonic* stage of intra-uterine life and producing lesions which differ in many respects from those developed in later life; the diseases, on the other hand, are due to causes probably similar to those active in extra-uterine existence which affect the organism in the *fœtal* period of its antenatal life and lead to disorders closely resembling the dermatoses of the adult or child. Practically, however, it is very difficult to say in which of the two groups certain cutaneous lesions ought to be placed. Light will probably be thrown upon this subject from two directions: from the investigation of the normal development of the skin, and especially of its epitrichial layer, and from the careful examination of skin diseases in the early stages of fœtal life. Here it may be remarked parenthetically that it is astonishing how few cases of skin abnormalities in fœtuses younger than the sixth month have been put on record.

The term "congenital" has been applied somewhat loosely to some skin diseases which are not present at birth, but which are apparently predisposed to by some antenatal peculiarity existing in the cutaneous structures. An example of such may be present in the *xeroderma pigmentosum* of Kaposi. It is permissible to think that something in the intra-uterine environment may be the means of delaying the appearance of the typical manifestations of the disease, or that something in the new surroundings of the infant after birth may hasten their development. Possibly, also, both these factors may be at work. With regard, however, to such "congenital" skin affections little need here be said; all the space at my disposal must be given to the cutaneous lesions which are truly congenital,—*i.e.*, are present at birth.

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[1] Ballantyne, J. W., Diseases of the Fœtus, 1895, vol. ii. p. 227.

CLASSIFICATION.

Congenital cutaneous affections may be provisionally grouped in three divisions: (1) the transmitted diseases, such as the eruptions of the fevers and of syphilis; (2) the idiopathic morbid states which, so far as we at present know, take their origin in the fœtus itself and are not transmitted to it by the mother, examples of which are found in fœtal ichthyosis, hypertrichosis, etc.; and (3) the cutaneous neoplasms, the desmoses, such as elephantiasis congenita and some of the nævi. Elsewhere[1] I have considerably elaborated this classification, but for my present purpose the arrangement given above will serve.

TRANSMITTED DISEASES.

Exanthemata.—When during pregnancy a woman is attacked by one of the eruptive fevers her fœtus may when expelled from the uterus exhibit the same cutaneous lesions as those seen on her own body. Further, a child may at the time of birth be affected with one of the exanthemata, and yet the mother may, through immunity acquired from a previous attack, show no sign thereof; there may, however, be a history of exposure to infection. The exanthema whose characters when present at birth have been most fully investigated is *small-pox*. The variolous eruption may, as at other times in life, be discrete or confluent, and it may show itself in the papular, pustular, or cicatricial form, according as the disease has existed a shorter or a longer time before birth; but the cutaneous lesions have certain peculiarities which are doubtless due to their evolution in a warm fluid medium—the liquor amnii—and away from the effects of light. The face is frequently almost entirely spared by the eruption; the pustules resemble rather those that form on a moist mucous surface,—*e.g.*, the buccal in the adult,—there is a less degree of suppuration, crusts are seldom formed, and the resulting cicatrices are feebly marked. These characters all find an explanation in the fact that the skin of the variolous fœtus is bathed in the liquor amnii and protected from friction and the atmospheric air.

The eruptions of *measles*, *scarlet fever*, and *varicella* may also be present at birth; but their occurrence has been rarely noted, probably in part because the desquamation and erythema which accompany the first two n [illegible] ve been regarded as the normal cutaneous changes which happen in [illegible] rn infant. I have had opportunities of watching both these ex- [illegible] as they appear at birth, and was struck by the degree of involvement of the dermis in the case of measles, and by the close resemblance of the scarlatinal rash to the physiological erythema of the new-born. In fact, in the case of scarlet fever in the infant at birth the skin condition is not in itself sufficient for diagnosis. It would seem that the stage reached by the eruption is the same in mother and infant; at any rate, an examination of the data in the recorded cases apparently warrants this conclusion; this fact may be explained by simultaneous infection of mother and fœtus, or by a difference in the length of the incubation period in the latter. The skin changes in fœtal *typhoid fever* have not been much investigated.

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[1] Op cit., p. 227.

Erysipelas and Sepsis.—The passage of streptococci from mother to fœtus has been shown to be an occasional occurrence, and the fœtal erysipelas or sepsis thus induced may be accompanied by certain cutaneous changes. Thus, a *lamellar exfoliation* of the cuticle in the infants of women suffering from erysipelas has been observed by Kaltenbach, Runge, and others, and a noteworthy fact was the slight degree in which the skin of the face and limbs was involved. Septic conditions also may arise *in utero* and be accompanied by cutaneous changes evident at birth or very soon thereafter; among the changes thus produced may be mentioned *pemphigoid eruptions, jaundice, petechiæ,* and *purpuric spots,* and possibly also *dermatitis exfoliativa neonatorum,* which, according to Ritter, who first described it, is a pyæmic manifestation. It is worthy of note that Moncorvo[1] found the streptococci of Fehleisen in the blood of an infant with *congenital elephantiasis* whose mother had suffered from lymphangitis in pregnancy; he believed that the elephantiatic new formation was due to a local lymphangitic process set up in fœtal life by streptococci coming from the maternal circulation.

Syphilis.—The eruptions which may affect an infant suffering from congenital syphilis, and which appear soon after birth, have been dealt with by Dr. Abner Post[2] elsewhere; but there are certain less widely known but more truly congenital cutaneous manifestations which are sometimes seen in the fœtus. A *pseudo-ichthyosis* has, for instance, been met with at birth in undoubtedly syphilitic babies. The lesion differs from that of true fœtal ichthyosis in the character of the desquamation, which is moist rather than dry, and in the thinness of the epidermic plates. Closely allied to this manifestation is that which I have placed under the general designation of *fœtal keratolysis.*[3] That this abnormal looseness of attachment of the epidermis in the living fœtus is, in some cases at least, syphilitic must be regarded as certain. It is not to be confounded with the post-mortem changes in the cuticle which are so evident in the macerated fœtus. Sometimes the syphilitic taint shows itself at birth in a simple *erythema,* which, if it exist in association with keratolysis, increases the resemblance which the condition bears to maceration. Finally, the cutaneous change may take the form of *pemphigus*. There is a congenital pemphigus which is not specific, but in the syphilitic form the bullæ are soft, contain a yellowish or greenish pus, and affect specially the palms and the soles. During the act of birth many of the blebs may burst and leave raw surfaces or areas to which the cuticle is but loosely attached. It is possible that some of the appearances of maceration in living infants are thus derived.

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[1] Moncorvo, Congenital Elephantiasis, Edinburgh Medical Journal, 1896, vol. xli. p. 761.

[2] This Cyclopædia, 1889, vol. ii. p. 202.

[3] Ballantyne, J. W., Diseases of the Fœtus, 1895, vol. ii. p. 188.

IDIOPATHIC DISEASES.

In this group must be placed certain skin affections which, so far as is at present known, originate in the fœtus independently of the state of the mother in pregnancy. Doubtless further discoveries may diminish the number of diseases thus classified, leading to a transference of some of them to the group of the transmitted maladies; in the mean time they must be provisionally regarded as idiopathic. Some of the most interesting of all congenital skin affections are met with in the first division of this group, that, namely, which contains the epidermidoses, and specially the keratoses, and these I shall now proceed to consider.

Fœtal Ichthyosis (*Grave Type*).—Fœtal or congenital ichthyosis of the grave type may be regarded as essentially a hyperkeratosis, developed probably about the fourth month of intra-uterine life, and characterized by the existence over the whole surface of the body of horny epidermic plates, separated from each other by fissures and furrows. (*Vide* Plates I. and II.) The condition of the skin is associated with deformities of the mouth, nose, eyes, ears, and limbs, which are largely of the nature of arrested growth and (in the case of the mouth and eyes) of ectropion. The peculiar appearance of the infant suffering from this disease has gained for him the name "Harlequin Fœtus." The parents of such infants are generally free from skin disease of any kind; but a noteworthy fact is the occurrence of more than one case in the same family. In a case reported by Oestreicher[3] the mother had three healthy infants by her first husband and three ichthyotic fœtuses by her second, so that a paternal influence must sometimes be taken into account. The infants are weakly at birth, and in all the recorded cases death took place a few days after birth. The state of the mouth interferes much with suckling. The essential feature in the cutaneous lesion is an enormous hypertrophy of the stratum corneum of the epidermis. Whether or not it is truly an ichthyosis must be left to skilled dermatologists to settle, but that it is connected in some way with an anomalous development of the fœtal epitrichium[16] is fairly well established. No treatment has yet proved successful, but the two chief therapeutic indications must be the softening and removal of the epidermic covering and the maintenance of the infant's strength.

Fœtal Ichthyosis (*Mild Type*).—An infant affected with the mild type of fœtal ichthyosis is born with a continuous layer of a collodion-like substance over the whole body; later this substance desquamates in small

PLATE I.

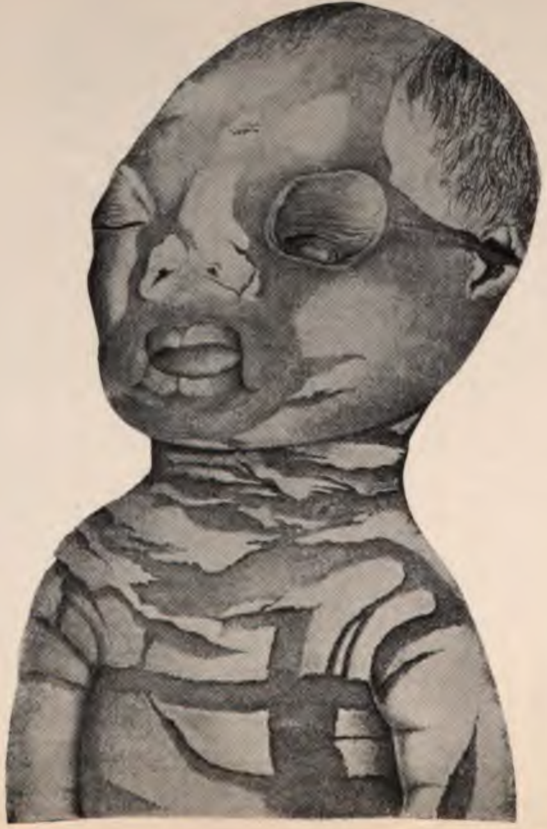
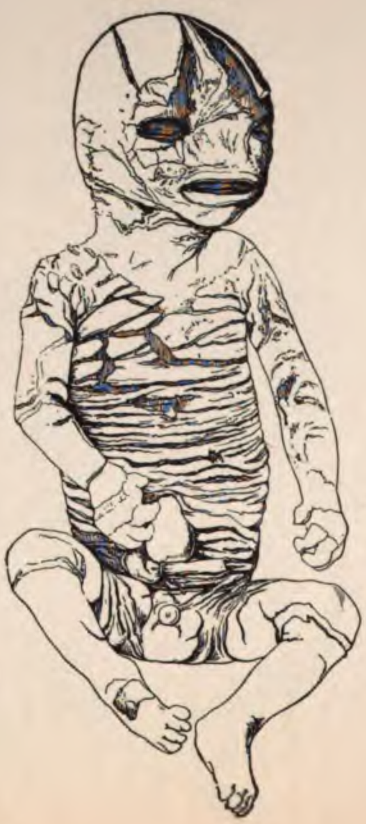


PLATE II.



Fœtal ichthyosis.

PLATE III.

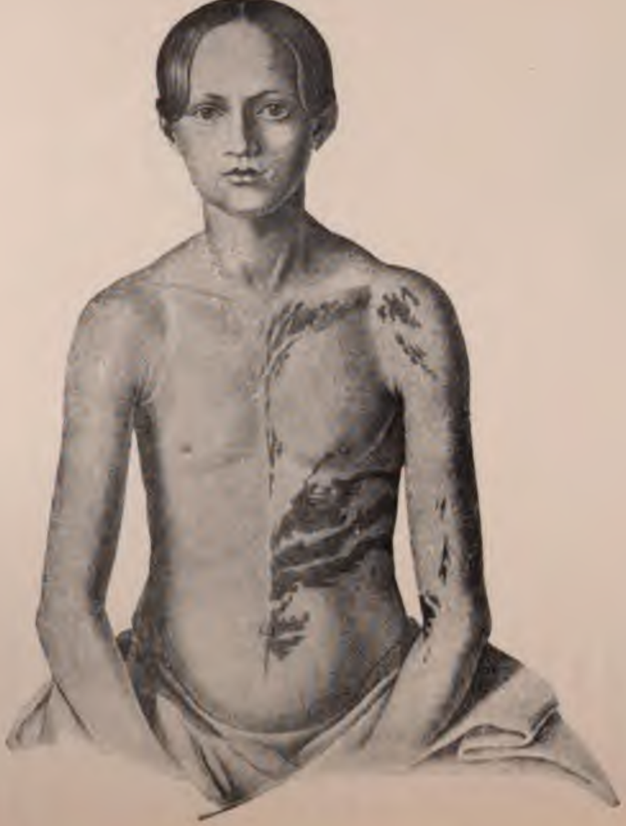
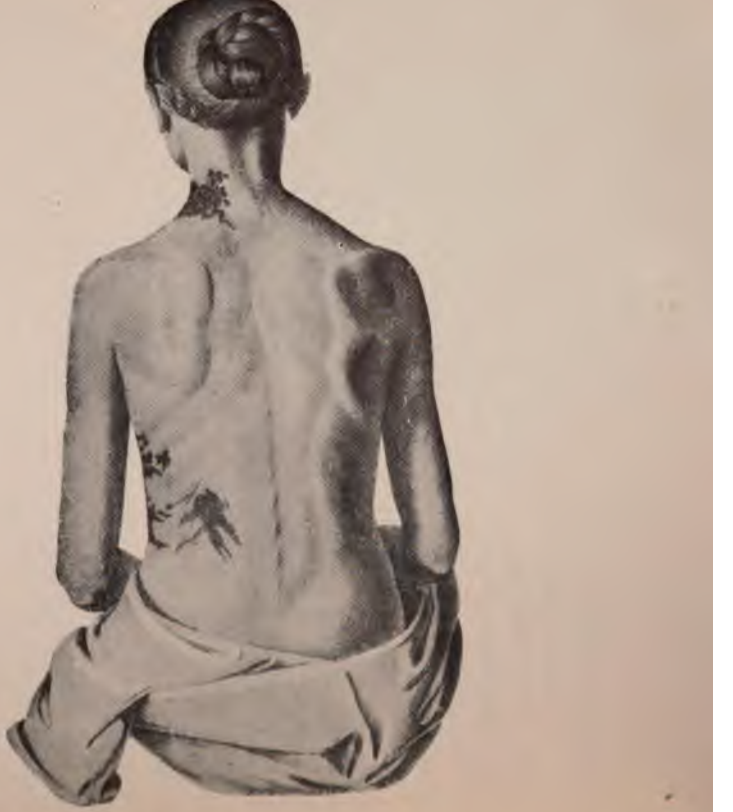


PLATE IV.



Nerve-nævus.

tissue-paper-like flakes. It is sometimes accompanied by an ectropion condition of the mouth, eyes, and anus. To it such names as "collodion fœtus" and "alligator infant" have been given. Family prevalence is the only indication of heredity in the history of the reported cases of this malady. It is not fatal to life; in fact, most of the patients reached adult life, and one was sixty-one years old when reported upon. In one or two instances there was recovery from the disease, in others there was a localized involution of the malady with a tendency to revert, but in most of the cases the lesion either remained *in statu quo* (as adult ichthyosis or xeroderma) or showed an increase in severity with advancing age. In nature the disease is a hyperkeratosis due probably to a permanence of the epitrichial layer,[16] but the degree of hyperplasia of the horny part of the epidermis is less than in the grave type of fœtal ichthyosis. The internal administration of such medicines as mercury, cod-liver oil, alkalies, and iodide of iron has not proved of much service; but external therapeutic measures, such as warm alkaline baths, friction with glycerin or lanolin, or the application of salicylic acid as a plaster, have mitigated the severity of the cutaneous lesions to some extent.

Congenital Ichthyosis Hystrix.—A few cases are on record in which there was a wide-spread development of spines upon the surface of the body at the time of birth, but more commonly this major degree of ichthyosis hystrix does not appear till some weeks after birth, as was the case in the famous Lambert family of "Porcupine Men." It is possible, however, that even in these apparently non-congenital cases a careful examination of the skin at the time of birth would have shown some anomaly; in fact, Crocker has recorded a case in which bruised places and raw-looking areas were then visible, and I have seen a female patient whose parents stated that on the second day of life numerous blisters developed in the regions ultimately affected by the ichthyosis.

Ichthyosis Linearis Neuropathica.—Under the names of *nerve-nævus, papillary nævus, nævus neuroticus unius lateris, papilloma neuroticum, nævus verrucosus unius lateris, nævus linearis ichthyosiformis*, and *ichthyosis herpetiformis* has been described a skin disease, generally present at birth, which may be regarded as a localized form of congenital ichthyosis hystrix. It is characterized by the presence of papillary growths, more or less pigmented, usually limited very exactly to one side of the body, and nearly always following the distribution of one or more of the cutaneous nerves. (*Vide* Plates III. and IV.) I have found no record of direct heredity or even of family prevalence in connection with this disease. The general health is little affected by the cutaneous lesion, but the occasional association of nervous troubles, such as epilepsy, is noteworthy. It has been regarded as due to intra-uterine nerve disease, and Hutchinson has even supposed it to be the result of fœtal herpes zoster. I am inclined to look upon it as an intermediate type between ichthyosis hystrix and congenital verrucæ or warty nævi. Treatment has usually taken the form of destruction or removal of the warty growths by nitric acid or the knife, but division of the nerve supplying the affected area has, in the hands of Wherry, given a more permanent cure.

Tylosis Palmæ et Plantæ.—Tylosis of the palms and soles, also known as *ichthyosis palmaris et plantaris*, *keratoma plantare et palmare hereditarium*, and *inherited keratosis of the palms and soles*, may be defined as an hypertrophy of the horny layer of the epidermis affecting only the limited areas above mentioned, and leading to the development there of a more or less hard plate of tissue. It is commonly congenital in, at any rate, the wide sense of the word, and the most striking feature in the history of such cases is the existence of heredity. In an instance seen by Dr. G. Elder and myself,[47] a little girl, her mother, her aunt, her great-grandmother, and her great-grand-aunt were all affected, although the grandmother escaped, and in other cases even more wonderful genealogies have been reported. In a second family, under the care of Dr. Ronaldson, of Haddington, I found several of the children affected, but the parents free from the disorder,—*i.e.*, family prevalence. The condition seems first to become evident when pressure and friction act on the palms and soles. The thickened epidermis gives rise to no pain, but sensibility is diminished; desquamation occurs usually once or twice a year. Whether the lesion is a hyperkeratosis or a hyperacanthosis may be left at present unsettled; that it may be caused by a localized persistence of the epitrichium is quite possible, but not proved. Glycerin to soften and pumice-stone to remove the hardened cuticle have been the usual therapeutic measures; but Dr. Elder and I, in the above-named case, obtained very distinct improvement from painting the parts with a solution of salicylic acid in ether (five per cent.).

Fœtal Keratolysis.—I have given the name *fœtal keratolysis* to a group of congenital cutaneous affections characterized by an abnormal loosening or actual separation of the cuticle. Doubtless, as has been already stated, in many of these cases we have to deal with the result of fœtal scarlatina, measles, erysipelas, and syphilis; but there remain a number of instances in which there is no evidence of the existence of any of these causal factors, and in which it must for the present be conceded that the exfoliation of the cuticle is idiopathic in origin. It may then be due to anasarca, to a nutritional disturbance of the cutaneous tissues, or to a prolongation of pregnancy which results in the birth of an over-ripe fœtus showing the desquamation which naturally only occurs some days after birth. It may also be that some cases are truly examples of the mildest type of fœtal ichthyosis. It is important to bear this condition in mind and to distinguish between it and the macerative changes met with in the *dead* fœtus, for many works regard desquamation of the cuticle in the new-born as a clear indication of intra-uterine death.

Congenital Hypertrichosis.—Among the congenital trichoses (morbid conditions of the hair) *hypertrichosis* or abnormal hairiness, general or local, occupies a prominent place. About the middle of fœtal life the whole body is covered with a fine down, and this condition may persist as *hypertrichosis universalis*. Patients thus affected are known as "hairy men," "hommes-chiens," or "missing links;" and well-known examples are the Shwe-Maong family of Burmah, Julia Pastrana, the Russian "hairy men," and Krao. The condition is sometimes associated with defective development of the teeth, and sometimes with a premature awakening of sexual instincts. It has not yet been clearly shown whether the hair seen at birth is the persistent fœtal lanugo or a crop which has grown after the lanugo has fallen off. It is probably correct to regard the condition as one of delayed development, possibly atavistic. *Localized hirsuties* may be present at birth, and its commonest form is the well-known hairy nævus on the face, back, or loins; but an excessive hairiness is also commonly associated with such defects as spina bifida occulta, anencephaly, hemiatrophy,[61] and encephalocele. It has been observed also in a case of congenital prolapsus uteri.[66] Such cases of hairiness (both general and local) have often been ascribed to "maternal impressions." Treatment, save by electricity in the most localized forms, is seldom successful; but the condition is troublesome only on account of the deformity caused by it.

Congenital Hypotrichosis.—Congenital *alopecia* or *hypotrichosis* may, like the anomaly just described, be *general* or *local*. The former variety is commonly hereditary; the latter is not so, but most probably is due in some obscure fashion to local alterations in the nutrition of the skin, such as the antenatal spontaneous cure of a vascular nævus. Dental defects may occur in association with the want of the hair; but the whole subject of this cutaneous anomaly requires investigation.

Paratrichoses.—The presence of *moniliform* or *beaded hairs* is an anomaly which is probably very often truly congenital, although it may not be noticed till childhood. It is known as *monilethrix* or *trichorrhexis nodosa*. Another paratrichotic condition is the occurrence of a congenital patch of white hair somewhere on the scalp, on the brow most commonly, and less frequently over the temple or back of the head. This *canities congenita* shows a very evident hereditary tendency, cases having been reported in which it affected both males and females for four generations.

Miscellaneous Epidermidoses.—Among the congenital disorders affecting chiefly the epidermis there yet remain for short consideration the *chromatoses* or pigmentary anomalies and the malformations of the nails. Into the question of localized excess of pigment (*nævus pigmentosus*) I shall not enter, for that has been elsewhere dealt with in this Cyclopædia (vol. ii. p. 103). Congenital cases of wide-spread or universal *melanoderma* have been very rarely put on record, the oft-quoted instance of Hannah West having been reported by W. C. Wells near the beginning of the century. It would seem that such pigmentary anomalies are to be ascribed to intra-uterine disturbances in innervation. The absence of the cutaneous pigment is met with as a general condition in *albinism*, when the skin is of a pure white or pinkish color; the pigment is also absent from the hair, iris, and choroid. *Localized achromatosis* is seen specially in the dark races, in whom it occasionally appears as irregular white streaks or patches following not uncommonly the distribution of a cutaneous nerve, and being accompanied by a whitening of the hairs of the affected region. A unilateral (right-sided) case of this kind has recently been described by Jonathan Hutchinson; the subject was a Hindoo man.

The nails may show a congenital overgrowth or atrophy. Cases of excess of growth, especially when combined with curving of the nail (*onychogryposis*), are met with in association with congenital ichthyosis. Congenital absence (*hypo-onychosis*) is very rare, but has been met with in a brother and sister suffering also from congenital alopecia; in this case the nails were well grown at the age of seven years.

Functional Disturbances of the Skin-Glands.—An excessive or an insufficient secretion of sebum or of sweat may occur as congenital peculiarities. An unnatural dryness of the skin (*xeroderma congenita*) may be the mildest manifestation of an ichthyotic tendency; on the other hand, an excessive secretion of sebum (*seborrhœa congenita*) has been advanced by some dermatologists as the lesion of grave fœtal ichthyosis. The vernix caseosa may be regarded as a sort of physiological *hypersteatosis* of intra-uterine life. Its absence would seem to predispose to eczema and possibly to other skin affections in infancy; at any rate, the worst case of eczema in early infancy that I have seen occurred in an infant who at the time of birth was perfectly devoid of vernix caseosa. Little is known of congenital peculiarities in the secretion of sweat; but I am acquainted with the case of a young man who has always suffered from unilateral *hyperidrosis* which has not yielded to any form of treatment. *Comedo, milium,* and *acne* spots may be present at birth; and with regard to acne I have seen a case in which both mother and infant were thus affected.

Congenital (Non-Syphilitic) Pemphigus.—The name *congenital pemphigus* is sometimes given to those cases in which there is simply a prenatal and often hereditary predisposition to the formation of bullæ on the skin as a result of some slight irritation, but in a stricter sense it is applicable only to the instances of non-syphilitic bullous eruptions in the fœtus at the time of birth. In the latter group of cases the bullæ are rarely seen at birth, for the passage of the child through the maternal passages has led to their rupture; but the loosened cuticle or actual bare areas may mark the position which they occupied. There is also a pemphigus neonatorum which is probably one of the manifestations of infantile sepsis. (*Vide supra.*)

Atrophic Dermatoses.—Among the atrophic dermatoses evident at birth must be placed the so-called congenital *absence of the skin* and congenital *ulcers.* Both these states are doubtless due to the more or less marked adhesion and pressure of the amnion during fœtal life. Cases have been recently reported by Ahlfeld,[72] Hochstetter,[73] Matthes,[74] Wallé,[76] and Goldberger,[75] and in these the region affected was sometimes the scalp and sometimes the trunk. In an example seen by H. von Hebra in 1880 there was a symmetrical patch of defect, indicated by a bright red area, on each side of the head in the frontal region, and microscopic sections showed that the lesion was really an absence of the skin-layers in the region named. In Matthes's case also, and in that seen by Ahlfeld, the part affected was the scalp; but in Hochstetter's the defect took the form of a bright red triangular scar situated on each side of the abdomen a little above the level of the umbilicus. The possible medico-legal importance of such conditions cannot be overlooked, for in their appearance they closely simulate the results of traumatism.

Hypertrophic Dermatoses and Desmoses.—Cases, such as that recently reported by Reynolds Wilson, of Philadelphia,[78] which show more or less marked hypertrophy of the constituent parts of the skin are with difficulty separated from the neoplasms of the cutis. The conditions known as *congenital elephantiasis*, *cystic elephantiasis*, truly congenital *sclerema*, and *general obesity* of the fœtus may be placed by themselves, but it is almost impossible to pass over the resemblances which exist between them and the localized hypertrophies named *fibroma*, *myoma*, *lipoma*, and *adenoma cutis*, as well as certain varieties of so-called *congenital elephantiasis*. Further, the conditions named are affections of the subcutaneous tissue rather than of the skin. They have been described in an early volume (vol. ii.) of this Cyclopædia.

Atrophic Conditions of the Subcutaneous Tissue.—A few words may be said regarding the atrophic states of the subcutaneous tissues which are on rare occasions met with at birth. It is sometimes found that the infant of a woman who has suffered from some serious renal or cardiac condition in pregnancy shows an almost total absence of the subcutaneous fat, but this is often masked by a certain amount of œdema or anasarca. The anomaly may persist in adult life, as is seen in the individuals exhibited in shows under the name "Living Skeletons." In some of these persons, at any rate, the state would appear to have been antenatally acquired. A condition probably closely allied is that known as *cutis laxa*, without tumor formations or *dermatolysis* in the strict sense of the word. The abnormal extensibility of the skin in these cases has gained for the individuals thus affected the name "*Elastic-Skinned Men.*" "Pierre, der anatomische Wundermensch," afforded a typical instance of this deformity; he could draw out large folds of skin from his face, neck, chest, and limbs, yet the integument retained its normal color, temperature, and secretions. Seifert[79] has found in a case examined microscopically that along with complete atrophy of the connective tissue bundles of the skin there is a transformation of the firm tissue of the dermis into an unformed myxoma-like structure. I am inclined to regard the anomaly as due essentially to a weakening of the structural elements which normally bind the skin to the underlying structures, caused probably by some fault in nutrition occurring during fœtal life. It does not usually call for treatment, leading only to discomfort in grasping and walking.

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the condition bears to maceration. Finally, the cutaneous change may take the form of *pemphigus*. There is a congenital pemphigus which is not specific, but in the syphilitic form the bullæ are soft, contain a yellowish or greenish pus, and affect specially the palms and the soles. During the act of birth many of the blebs may burst and leave raw surfaces or areas to which the cuticle is but loosely attached. It is possible that some of the appearances of maceration in living infants are thus derived.

IDIOPATHIC DISEASES.

In this group must be placed certain skin affections which, so far as is at present known, originate in the fœtus independently of the state of the mother in pregnancy. Doubtless further discoveries may diminish the number of diseases thus classified, leading to a transference of some of them to the group of the transmitted maladies; in the mean time they must be provisionally regarded as idiopathic. Some of the most interesting of all congenital skin affections are met with in the first division of this group, that, namely, which contains the epidermidoses, and specially the keratoses, and these I shall now proceed to consider.

Fœtal Ichthyosis (*Grave Type*).—Fœtal or congenital ichthyosis of the grave type may be regarded as essentially a hyperkeratosis, developed probably about the fourth month of intra-uterine life, and characterized by the existence over the whole surface of the body of horny epidermic plates, separated from each other by fissures and furrows. (*Vide* Plates I. and II.) The condition of the skin is associated with deformities of the mouth, nose, eyes, ears, and limbs, which are largely of the nature of arrested growth and (in the case of the mouth and eyes) of ectropion. The peculiar appearance of the infant suffering from this disease has gained for him the name "Harlequin Fœtus." The parents of such infants are generally free from skin disease of any kind; but a noteworthy fact is the occurrence of more than one case in the same family. In a case reported by Oestreicher[3] the mother had three healthy infants by her first husband and three ichthyotic fœtuses by her second, so that a paternal influence must sometimes be taken into account. The infants are weakly at birth, and in all the recorded cases death took place a few days after birth. The state of the mouth interferes much with suckling. The essential feature in the cutaneous lesion is an enormous hypertrophy of the stratum corneum of the epidermis. Whether or not it is truly an ichthyosis must be left to skilled dermatologists to settle, but that it is connected in some way with an anomalous development of the fœtal epitrichium[16] is fairly well established. No treatment has yet proved successful, but the two chief therapeutic indications must be the softening and removal of the epidermic covering and the maintenance of the infant's strength.

Fœtal Ichthyosis (*Mild Type*).—An infant affected with the mild type of fœtal ichthyosis is born with a continuous layer of a collodion-like substance over the whole body; later this substance desquamates in small

PLATE I.

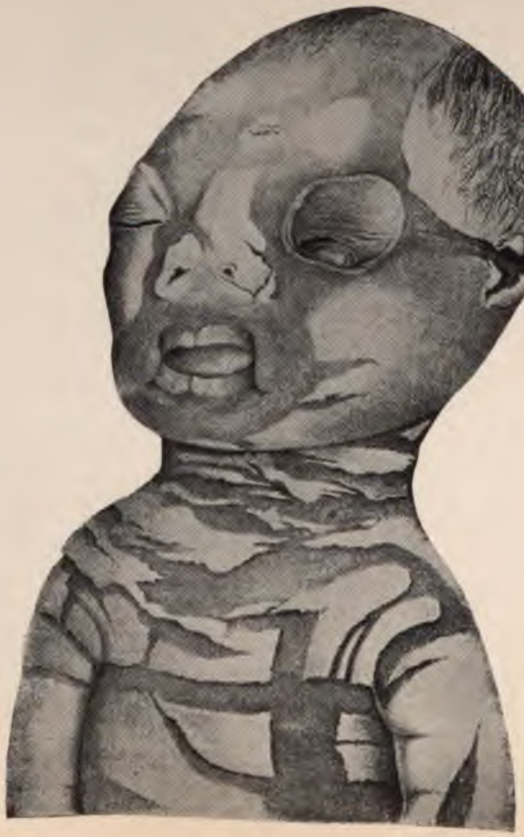
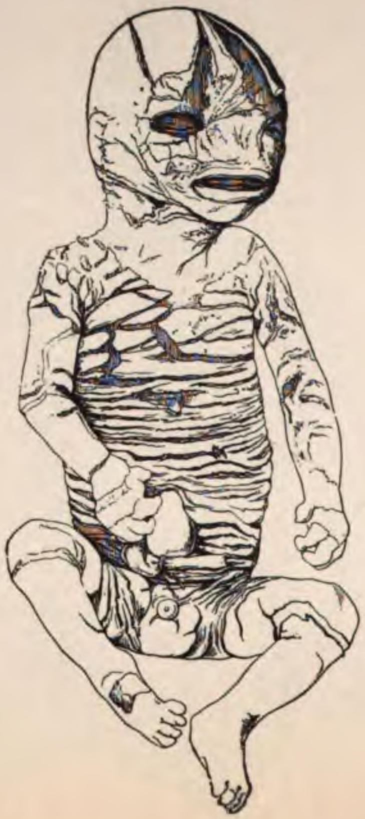


PLATE II.



Fœtal ichthyosis.

DISEASES OF THE EAR.[1]

By CHARLES H. BURNETT, M.D.

Piercing the Lobule.—Syphilis, lupus, and tubercle have been communicated by an infected needle used in piercing the lobule of the auricle preparatory to wearing ear-rings.

Examination of the Ear.—It is possible to examine the meatus, auditory canal, and membrana tympani without an ear-funnel. As the latter is likely to wound the skin of the meatus when employed by one unused to such examination, the general physician should learn to examine the ear,—*i.e.*, illuminate it with reflected light without a speculum or ear-funnel.

Vernix Caseosa.—The vernix caseosa and all secretions which naturally fill the external auditory canal of the new-born child should be let alone. They are placed there by nature, and in due time are extruded from the canal by the outward growth of the skin, covering the outer surface of the drum-head and walls of the auditory canal. Efforts at their artificial removal on the part of nurse, parent, or physician are always attended by irritation and more or less severe inflammation, even extending to the middle ear, as I have frequently observed.

Foreign Bodies in the Ear.—I am informed that in some of the London hospitals resident physicians are forbidden to attempt to remove foreign bodies from the ear, but are obliged to summon at once for such an interference the aurist of the institution. I have observed entire destruction of the membrana tympani from the unskilful attempts of a resident physician to remove a foreign body supposed to be in the ear of a child of eight years. The foreign body was never found, and I have every reason to believe there never was a foreign body in this child's ear. However, the drum-membrane was destroyed, chronic otorrhœa established, and the hearing permanently injured.

Paracentesis of the Membrana Tympani.—While paracentesis in acute otitis media must not be delayed beyond twelve hours, the surgeon should not be too swift to incise the drum-membrane. While the ear is still painful and discharge has not occurred, it is entirely rational to employ, in addition to dry heat about the ear, warm instillations of a watery solution of carbolic acid (1 to 40). These sometimes exercise an abortive effect. But after paracentesis, or after spontaneous rupture of the membrana, *nothing* should be put into the ear, for fear of irritation—secondary infection, in fact —of the perforated membrana and the inflamed drum-cavity beyond. Acute otitis media is due usually to the entrance of streptococci into the middle ear from the nares and Eustachian tube. This tends to run a benignant course terminating in a rupture of the drum-head and the escape of secretion containing the infective cocci. The discharge, therefore, is beneficent, and should not be checked any more than the resolution of a pneumonia with its expectoration should be checked. It is known, furthermore, that staphylococci, the factors in chronicity of aural discharge, are ever present in the outer ear. In the latter position they are harmless so long as the skin of the canal is intact or the membrana imperforate. After the membrana is open, the entrance of staphylococci into the middle ear through the perforation in the drum-head or in any other way will produce secondary infection of the drum-cavity and of the mastoid. Hence after the membrana is either artificially or spontaneously perforated, all syringings, moppings, probings, or insufflations tend to force staphylococci through the perforated membrana into the already inflamed middle ear. And in fact this is the way the sequelæ of acute otitis media occur, not as *necessary* results of the disease, but as *artificial* results of an irritating, irrational local treatment. The best time—the only proper time, in fact—to syringe the acutely inflamed ear with an antiseptic is before the membrana is perforated. This frees the outer ear of staphylococci, at least for a time, renders it temporarily aseptic, and when perforation of the membrana occurs, secondary infection is so much the less likely to ensue. If, in addition to such abstention from all local treatment as just named, the auditory canal be drained by inserting half-way down its calibre a strip of iodoform gauze (five per cent.) or carbolic acid gauze (five per cent.) one inch and a half long by a quarter of an inch wide, aseptic drainage will be effected. Under such treatment, in the worst cases, entire cure with return to good hearing may be confidently expected in two or three weeks. Of course, if, before this form of aseptic treatment, secondary infection has been caused by some form of so-called antiseptic treatment, much will have to be overcome before an aseptic treatment can be successfully applied.

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[1] This article is supplemental to that in vol. iv.

Inflation of the Naso-Pharynx and Tympana.—I feel it my duty to caution against the frequent and forcible inflation of the naso-pharynx and tympana in any form of ear disease, but especially in acute otitis media. If used at all, it must be with great gentleness, as a means of clearing the nares and naso-pharynx rather than of inflation of the tympana. The latter effect is to be deprecated in acute otitis media, because it is liable to force fresh germs into the already inflamed middle ear or pyogenic germs from the middle ear into the mastoid cavity, where there will be set up an acute mastoiditis. Furthermore, if inflation of the simultaneously diseased naso-pharynx is persevered in, pathogenic germs may be forced from the naso-pharynx into the unaffected ear and an otitis set up in it. For these reasons I have long abstained from using *any* form of inflation of the tympana in acute otitis media. Politzer's inflation, if employed at all, must be gently done, and only to clean the nostrils; it may with advantage be omitted entirely.

Acute Otitis Media.—Leeches or bloodletting in any form are not necessary in acute otitis media, and are used less and less frequently as our knowledge of the germ origin of otitis is increased. Paracentesis is the most important act the surgeon can perform and in many cases the greatest aid the patient can receive. After paracentesis is performed the ear should be drained, as stated in vol. iv. p. 22, and *nothing* should be dropped, mopped, or syringed into it. Pus may escape from the middle ear by the Eustachian tube, according to some authorities, but I have never seen it occur. In any event, the escape of pus from the middle ear by this pathway must not be depended upon. If spontaneous rupture does not occur in from six to twelve hours, and the pain continues, pus is being retained in the drum-cavity under tension, and in a child especially there is then danger of the passage of the pus from the tympanic space into the cranial cavity with fatal results.

Acute Purulent Otitis Media; Improper Treatment; Death in Three Weeks from Cerebellar Abscess.—The following case will show the dangers attending postponement of paracentesis and waiting for spontaneous rupture of the drum-membrane in acute otitis media. The patient, a little girl of eight years, was said to have had grippe without ear disease two months previous to the time I was called to see her. Three weeks before I examined the case she was allowed to go coasting. That night she complained of earache in her right ear, and a homœopathic physician was summoned. His treatment, unknown to me, failed to give relief; he certainly did not perform paracentesis of the membrana tympani. The child continued to suffer pain in the ear, night and day, for a week, when spontaneous rupture of the membrana occurred and the ear-pain ceased. But the child seemed dull and miserable, according to the parents' statement, and began to complain of frontal headache. She also became weak and began to emaciate and lose appetite. The bowels and kidneys were not deranged, but the little patient expressed unwillingness to move about, complained of headache, and finally went to bed voluntarily at the end of the second week of her suffering. From this time all the symptoms began to grow worse; emaciation, anorexia, and hebetude became marked. The child gradually passed into a stupor from which she could be aroused, but would cry at such times, as though it pained her to be disturbed or moved. The case was now abandoned by the homœopathic physician, and two regular physicians were called in by the parents of the child. A brain malady was diagnosticated by them. Twenty-four hours later I was asked to see the case as one of cerebral disease from otitis.

The child was found lying on her left side, her head thrown back and her legs drawn up. Her lips were parted, the lips and the tongue being covered with sordes. There was apparently no fever, the pulse was *slow*, and the child nearly unconscious. A little muco-pus came from her right ear. A perforation was detected in the upper anterior quadrant of the red and swollen membrana tympani by examination with the otoscope. An abscess of the brain was diagnosed, but its precise location was not determined before death.

At my suggestion, Dr. W. W. Keen was summoned to the case with a view of trephining for relief of the cerebral abscess, and such an operation was performed by him about sixteen hours after I first saw the child.

"Ether was given and an incision was first made over the mastoid and the mastoid antrum opened, followed by free gouging of the bone into the mastoid cells. These were found to be filled with cheesy pus, and the bone was found to be very soft. I then made an inch trephine opening, the centre pin being placed an inch and a quarter above and the same distance behind the external auditory meatus. As soon as the dura was exposed a Horsley's dural separator was passed downward and slightly forward until it struck the ridge between the anterior and posterior surfaces of the petrous bone. Then it was passed into both the anterior and middle fossa of the skull, separating the dura for some distance from the petrous bone. No pus was found in either place. The dura, which looked markedly yellow, was then incised, and the separator passed between the brain and the dura for some distance, the result being negative.

"As it was possible that there was an abscess in the temporo-sphenoidal lobe, though I deemed it unlikely by reason of the temperature (100.4° F.) and the normal pupils, I passed a grooved director first in the direction of the ala of the opposite side of the nose, and secondly in the direction of the opposite pupil. No pus was found by either puncture. I did not deem it wise, owing to the condition of the patient, to trephine over the cerebellum, though the question was carefully considered. The wounds were closed, leaving a slight opening for drainage from the brain, and a drainage-tube was inserted in the mastoid cells. The hemorrhage in separating the dura from the bone had been quite free, but was easily checked by packing. The child recovered well from the ether, and was none the worse for the operation, so far as I could judge. She gradually sank and died eleven hours after operation.

"Autopsy twelve hours later. Only the brain was examined. While removing the brain, an abscess in the right hemisphere of the cerebellum burst through the thin cortex that was left and discharged about two fluidounces of odorless pus.

"On the posterior surface of the petrous bone a communication existed with the middle ear. The tracks of the punctures into the temporo-sphenoidal lobe were perceptible as small lesions in cross-section. No bleeding or other damage had been caused.

"The only question to be considered is whether the cerebellum should have been punctured after failure to find the pus in the temporo-sphenoidal lobe. Looking back upon it after an interval, my decided conviction is that it was a mistake not to do so. The results would have been the same to life in this particular case, but surgically it was an error, and especially might prove such in some other case in which life might be saved."—*Extract from Dr. Keen's Notes.*

Dr. William J. Taylor, who made the post-mortem examination in this case, informed me more minutely as to the nature of the communication between the middle ear and the posterior surface of the petrous bone. It seemed like a widened vascular canal, not necrotic, and to its opening into the cranial cavity the approximating meningeal surface of the cerebellum was adherent. This, of course, makes plain the path of communication between the ear and the brain.

The lesson so plainly taught by this case is that an acute otitis media with earache and an imperforate membrana tympani should not be allowed to continue many hours without paracentesis, because the products of inflammation in the drum-cavity, being pent up under tension, not only cause great suffering, but may be forced into the mastoid cells or the cranial cavity, or into the vessels near it, and thus gain access to the brain-substance, where an abscess is formed, usually with the result observed in this case. This little child's life could have been saved by the judicious and skilful employment of a darning-needle, rendered aseptic, had no better instrument been at hand for piercing her membrana the night the earache set in.

Drainage in Chronic Purulent Otitis Media.—Sometimes in this disease the employment of drainage by means of a strip of gauze proves superior to syringing secretion from the ear.

Chromic Acid.—The end of the probe used for conveying chromic acid to granulations in the ear should be not more than a millimetre in diameter. Alcohol is nearly as good as chromic acid and much safer. The former should never be used by the inexpert.

Nitrate of Silver.—I have changed my views entirely respecting the instillation of silver nitrate, and employ it no more. Sometimes the hypertrophied and secreting surface of the tympanic mucous membrane may be mopped advantageously with strong solutions of silver nitrate, varying from fifty to one hundred grains to the fluidounce of water, or even stronger. It is not necessary to follow such treatment with an application of chloride of sodium. Citrate of silver, powdered, is much better and safer as an antiseptic.

Excision in Attic Cases.—In chronic purulency of the attic, or recessus epitympanicus, with intact membrana vibrans,—all that part below the folds of the membrana,—the latter need not be entirely excised, but only so much of it as will be necessary to release the malleus handle. The head of the hammer is always found to be necrotic in cases of chronic attic suppuration, and must be removed in the operation of necronectomy.

Hydrogen Dioxide.—I have found that hydrogen dioxide is too expansive in its action for safe using in the acutely inflamed middle ear. It is likely to drive inward quite as much pus as it brings out. That forced inward may reach the brain-cavity through the tegmen tympani, and, in fact, it does thus reach the mastoid cavity, where acute mastoiditis is set up.

Secondary Infection.—If the primary inflammation in the middle ear is not improperly treated, as detailed above, there will be no secondary infection. Much that was once thought to be the necessary result of acute otitis media is now known to be the result of bad treatment, by which staphylococci especially are forced into the middle ear, causing secondary infection of that space and sometimes of the adjacent cranial and mastoid spaces.

Chronic Hypertrophic Catarrh of the Middle Ear.—Anglo-Saxon children born in tropical countries are found to be liable to chronic catarrhal deafness. This is due to trophic disturbances in the middle and internal ears rather than to so-called colds in the head. In the treatment of chronic catarrhal otitis media, watery solutions in any form must be used with the greatest caution in the nares, and in many cases avoided, as they have a "water-logging" effect on the mucous membranes of the nares and naso-pharynx, thus practically increasing the hypertrophy and augmenting the deafness and tinnitus. Oleaginous solutions or suspensions are better in such cases for spraying, just as they are far superior to watery solutions as applications to the Eustachian tubes. For many years I have employed as the vehicle in nasal sprays and applications to the Eustachian tubes glycoline, a superior form of fluid petrolatum. The nares should be sprayed in chronic hypertrophic naso-pharyngeal catarrh two or three times a week at most. If inflation of the tympana is employed, it should be applied after the nares have been sprayed, and with great gentleness, as already stated. It is of slight value at best, as not inflation, but passive motion of the membrana and the ossicula is demanded in chronic aural catarrh. This can be exerted far better by the employment of Siegle's pneumatic speculum, inserted in the auditory meatus and operated by the surgeon under his own eye. I have known the hearing to be improved at once and the tinnitus quelled for long periods by using this little instrument, when ordinary inflation by Politzer's air-bag failed. Furthermore, the application of Siegle's pneumatic speculum is agreeable, whereas all forms of tympanic inflation are very disagreeable, and in children not easily carried out.

Atrophic Nasal Catarrh.—Atrophic nasal catarrh (ozæna), with concomitant tinnitus and impaired hearing, is observed sometimes in children. Abel[1] has described a non-fetid form of atrophic nasal catarrh, and I have observed two cases in a mother and her child of four years. The atrophy and the peculiar secretion were present in these cases, but fetor was absent.

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[1] See article by Gradenigo, Annales des Maladies de l'Oreille, August, 1896.

Abel maintains that the first two are essential characteristics, but that the fetor, strange as it may sound, is not essential in this malady. The absence of fetor in such cases becomes a clinical disadvantage to the patient, because the atrophic process may in consequence longer escape detection.

The treatment—a stimulating one—remains the same for any form of atrophic catarrh of the nose, naso-pharynx, and Eustachian tubes (vol. iv. p. 36). Such applications may be made to the affected parts once or twice a day by the patient, if old enough, or by a care-taker, if the patient be young. The stronger topical applications to the nares, naso-pharynx, and fauces, consisting of tinctures of sanguinaria and myrrh and alcoholic solutions of thymol, may be made several times a week by the physician if demanded by the symptoms.

Secondary infection can be avoided by the treatment laid down in vol. iv. p. 37, as it is practically what Gradenigo and Pes have since urged on bacteriological grounds.[1]

Warm aqueous solutions of carbolic acid (1 to 40) may be used to try to abort the process in the ear and to render aseptic the outer surface of the membrana tympani and the auditory canal. In this way secondary infection of the drum-cavity is much less likely to occur after paracentesis is performed or spontaneous rupture of the drum-membrane takes place.

Leeching and, in fact, all forms of bloodletting are valueless. They remove no pathogenic germs from the middle ear. They are disagreeable and more or less shocking to an adult. In the case of a child they are worse than useless, as they frighten the patient. In acute otitis media in a child, or in any one, first apply *dry* heat about the external ear. Instillations of warm water and of warm watery solutions of carbolic acid (1 to 40), as given above, may be tried in the intervals between the applications of dry heat, if they can be endured. My experience is that in acute otitis media the simultaneously congested and inflamed membrana tympani cannot endure the pressure of the column of warm fluid placed in the auditory canal. I have always found that the earache was rendered worse by such treatment. After discharge sets in, of course nothing must be put into the ear, for fear of secondary infection of the drum-cavity, as already stated (vol. iv. p. 37). If dry heat, or heat in any form, applied to the ear fails to give relief to the pain in the ear in six hours, or at most twelve hours, and spontaneous rupture of the membrana does not occur, paracentesis of the drum-membrane is demanded in order to save not only the hearing, but the life of the child (vol. iv. p. 36). Earache which has lasted several hours does not always cease immediately after spontaneous rupture or paracentesis. Amelioration of the symptoms, however, usually sets in at once. When paracentesis has been deferred for a week or ten days, as I have known it to be both in children and in adults, the pain may continue for a day or two after the operation and the prompt escape of pus. Such phenomena of pain indicate a deep invasion of the muco-periosteal lining of the middle ear cavities, and must be carefully watched.

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[1] Archiv für Ohrenheilkunde, 1894.

After paracentesis or spontaneous rupture of the membrana, the auditory canal may be drained by the insertion of a short strip of antiseptic gauze (vol. iv. pp. 36, 37), or the meatus may be simply gently stopped with a ball of such gauze and nothing more done. An experienced eye may see in such cases opportunities for gentle local mopping of the canal-walls and the fundus of the auditory canal with mild antiseptics; but the greatest caution is always necessary, even on the part of the most skilful hand, not to irritate the acutely inflamed ear and provoke secondary infection. I see every day cases of secondary infection of acute otitis media in children and in adults induced by improper treatment in the primary acute stages. Sometimes this improper and infectious treatment is applied by the patient or his friends, and, I regret to say, sometimes by his physician.

Instruction of the Mute Child.—Much more is now done in the instruction of the mute, the stammerer, and the stutterer than when the first edition of this work was published. Carefully conducted schools for the instruction of the partially and totally mute child in lip-reading and articulation, like that of Miss Garrett at Bala, Philadelphia, are more numerous.

The partially mute as well as the totally mute child, the stuttering and stammering child as well as the child with pathological speech-defects, is taught lip-reading and articulation, and also "expression-reading," as set forth in a little work by Miss Lillie Eginton Warren, of New York.[1] Speech-defects in children especially are now studied and treated in special departments in medical colleges and hospitals by those devoting themselves to the treatment of such maladies, as in the Philadelphia Polyclinic, by Dr. G. Hudson Makuen, lecturer on speech-defects in that institution.[2]

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[1] Edgar S. Werner, New York, 1895.

[2] See Therapeutic Gazette, December 15, 1896.

DISEASES OF THE EYELIDS, OF THE EXTERNAL TUNICS OF THE EYE, AND OF THE IRIS AND THE CILIARY BODY.

By G. E. de SCHWEINITZ, A.M., M.D.

The following pages are devoted to a brief review of certain diseases of the eyes which the general practitioner of medicine and surgery may study without the use of instruments of precision,—for example, the ophthalmoscope. It is understood that these pages are to be read in connection with the previous articles on the eye, Part II., vol. iv., and that they constitute in part an addendum and in part a review.

BLEPHARITIS.

Although blepharitis is a general term which describes the various types of acute and chronic inflammation of the border of the lid, it includes a variety of affections of diverse origin. The recognition of the proper etiological factor in each case is important in order to apply proper remedies at an early stage of this troublesome affection, and before its progress has induced organic changes in the margin of the lid which practically resist all treatment. The affection may be considered etiologically under the following subdivisions:

(1) *A hyperæmia of the lid-border* in which there is simply a fringe of passive congestion, without the formation of crusts,—the so-called vasomotor blepharitis.

(2) *Simple or squamous blepharitis*, which depends upon an abnormal secretion of the sebaceous glands, and is really a seborrhœa associated with the formation of scales and crusts on the margins of the lids, precisely as when this disease attacks other areas,—for example, the eyebrows or the margin of the scalp.

(3) *Eczema of the border of the lid*, which may be (*a*) superficial, or a simple marginal eczema; (*b*) solitary, and associated with an adenitis of the glands of the lid-margin; and (*c*) pustular, or accompanied by various types of small ulcers covered with thick crusts and leading to the destructive processes already described (vol. iv. p. 52).

(4) A final type is sometimes seen in association with acne, and consists in a deposit of acne pimples on the margin of the lid.

Treatment.—With these etiological considerations in view, the treatment should consist, in the first or vaso-motor variety, of local measures to reduce the hyperæmia, and hence irritating salves are contra-indicated. Koenigstein's douche (vol. iv. p. 52) is suitable, and the edge of the lid may be nightly smeared with the ointment recommended by W. Allen Jamieson, which is composed of lanolin, three drachms; oil of sweet almond, one-half drachm; and distilled water, one-half drachm.

In the seborrhœic varieties, after removal of the crusts with an alkaline solution, a three per cent. milk of sulphur salve, with or without the addition of resorcin (three per cent.), is advisable.

In the eczematous varieties the ordinary local applications for this affection are required, and among these the most suitable are diachylon ointment, boric acid ointment, aristol ointment, and particularly the ointment of the yellow oxide of mercury, one grain to the drachm of either simple vaseline or of Jamieson's ointment.

In unfavorable cases associated with much ulceration, touching the ulcers with nitrate of silver, ten grains to the ounce, or iodine, or, according to Despagnet, with a mixture of corrosive sublimate in glycerin (1 to 100), is advisable, care being taken that these strong solutions do not come in contact with the cornea. If with this affection there is associated seborrhœa of the face or eczema or acne of the same region, it is useless to try to cure the lids until the more extensive lesions of the face have been remedied. The methods of dealing with misplaced cilia and other similar procedures have been already fully described. Constitutional treatment is not to be neglected, especially the remedies which overcome anæmia and struma, and the proper examination and treatment of the naso-pharynx, which nearly always is inflamed, are of paramount importance.

It is presupposed that in each case of blepharitis of suitable age any anomaly of the refraction of the eye has been corrected. Many of the milder forms are caused by refractive errors, and even in the stubborn varieties suitable glasses materially aid in the cure.

Parasitic Blepharitis.—This may be caused by the presence of the trichophyton fungus in the margin of the lid, the so-called *blepharitis trichophytica;* or by the pediculus pubis, the so-called *blepharitis pediculosa,* or *phthiriasis;* and, finally, by the presence of the *demodex folliculorum* in the hair-follicles of the eyelids, when the affection may resemble a blepharo-adenitis. Of these three parasitic affections one only is of importance,—namely, phthiriasis, which is much more common than the books would lead one to suppose.[1] The parasites and their eggs may be removed by an application of blue ointment, or by careful pencilling with a strong bichloride of mercury solution.

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[1] Consult article by Dr. P. N. K. Schwenk, Wills Hospital Reports, vol. i., No. 11, 1895.

Tarsitis, or a chronic (rarely acute) inflammation of the tarsus, may appear as an idiopathic affection, and closely resembles a chronic blepharitis. At other times blepharitis appears to be associated with a thickening of the tarsus, the so-called *hypertrophic blepharitis*.

Most frequently, however, tarsitis is syphilitic in origin, and consists of a gummatous infiltration of this structure. It occurs in children who are the subjects of the inherited form of this disease. Under these circumstances, local measures—that is, stimulating and resolvent ointments; for example, the yellow oxide of mercury—will not suffice, but the constitutional treatment of syphilis, particularly inunctions of unguentum hydrargyrum, is required.

Tumors and Hypertrophies.—Among affections belonging to this class attention is especially directed once more to *nævi*, which so often appear as small red spots upon the eyelids, and which, if allowed to grow, may spread in a most disfiguring manner into the orbit, or upon the forehead or the face. It is the simplest matter to destroy the angioma, when it is of small size, by touching it with a drop of nitric acid, and physicians should never fail to warn the parents of children that a small red spot of the character described may grow extensively. Larger growths may be excised or obliterated by the method of *electrolysis*.

Inflammation of the Meibomian gland resulting in *chalazion* is not nearly so common an affection in children as in adults. When it occurs, the ordinary treatment of evacuation through the conjunctival surface and curetting the sac, or in external cases removal through the skin-surface, may be practised. It should be remembered, however, that occasionally *sarcomata of the lid* exactly simulate these tarsal cysts in the early stages of their career, when they may appear as somewhat elastic growths over which the integument is movable. Later rapid growth, ulceration, and involvement of the orbit arise. In an analysis of thirty-five cases by W. H. Wilmer,[1] the ages varying from ten months to seventy-six years, death is mentioned as having resulted from the disease in sixteen per cent., all children, while in forty per cent. the return of the growth is recorded. The gravity of the prognosis becomes evident from these figures, and the necessity for early diagnosis and prompt operative interference is urgent.

Molluscum contagiosum, according to all evidence, appears to be correctly named, and the contagiousness of this tumor to be firmly established. As Muetze[2] has shown, the disease on the margin of the lid may be the cause of conjunctival catarrh, and represents an unquestionably contagious affection. He is not prepared to state whether or not the mollusk is caused by a parasite, and regards the molluscum corpuscles as a product of degeneration of the epithelial cells caused by the contagion, whatever this may be.

Abscess and furuncle of the lid, usually arising in debilitated children, sometimes without ascertainable cause, but also resulting from exposure, from injury, and from diseases of the orbit, have in a number of instances been directly traceable to the depressing effects of epidemic influenza. Cases of this character were reported in 1890 by Landolt and by the writer in connection with the epidemic then prevalent. This experience has been confirmed during the past and the present year. The ordinary treatment of lid-abscess—namely, early opening and the use of hot compresses—is indicated.

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[1] Transactions of the American Ophthalmological Society, vol. vii., Part I., 1894, p. 92.

[2] Archives of Ophthalmology, 1897, vol. xxvi. p. 15.

CONJUNCTIVITIS.

Acute conjunctivitis appears under a variety of conditions, the inflammation of the conjunctiva varying from a slight hyperæmia of this membrane and a moderate muco-purulent discharge to considerable swelling of the tissues, with profuse muco-purulent, stringy discharge, great congestion of the ocular conjunctiva, and subconjunctival hemorrhages. This latter severe variety, or *acute contagious conjunctivitis* (vulgarly called "pink-eye"), appears to be due in a great majority of cases to the bacillus isolated by Dr. John E. Weeks, of New York.

In other regions a very similar conjunctivitis, if not an identical one, results from the contagion of the pneumococcus of Fraenkel. Pneumococcus conjunctivitis has been particularly well studied by H. Gifford, of Omaha,[1] who gives the literature of the subject, and who shows that it is not confined to children, as was first supposed by Moran. In other cases of acute epidemic conjunctivitis the active agent appears to have been a streptococcus or staphylococcus (Gasparini), sometimes a diplococcus (Wildbrand, Saenger), and recently the bacillus septatus (Gelpke).

A well-established *subacute* or *chronic* conjunctivitis is due to a *diplobacillus* first isolated by Moran. It is best treated by a one-half per cent. solution of sulphate of zinc.

These etiological considerations are important from the therapeutic stand-point, because acute contagious conjunctivitis is very common in children, and spreads with great rapidity from one member to another in a household, and with alarming virulence through schools, eleemosynary institutions, and similar buildings. Furthermore, the investigations of Uhthoff and Axenfeld have demonstrated that sloughing ulcers of the cornea, particularly hypopyon keratitis, are in many instances due to the pneumococcus of Fraenkel. Therefore, while acute contagious conjunctivitis is not ordinarily associated with corneal ulceration, although, according to Morax, it may occasionally have this association, it is quite capable in a susceptible eye, or in one in which a corneal abrasion occurs, of infecting the cornea and producing destructive inflammation.

The *treatment* of acute contagious conjunctivitis requires, first, prompt isolation of its subjects, the use of separate towels and washing utensils, and careful local treatment, which should include frequent irrigation of the conjunctival cul-de-sac with the following prescription: boric acid, ten grains; common salt, four grains; distilled water, one ounce; or with a weak solution of bichloride of mercury (1 to 10,000), or a solution of formaldehyde (1 to 6000); if the discharge is very free, painting the everted lids with a solution of nitrate of silver, five grains to the ounce, and frequent washing of the margins of the lids with castile soap and tepid water.

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[1] Archives of Ophthalmology, 1896, vol. xxv. p. 314.

Purulent Conjunctivitis.—This affection, so far as it interests physicians in connection with children, appears chiefly under that form which is known usually by the term *ophthalmia neonatorum*, but which more properly is designated *conjunctivitis neonatorum*. While all severe cases of this disease are known to be due to the presence of the gonococcus of Neisser, recent investigations, especially by Chartres,[1] show that in addition to the gonococci there may be found ordinary micrococci, Loeffler's bacillus, and streptococci. The most fatal cases appear to be those in which the streptococci, in conjunction with the gonococci, are the active agents; hence the importance of bacteriological examination.

Finally, it seems that a non-specific variety in which none of the active micro-organisms are present may arise. Therefore a purulent vaginal discharge (gonorrhœal) is not a *sine quâ non* of this affection, but it may arise from the introduction of any muco-purulent discharge during birth, while careless bathing by the use of soiled towels or sponges after birth are evident sources of infection.

It is probable that injudicious intra-vaginal antisepsis with strong solutions of bichloride of mercury may originate a vaginitis itself capable of inducing one form of conjunctivitis neonatorum, and the best obstetricians confine the application of germicidal solutions, in uncomplicated labors, to the external genitalia.

Exceptionally it should be remembered that inoculation occurs *in utero*, owing either to the penetrating power of the gonococcus or to infection after rupture of the membranes. This is the so-called *ante-partum conjunctivitis*.

The symptoms and consequences of this disease have been fully described in the preceding volume.

The prognosis necessarily depends upon the type of the disease and upon the results of bacteriological examination. It is grave in proportion to the virulence of the affection and to the presence of pathogenic micro organisms. Under the guidance of competent medical advice, *if the patient is seen while the cornea is still clear*, except in those examples which assume a diphtheritic type, or in those occurring in children exhibiting great physical depression, the case should be brought to a successful termination. The chief fault lies in the indifference of attendants to what seems to them at first a trivial inflammation.

Treatment.—Although the treatment of this affection has been thoroughly described in the previous volume, it is so important for the preservation of the sight of the afflicted child that at the risk of repetition it is again summarized. Treatment should meet four indications:

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[1] Arch. Clin. de Bordeaux, No. 12, December 12, 1896.

(*a*) During the earlier stages, when the inflammatory swelling of the lids is great, in addition to proper cleanliness, the local application of cold is the most useful agent. This should be applied as follows: upon a block of ice square compresses of patent lint are laid, which in turn are placed upon the swollen lids and are as frequently changed as may be needful to keep up a uniform cold impression.

(*b*) The discharge should be constantly removed, and, if possible, by a trained hand. In order to accomplish this, proceed as follows: gently separate the lids, wipe away the tenacious secretion with bits of moistened lint or absorbent cotton, and irrigate the conjunctival sac freely with an antiseptic solution. For this purpose a saturated solution of boric acid, or a solution of bichloride of mercury, one grain to the pint, may be employed. Equally valuable is a solution of formaldehyde (1 to 3000 or 1 to 6000); or copious irrigations of permanganate of potassium (1 to 2000) may be practised. The irrigations may be made with an ordinary pipette, or with one attached to a vessel held a short distance above the head of the patient, the fluid being conducted through a tube to which the pipette is attached or to which a special lid irrigator is fastened. Great care should be taken that the point of the pipette does not injure the surface of the cornea.

(*c*) As soon as the discharge becomes free and creamy, nitrate of silver should be employed, as follows: carefully evert the lid and secure complete exposure of the inflamed tarsal conjunctiva; remove all discharge and flakes of lymph by irrigating the surfaces with a cleansing lotion, wiping away the adherent particles with moistened cotton; carefully touch the area thus prepared with a cotton mop which has been dipped in a solution of nitrate of silver, ten grains to the ounce; neutralize the excess with a solution of common salt, and keep applying the saline solution until a clean red surface is secured; return the lids to their proper position and carefully inspect the cornea before leaving the case, and see that this inspection is made at each dressing of the eye; finally, grease the margins of the lids with pure vaseline, some of which should be introduced within the conjunctival cul-de-sac.

(*d*) Should the cornea become hazy, or should a small ulcer form, atropine drops may be used in a strength of two grains to the ounce. Exceptionally in peripheral ulcers eserine (gr. $\frac{1}{8}$ to f℥i) may be used.

If there is corneal haze, indicating low vitality of this membrane, the cold compresses may be replaced by *hot* applications, which should consist of squares of lint wrung out in a slightly carbolized solution of a temperature of 120° F.

This is the author's method of treating severe cases of ophthalmia neonatorum. Many other solutions are recommended; for example, alum, eight grains to the ounce, peroxide of hydrogen, cyanuret of mercury (1 to 1500), and aqua chlorinata. While the author has no objection to the use of bichloride of mercury in proper strength (1 to 8000), he is convinced that in many instances a sense of false security has arisen simply because the drug has been employed and because of its vaunted germicidal properties. Strong solutions of sublimate may occasion cloudiness and even ulceration of the cornea, and should not be used. Success in treatment depends largely upon seeing the case early, upon faithfulness of the attendants, and upon assiduous attention to details.

Prophylaxis.—Credé's method of prophylaxis—that is, the instillation of two drops of a two per cent. solution of nitrate of silver in the eyes of the newly-born child, followed by small compresses soaked in a solution of salicylic acid laid upon the closed lids—is to be used always where previous examinations have shown undoubted infective discharges in the genito-urinary passages of the mother. Carelessly used, it may be followed by what may be termed a traumatic conjunctivitis. The author has himself described one case of violent hemorrhage from the conjunctiva following its use, and has seen several instances of very sharp conjunctivitis, in one associated with the formation of a false membrane and an abrasion of the cornea which resulted in a corneal macula. Equally good results will follow the instillation of a weaker solution of silver (1 to 500) when this is associated with painstaking cleanliness during birth and also during childbed. When there is no reason to suspect gonorrhœal infection, or when examinations prior to birth have proved the absence of irritating vaginal discharges, it is sufficient to cleanse carefully the surface of the lids as soon as the child is born, then gently separate them and flush out the conjunctival cul-de-sac with a saturated solution of boric acid, or with aqua chlorinata one-half strength.. From this it is evident that Credé's method, or a modified Credé's method, is *absolutely necessary* in any case where contagion may be suspected, but that milder measures will suffice in the ordinary private obstetrical practice, *where examinations have shown the absence of infection*, and that reckless instillation of nitrate of silver is to be condemned.

Diphtheritic Conjunctivitis.—The formation of a diphtheritic membrane upon the conjunctiva is so serious an affection that any advance in the therapeutics which in so many instances have proved ineffectual is necessarily a matter of paramount interest. This is particularly the case because, while the disease is very prevalent in certain portions of Germany and France, and not so frequent in our own country, its occurrence, so far as the experience of the writer is concerned, is rather on the increase. While in general terms it has been stated that its treatment should be similar to that of conjunctivitis neonatorum, there are many points of essential difference. Valude condemns cold compresses, nitrate of silver, and sublimate lotion, and justly so. He believes that the essential basis of the treatment should consist of antiseptic sprays followed by the application of iodoform salve. Sourdille washes the conjunctiva with biniodide of mercury (1 to 20,000), removing the false membrane with a solution of biborate of sodium, and touching all the affected areas of the conjunctiva with a cotton mop dipped in a preparation composed of five drachms of glycerin and half a drachm of carbolic acid. In the interval he employs a salve of methyl blue (1 to 1000), because it is stated that Loeffler's bacillus does not grow in bouillon containing pyoktanin, even in very minute proportions. Both of these authors commend lemon-juice applications.

The success which has attended the use of antitoxin serum in the treatment of pharyngo-laryngeal diphtheria has directed the attention of ophthalmologists to this means of medication in the management of cases of diphtheria of the conjunctiva. Favorable cases have been reported by Coppez, Desseaux, Jessup, Morax, Weeks, and many other observers. Inasmuch as it is sometimes difficult to distinguish between a true diphtheritic conjunctivitis and a case of pseudo-membranous conjunctivitis, Morax has advised, in the absence of a bacteriological examination, that all cases should be submitted to an injection of serum. Locally, the eyes may be treated with a hot boric acid solution, atropine or eserine being employed, according to the circumstances. The author has had no personal experience with the use of antitoxin serum in the treatment of diphtheritic conjunctivitis, but would certainly employ it in any case that came under his charge.

Granular Conjunctivitis.—This inflammation of the conjunctiva, characterized by the appearance of rounded granulations,—that is, the *trachoma bodies,*—which results in cicatricial changes in the lids and vascularization and ulceration of the cornea, has been fully described in the previous volume, but requires reference here on account of certain recent views in regard to its etiology, classification, and treatment.

While it is probable that granular lids depend upon a micro-organism, perhaps the diplococcus of Sattler and Michel, the cause of trachoma has not been positively identified; neither has the fungus described by Noiszewski nor have the parasitic protozoa found by Pfeiffer been proved to be the specific cause. The dispute in regard to the identity or non-identity of folliculosis and granular lids has also not been settled, although it seems to the author that the weight of testimony tends to prove that the trachoma bodies should be regarded in one sense as new growths of special pathological character, and not simply derivatives from the natural lymphatic follicles. While it is impossible to separate sharply the various types of granular lids, the following varieties may be mentioned:

(1) Papillary trachoma, in which the trachoma bodies or follicles are sparsely present and are hidden from view by hypertrophied conjunctival papillæ.

(2) Follicular trachoma, in which the presence of follicles is the chief characteristic. Some authors consider the ordinary follicular conjunctivitis a variety of this type. In one form, designated by Knapp non-inflammatory follicular trachoma, the spawn-like granulations develop in the conjunctiva without evidence of inflammation, and may be regarded as analogous to naso-pharyngeal adenoid hypertrophies.

(3) Mixed trachoma, in which the follicles lie among the hypertrophied and inflamed papillæ, but are not hidden by them.

(4) Sclerosing trachoma, in which, after an initial stage of ordinary granulations, fibrous flattened excrescences develop in the upper and retrotarsal conjunctiva.

(5) Cicatricial trachoma, in which atrophy and scar tissue alone are manifest.

Treatment.—If the trachoma is acute, or if a chronic case undergoes an acute exacerbation, and, added to the presence of hypertrophied conjunctival papillæ and trachoma bodies, there is high-grade inflammatory reaction with profuse discharge, the treatment resolves itself into one similar to that suited to purulent conjunctivitis,—namely, frequent irrigation of the conjunctival cul-de-sac with mild antiseptic lotions, bichloride (1 to 8000), formaldehyde (1 to 2000), saturated boric acid, etc., together with applications of a solution of nitrate of silver to the everted lids in the manner already described. The medicinal treatment of the ordinary forms of chronic trachoma, in the author's opinion, should include the following applications:

(*a*) Nitrate of silver, ten grains to the ounce, during any stage associated with much discharge.

(*b*) Copious irrigations of permanganate of potassium (1 to 1000), which may be employed in connection with nitrate of silver in cases associated with much discharge and exuberance of the papillæ.

(*c*) Strong solutions of bichloride of mercury (1 to 500) applied to the everted lids with a cotton mop, associated with frequent irrigation of the conjunctival cul-de-sac with a tepid solution of the same drug, one grain to the pint; suitable in practically any stage of granular lids, but especially when there is decided development of the follicles.

(*d*) Sulphate of copper in the form of a smooth crystal, which is rubbed over the everted lids and well across the retrotarsal folds; useful in any stage except that in which there is much discharge, and particularly valuable in the later periods of the disease.

(*e*) Boro-glyceride, twenty or fifty per cent., or a solution of tannin and glycerin, from twenty to thirty grains to the ounce, applied in the usual manner to the everted conjunctiva; most valuable in milder cases, or after cicatrization has begun, or when stronger solutions of caustics and astringents are followed by unfavorable reaction.

Except in the acute stages, however, the duration of this tedious disease is materially shortened by suitable operative interference. Of the various operations proposed, the *expression of the trachomatous material* by means of a suitable forceps, Knapp's roller forceps being the most valuable, is the most effectual operation. It is particularly suited to cases of spawn-like granulations (follicular trachoma and non-inflammatory follicular trachoma) and diffuse hyaline infiltration. It may also be used in cicatricial trachoma when patches of hyaline degeneration are present. After the lids have been thoroughly rolled and all of the morbid material has been expressed, cold compresses should be applied until the reaction has subsided, and each day the lids should be carefully everted, the lymph removed, the swollen mucous membrane exposed and treated with a solution of nitrate of silver, five grains to the ounce, until the discharge ceases, when the cure may be completed by daily touchings with a crystal of sulphate of copper. This subsequent treatment is essential, otherwise adhesions and disastrous results may follow the operation.

The severer operations of *grattage*, or scrubbing into the granulations a strong solution of bichloride of mercury (1 to 1000) with an ordinary tooth-brush, excision of the retrotarsal fold, the application of the galvano-cautery, and abscission of the granulations are, in the judgment of the author, not often required, and should be employed only with the greatest caution.

The same precautions that have already been given in regard to acute contagious conjunctivitis apply with even greater force in the case of trachoma when it breaks out in asylums and similar institutions. When trachoma, either in its severe or in its milder and follicular types, becomes epidemic in a public institution, it may require many months before it is stamped out. Those interested in this subject should read the excellent directions given by Stephenson in his work on Epidemic Ophthalmia.

Phlyctenular Kerato-Conjunctivitis.—This disease, characterized by the formation of single or multiple vesicles or pustules on some portion of the cornea or its immediate neighborhood, furnishes the greatest number of ulcers of the cornea found in early life, and is so important on account of the unfortunate results which may occur, so far as vision is concerned, if it is neglected or improperly treated, that a few points in its etiology and management require review. Although commonly seen in strumous subjects or in those whose nutrition is much below par, and therefore representing a disease which is to a certain extent a local manifestation of a general complaint, the relation of the naso-pharynx to its development is of paramount importance. Practically always there will be found inflammatory diseases of the nasal passages, an irritating rhinitis being a constantly associated disorder; also obstructive disease, and particularly adenoid vegetations in the pharynx. The very fact that the affection often follows in the wake of measles and other acute exanthemata, which of themselves are so frequently complicated with various types of rhinitis, indicates the close etiological relationship of naso-pharyngeal disease.

A no less important influence resides in improper diet. It is a matter of common observation that children who are allowed to have pastries, sweetmeats, tea, and coffee will not be cured of phlyctenular kerato-conjunctivitis until proper dietetic principles are instituted.

Treatment.—The local treatment of this affection has been described in the preceding volume, and consists in keeping the pupil dilated with atropine until irritation has passed away, when some stimulating drug—

for example, the yellow oxide of mercury—may be applied to the conjunctival cul-de-sac in a strength of one grain to the drachm of cosmoline, or calomel may be dusted into the conjunctival cul-de-sac, provided that no form of iodine is being exhibited internally. The best possible hygienic surroundings must be obtained, with fresh air and wholesome food. Cod-liver oil, iron, quinine, and arsenic are the most acceptable internal remedies. Thorough examination of the rhino-pharynx is of paramount importance, and treatment of this region according to the indications. In stubborn forms of recurring phlyctenular keratitis, associated with ulceration of the cornea, these measures are not sufficient, and the treatment resolves itself into the management of purulent ulcer of the cornea.

While the prognosis of this disease is good if it is properly treated, it becomes a very serious affection if neglected. In severe cases there may be decided loss of the corneal substance and the development of distinct scar tissue.

CORNEAL ULCERS.

As already stated, the majority of corneal ulcers in childhood may be traced to the affection which has been briefly described. In addition to these may be mentioned the small central ulcer, the excavated or gouged-out ulcer, the shallow central ulcer, and the infecting or sloughing ulcer which is caused by infection with the pneumococcus, all of which, both from the etiological and the therapeutic stand-point, are referred to in detail in vol. iv. pp. 105–111.

The treatment of severe ulceration of the cornea, no matter what its etiology, is so important for the future well-being of the ocular tissues that a summary of the author's methods, particularly in his service in the Jefferson Medical College Hospital, in the order in which the treatment is instituted, is appended:

(1) A thorough search is made for the cause of the ulcer, and if possible it is removed. This search includes careful examination for the presence of a foreign body, a misplaced cilium, conjunctival inflammation, diseased lachrymo-nasal ducts, affections of the rhino-pharynx, carious teeth, and constitutional affections of all types. Whatever the cause, it must be removed as quickly and as radically as possible.

(2) At frequent intervals moist heat is applied by means of lint or flannel compresses dipped in water at a temperature of 120° F.

(3) If there is unhealthy conjunctival discharge, a solution of mercuric chloride (1 to 6000), or a saturated solution of boric acid, or formaldehyde (1 to 3000) is instilled in the conjunctival cul-de-sac at frequent intervals.

(4) Sterile atropine drops, two grains to the ounce, are instilled with sufficient frequency to maintain mydriasis if there is a tendency to iritis, while eserine, from one-quarter to one-half grain to the fluidounce, is used in peripheral ulcers with a tendency to perforate the cornea. On account of the ciliary irritation produced by eserine, it may be employed several times

during the day, and the atropine drops once or twice at night. Should an attack of iritis complicate the case at any time, the eserine must be discontinued and the atropine used more frequently.

(5) In mild cases the eyes may be protected by means of a pair of smoked glasses, but in severe cases, in the absence of purulent conjunctival discharge, a bandage should be applied lightly, but firmly, over a dry antiseptic dressing, and should keep the lids closed and at rest without making undue pressure on the eyeball. It should be worn until the floor of the ulcer is covered with epithelium, which protects it from external irritation. Before applying the bandage, atropine is instilled, and iodoform which has been pulverized and sterilized is dusted on the ulcer.

(6) If the ulcer shows a tendency to spread rapidly, it is curetted by means of a specially devised instrument, and immediately afterwards is gently touched with a probe dipped in pure carbolic acid, or with a wisp of twisted cotton dipped in a solution of nitrate of silver (gr. x to f℥i), tincture of iodine or formaldehyde, 1 to 60. Preference is given to the tincture of iodine or formaldehyde. These applications must touch *only* the ulcerated surface.

(7) If the ulcer continues to spread, the actual cautery may be used, preferably a galvano-cautery provided with a suitable point, or, if this is not at hand, a piece of platinum wire secured in a handle is heated as hot as possible in the flame of an alcohol lamp and applied to every portion of the ulcer, the area of which is outlined by means of fluoresceine. A drop of Gruebler's fluoresceine (2.5 per cent. solution) will color green any portion of the cornea deprived of its epithelium, and therefore furnishes a reliable guide to the extent of the destructive process. After the application of the actual cautery the eye is dressed in the ordinary manner.

Subconjunctival injections of mercuric chloride have been highly recommended in corneal ulceration, but the author's experience has not given him a favorable impression of their value; certainly injections of physiological salt solution answer equally well. The treatment probably depends upon the promotion of lymphatic activity, and not upon any germicidal value of the fluid employed.

With the local treatment of corneal ulceration and all proper attention to the entire cephalic mucous membrane, constitutional measures must not be neglected. The same dietetic principles urged in connection with phlyctenular kerato-conjunctivitis are most important, and the various tonics, alteratives, and stimulants, according to the indications.

The treatment of the results of corneal ulceration requires no further mention than it has received in the previous volume, save only that if an eye goes to destruction, and an extensive staphyloma forms, so disfiguring as to require removal of the eye, it is better to substitute for enucleation Mules's operation,—that is, evisceration of the globe and the insertion of a glass ball into the scleral cup, which furnishes an excellent stump on which to place an artificial eye, and, moreover, prevents the flattening of the face which so commonly follows enucleation in children, owing to lack of development of the tissues upon the side on which the operation has been performed.

Interstitial Keratitis.—The most important corneal disease of the non-ulcerated variety which occurs in children is interstitial keratitis, fully described in vol. iv. pp. 112–116, a disease which is an inflammation of the chronic type, a diffuse keratitis, during which the cornea gradually becomes thick with haziness until it resembles ground glass, while superficial and deep vascularization accompanies the affection.

Although our views in regard to its etiology have not changed in so much that it is certain that a large percentage of the cases is due to inherited syphilis, it seems evident also that a goodly number should be attributed to rachitis, to scrofula, to malaria, to rheumatism, and sometimes simply to depressed nutrition. While the syphilitic cases generally present other marks of syphilis, particularly Hutchinson's teeth, it should always be remembered that this dental defect is not a pathognomonic symptom of the syphilitic affection, and may occur in children in whom all the evidences of syphilitic taint are lacking.

Treatment.—While it would be impossible to lay down hard and fast rules for the treatment of each case, in addition to what has already been said in vol. iv. on this subject, the following summary may be useful, and in the author's opinion represents a regimen applicable to each case, no matter whether it is syphilitic in origin or whether the parenchymatous keratitis has occurred from other causes, because the pathological lesion is similar,—namely, an infiltration in the true tissues of the cornea, which must be relieved or dispersed if vision is to be restored. Locally, all irritating applications are contra-indicated. Atropine drops or a similar mydriatic (scopolamine is very useful) should be employed sufficiently to maintain dilatation of the pupil and to prevent iritis. Frequently applied hot compresses are a useful adjuvant. Mercury, preferably by inunction, should always be ordered, and a gentle mercurial impression kept up for weeks at a time; for example, a child ten years of age may have rubbed into the skin a drachm of mercurial ointment daily without the least fear of salivation, although, of course, a suitable watch for symptoms indicating too great mercurial action is not to be neglected. Along with the mercury, according to the indications, should be exhibited cod-liver oil, arsenic, iron, and the phosphates; and, inasmuch as the affected children always present a markedly depressed state of nutrition, all those measures which are used with such success in the management of cases of neurasthenia are suitable,—namely, regular exercise, forced feeding, massage, and electricity. If possible, each case should be treated with the advantages of trained nursing and taken from the temptations of home surroundings. Under such treatment cases that were apparently hopeless in a few weeks clear up in a surprising manner, and corneas that looked little better than pieces of ground glass become comparatively clear.

DISEASES OF THE IRIS AND THE CILIARY BODY.

Diseases of the iris and, indeed, of the uveal tract are not common in children.

Iritis.—Congenital iritis is occasionally seen, and children may be born with occlusion of the pupil, and actual shrinking of the eyeball may ensue. Iritis also develops, but infrequently, during the first few months of life, from two to nine months, and in practically every instance is due to inherited syphilis. The ordinary symptoms of iritis are present,—namely, fine pericorneal injection, discoloration of the iris, sluggish or immobile pupil, abnormal reaction of the iris to a mydriatic, and the formation of attachments between the pupillary margin of the iris and the capsule of the lens, or posterior synechiæ. These features distinguish iritis from any other inflammatory complaint of the anterior portion of the eyeball.

Gumma of the iris is occasionally seen in children.

If the ciliary body is inflamed, opacities form in the vitreous and on the posterior layer of the cornea. In the latter position they manifest themselves as a triangular patch of punctate exudations, the so-called *keratitis punctata.* Sometimes iritis accompanies interstitial keratitis and entirely masks the original disease.

As the age of puberty is approached, diseases of the uveal tract become more frequent, and iritis, both plastic and serous, may be seen, the latter especially in girls with disturbances attending the development of menstruation.

Lardaceous deposits, or nodules, are sometimes seen in the iris of strumous and anæmic subjects,—the so-called *scrofulous iritis.*

Tubercle may appear in the same position and constitute a primary *tuberculosis of the iris.*

An insidious form of iritis, associated with vitreous opacities, occurs in the children of gouty parents. The author has seen several examples, and this taint should be suspected in the iritis of boys near the age of puberty.

Iritis may arise from injury, and under the influence of infection become purulent. A similar type of the disease may arise in connection with recurrent fever, pneumonia, typhus and typhoid fevers, and pyæmia.

Treatment.—It is all-important to recognize an iritis early, before the inflammation has bound down the iris to the capsule of the lens and exudates have been poured into the pupillary space. There is no excuse for not recognizing it. An examination of the pupil in all instances readily determines the affection. Locally, atropine drops sufficient to maintain mydriasis are indicated in practically all cases. A few exceptions to the rule occur in serous iritis, if the intra-ocular tension rises. In children of a proper age, if there are much pain and inflammatory reaction, leeches may be applied to the temple. Hot compresses are valuable in all forms of iritis, cold never being a permissible remedy, except in that type of iritis which is due to traumatism. The ordinary astringent applications suited to conjunctivitis are useless unless a conjunctivitis complicates the iritis. Internally, the most suitable remedies are iodide of potassium and bichloride of mercury. Mercury itself, in the author's opinion, is best given in the manner described under interstitial keratitis. Subconjunctival injections of physiological salt solution or mercuric chloride are followed occasionally by surprising relief.

SYMPATHETIC DISEASE.

Mention should be made of *sympathetic irritation* and *sympathetic inflammation*, or, in other words, those affections in which one eye is implicated as the result of disease or injury to the other. They represent two essentially different conditions, although they have the same origin, and are generally caused by one or other of the following conditions: wounds of the ciliary region which set up a traumatic irido-cyclitis; foreign bodies in the eye; perforating wounds or ulcers of the cornea in which the iris has become incarcerated; operations upon the eye; and luxation, wounds, and calcification of the lens.

Sympathetic irritation is a functional disturbance characterized by photophobia, lachrymation, blepharospasm, defective or impaired accommodation, neuralgic pains, and tenderness over the ciliary region. It recurs in the form of attacks, and while it may be the precursor of sympathetic inflammation, it is not necessarily so, and should be looked upon as a neurosis which will disappear entirely with the removal of the exciting cause, —*i.e.*, a blind eye, injured or diseased in the way already described.

Sympathetic inflammation is a serious organic disease which presents itself either as an irido-cyclitis, plastic or malignant, a serous iritis, a papillo-retinitis, or a choroido-retinitis. It arises most frequently after injuries of the ciliary region, the injured eye being called the *exciter* and the sympathizing eye the *sympathizer*.

The period of incubation, or that period of time between the reception of the injury or disease in the exciting eye and the development of inflammation in the sympathizing eye, varies from three to six weeks. Exceptionally the disease begins as early as the fourteenth day; sometimes it is postponed for long periods of time. We are uncertain what exactly is the nature of this disease, nor do we know the exact path of the morbid changes which precede the inflammation. There is reason to believe that the affection is of parasitic origin, and that either the micro-organisms or their results travel, perhaps by way of the optic nerves, from the exciting to the sympathizing eye.

Treatment.—The most important consideration is prophylaxis, or, in other words, the management of the eye originally affected. This depends upon the character and situation of the wound, or upon the stage of the disease and the amount of vision possessed by the injured or diseased organ. Frequently cases of injury are brought to the general practitioner, and he must be in a position to examine the patient intelligently and to decide what shall be done. It may sometimes happen, especially in private practice, where every advantage of nursing and careful watching is at hand, that eyes may be saved which would be sacrificed in the working-classes, but the attempt requires the gravest thought before it is made, because the onset of a sympathetic ophthalmitis may be insidious, and, when once begun, treatment rarely fully removes the structural changes which have taken place. The propriety of operating must be determined by regarding the following rules, which are modified from those given by Swanzy, and represent the published experiences of the best authorities.[1]

Enucleation or one of its substitutes should be performed on,—

"1. An eye with a wound so situated as to involve the ciliary region, and so extensive as to destroy sight immediately, or to make its ultimate destruction by inflammation of the iris and ciliary body reasonably certain.

"2. An eye with a wound in this region already complicated by severe inflammation of the iris or ciliary body, even if sight is not destroyed; or an eye containing a foreign body which judicious efforts have failed to extract, and in which severe iritis is present, even if sight is not destroyed.

"3. An eye the vision of which has been destroyed by plastic iridocyclitis, or one which has atrophied or shrunken, provided there are tenderness on pressure in the ciliary region and attacks of recurring irritation, or without waiting for signs of irritation.

"4. An eye whose sight has been destroyed, even though sympathetic inflammation has begun in the sympathizing eye, in the hope of removing a source of irritation and thus rendering treatment of the second eye more effectual.

"5. An eye in which the wound has involved the cornea, iris, or ciliary region, either with or without injury to the lens, and in which persistent sympathetic irritation in the fellow-eye has occurred, or in which there have been repeated relapses of sympathetic irritation.

"6. An eye either primarily lost by injury or in a state of atrophy, associated with signs of sympathetic irritation in the fellow-eye."

It is universally conceded that the enucleation of an eye (preventive enucleation) primarily injured, the visual function of which cannot be restored, is the surest way of preventing sympathetic ophthalmitis. It is to be remembered, however, that even a very early enucleation does not necessarily prevent sympathy in the fellow-eye, because the infective process may have begun before the operation, and may not develop for several weeks. In place of enucleation, evisceration has been practised, but has also been followed by sympathetic inflammation; neither does resection of the optic nerve (neurectomy) afford absolute security, but if the patient declines enucleation it should be used as a substitute.

If sympathetic inflammation has begun, the rules just quoted are not applicable, and enucleation must not be performed if there is any vision in the exciting eye, which in the end may prove to be the more useful organ.

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[1] See also De Schweinitz, Diseases of the Eye, second edition, p. 340.

The principles of treatment already enunciated in regard to iritis and irido-cyclitis are applicable.

In the treatment of the sympathetically affected eye operation usually has no place. Both iridectomy and sclerotomy have been advised, but it is better to await the subsidence of acute symptoms before attempting any surgical interference, unless the intra-ocular tension be inordinately raised, and then scleral incision may be practised.

The *general treatment* consists in confinement in a darkened room (moderate exercise, with eyes well bandaged, is permissible in subjects failing for lack of it); complete functional rest of the eyes, and atropine locally, provided there is no rise of tension and no atropine irritation; and leeches to the temple if the inflammation is florid. In robust subjects mercurial inunctions are useful; in more debilitated cases a course of tonics and alteratives is advisable. Under any circumstances full doses of quinine should be exhibited. Intra-ocular injections of bichloride of mercury should not be employed. Subconjunctival injections have been recommended. The author's experience with them in this disease has not been encouraging.

Prognosis.—The prognosis of sympathetic ophthalmitis is essentially grave. In some instances recovery occurs, but more frequently, especially in forms which appear as an irido-cyclitis or irido-choroiditis, the sight of the eye is lost and the organ shrinks. The varieties which appear as a serous iritis give the greatest hope for a good result.

INJURIES OF THE EYE AND ITS APPENDAGES; FOREIGN BODIES WITHIN THE EYE.

The importance of the management of the various injuries to which the eye is subject is so evident that a few paragraphs on this topic are here introduced.

Contusion and Concussion of the Eyeball.—An eyeball injured by a blow from a blunt object—for example, a billet of wood or a flying cork—may present the following symptoms: discoloration of the lid, injection of the bulbar conjunctival vessels, and hemorrhage into the anterior chamber (hyphæmia). This hemorrhage may be slight and collect in the lower portion of the chamber, or be so extensive as to fill it entirely. Sometimes, especially when there is a wound of the cornea, there is a dissemination of hæmatoidin in its layers, and this membrane assumes a greenish-brown hue, the so-called *blood-staining of the cornea*. The appearance closely resembles a crystalline lens dislocated into the anterior chamber, and is most puzzling when it is first seen.

Moderate hemorrhage of the anterior chamber is rapidly absorbed. Absorption may be facilitated by instilling a drop of atropine and covering the eye with a light pressure-bandage. In addition to, or in place of, these lesions, there may be dilatation of the pupil (*traumatic mydriasis*), accompanied sometimes by rupture of the sphincter, which can be detected as one or more small fissures by means of a convex lens and oblique illumination. The condition is not altered by treatment.

Under other circumstances the force of the blow ruptures the ciliary attachment of the iris, causing an opening, usually semilunar in shape, in the periphery of the iris at the corneo-scleral junction. It may be compared to a false pupil. This condition is known as *iridodialysis*. As long as any signs of irritation remain, atropine drops should be instilled, four grains to the ounce, and a light pressure-bandage should be worn. Occasionally the iris will form a reattachment, but usually the false opening remains, which gives but little trouble.

The contusion of the eyeball may be accompanied by *rupture of the cornea or of the sclera*. According to E. Treacher Collins, the rupture is nearly always found in the sclerotic about three millimetres distant from and concentric with the corneal margin, as this is the thinnest portion of the sclera. Ruptures may also occur in other situations. Usually the rupture includes all the coats of the eye, as well as the conjunctival covering,—that is to say, the wound is "compound;" but the conjunctiva may escape laceration and cover the torn sclera beneath it. The immediate effect of the rupture of the eyeball is extensive hemorrhage into the vitreous and anterior chamber, associated with prolapse of the vitreous humor. Sometimes the lens escapes entirely or lies beneath the conjunctiva, and in a similar way the iris may escape (*traumatic aniridia*). It is stated that rupture of the eyeball is practically never seen in children, but, while no doubt it is very rare as compared with its occurrence in adults, it occasionally happens. The author has seen one typical case caused by the blow of a whip the lash of which had been tied into a hard knot. There was extensive compound rupture of the eyeball, with prolapse of the vitreous, requiring enucleation. The treatment of these conditions is the same as that of wounds of the eyeball, and will be considered in a subsequent section.

Wounds of the Eyeball.—Wounds of the eyeball may be divided into those which are *superficial* and *non-penetrating* and those which are *deep* and *penetrating*. Wounds of the conjunctiva are usually lacerated, and generally are situated in the bulbar expansion of this membrane. The conjunctival cul-de-sac should be flushed with a weak antiseptic solution, preferably a saturated solution of boric acid, and the divided conjunctiva united with a few points of fine silk suture, which may be removed on the third day.

Superficial wounds of the cornea usually occur in the form of an abrasion, the epithelium having been scraped away by the impact of the wounding substance; for example, a finger-nail, an iron filing, or a piece of glass. The lesion gives rise to sharp pain, marked photophobia, and copious lachrymation. It may be perfectly outlined by dropping on the cornea a two per cent. solution of Gruebler's fluoresceine. Abrasions of the cornea are important because they are frequently the starting-point of serious corneal ulceration, especially if they have occurred in an eye in which there is some unhealthy secretion in the lachrymal passages.

The treatment consists in sterilization of the conjunctival cul-de-sac with a saturated solution of boric acid or a weak solution of bichloride of mercury (1 to 10,000) or formaldehyde (1 to 6000), and the application of a light, sterilized pressure-bandage. If there is much ciliary irritation a drop of atropine solution may be instilled. Usually in twenty-four hours the abrasion will heal and the bandage may be discontinued.

Penetrating wounds of the eyeball may be situated in any portion of the globe, but are common at the corneo-scleral junction, or between the corneal border and the equator of the eyeball. A penetrating wound of the cornea or of the corneo-scleral junction is followed by evacuation of the aqueous humor and generally by entanglement of the iris in the corneal wound, followed by prolapse and staphylomatous bulging. If the case is seen early, before the protruding iris has plugged the wound sufficiently to re-establish the anterior chamber, it may be possible to replace the iris with a spatula, and by the instillation of atropine or eserine, according to the situation, retain the iris in place and prevent further prolapse. During treatment a pressure-bandage must be applied. If this is not possible, after careful sterilization of the surrounding area, the prolapsed iris should be abscised precisely as in the operation for iridectomy, the eye bandaged, and for the first forty-eight hours iced compresses applied to prevent traumatic iritis.

If the case is seen when the iris has already become firmly attached to the wound and the anterior chamber has been restored, one of two methods may be adopted: the prolapsed iris or hernia may be abscised, the wound freed of iris-tissue, and, if it is gaping much, closed with a delicate silk suture; or the eye may be treated conservatively with a pressure-bandage to prevent staphyloma and the instillation of atropine to favor the reduction of the hernia and prevent iritis, the patient being kept in bed and as much as possible on his back. When the prolapse is at the corneo-scleral junction, and appears to be increasing in size and producing irritation, the author prefers the method of abscission, followed, if necessary, by a delicate suture. If the wound has extended across the cornea and it is possible to keep the patient in bed with a pressure-bandage, and there is no increase in the prolapse, the conservative method is well worth trial, as has recently been pointed out by Knapp.

If the wounding substance penetrates still deeper it may lacerate the iris, the capsule of the lens, or the lens proper, and the accident is then followed by opacity of the lens, or traumatic cataract. Under these circumstances the treatment already detailed is indicated, and when the eye is perfectly quiet, if desired, the cataract may be removed by discission or other methods.

Wounds passing through the ciliary body or penetrating the sclera farther on towards the equator are of serious import. If the lesion has been an extensive one, and especially if infection has entered and purulent iritis has begun, sight being lost, the eyeball should be enucleated or eviscerated to avoid the danger of sympathetic inflammation, as has already been explained.

If the wound is not too extensive, and especially if the ciliary body is not involved and infection has not begun, an attempt should be made to save the eye by suturing the wound, either the overlying conjunctiva or both it and the sclera, care being taken to avoid imprisonment of the underlying ciliary body or choroid in the wound. The eye must then be treated with a pressure-bandage, atropine, and iced compresses; the internal administration of small doses of mercury is advisable. These directions apply only when the surgeon has satisfied himself that there is no foreign body within the eye. Great care must be exercised in the management of cases of this character, and a steady watch for the signs of sympathetic trouble is required.

Burns and Scalds of the Conjunctiva and Cornea.—These are commonly inflicted with acids, lime, molten metal, flame, hot water, or steam, and are especially serious because they are apt to be followed by adhesion between the lids and the bulbar conjunctiva, or *symblepharon.*

If lime or metal is splashed in the eye, all particles should be removed at once; in the latter instance best accomplished by flooding the eye with water from a spigot. An acid may be neutralized with a weak alkali. Later atropine suspended in liquid vaseline, two grains to the ounce, may be introduced beneath the conjunctival cul-de-sac. Adhesions may be prevented by breaking up the granulation tissue frequently with a probe or by keeping between the opposing surfaces a sheet of gold-beater's skin. Powder grains may be picked from the cornea with a cataract-needle, or destroyed, as Edward Jackson advises, with the fine point of an electro-cautery needle. This leaves a small sterile ulcer which heals readily, leaving only a slight scar.

Foreign bodies on the cornea and conjunctiva require no special comment. They are removed in the ordinary way by everting the lid and lifting them from their place by means of an applicator on which has been twisted a bit of cotton. If embedded in the cornea, they must be picked out with a spud. It is most important that the instrument used be sterile. The author has seen very serious cases of corneal ulceration due to the reckless use of sharpened match-sticks or similar domestic implements in the attempt to remove foreign bodies.

Foreign Bodies within the Globe.—These usually consist of a chip of steel, a splinter of glass, a bullet, or pieces of brass filing. If the substance is not visible by the ophthalmoscope, owing to opacity in the media, a skiagraphic examination should immediately be made. This in a number of instances[1] has revealed the position of the foreign body.

If the foreign body is known to be of iron or of steel, an attempt should be made to dislodge it with an *electro-magnet,* introduced either through the wound of entrance or through one made for the purpose. Occasionally the body is so situated that it can be seized with delicate forceps and removed. If the surgeon has been unsuccessful in his attempts to remove the foreign body, if he is uncertain that he has a sterile wound, and if vision is much depreciated or lost, the eye should be enucleated or eviscerated, because sympathetic inflammation is almost sure to follow. In a few instances foreign bodies have been tolerated in the fundus for long periods of time, but these are the exceptions.

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[1] See paper by the author, American Journal of the Medical Sciences, May, 1897.

Lacerated, incised, and contused wounds of the eyelids call for the same treatment as wounds situated in other portions of the body.

Burns of the eyelids require no special comment except the caution that they may involve the conjunctival lining and cause symblepharon.

THE HYGIENE OF THE EYE IN CHILDHOOD.

By SIMEON SNELL, F.R.C.S. Ed.

MANY of the causes of blindness in childhood are preventable, and much can be done to obviate the onset and progress of eye-disease. Ophthalmology is to be regarded as an increasingly important branch of preventive medicine.

The census of England and Wales for 1891[1] returned the total number of persons afflicted with blindness as 23,467, being in the proportion of 809 to 1,000,000 of the population, or 1 in every 1236. The first year in which the record was taken was 1851, and since then there has been a falling off in the proportion of the blind to the general population. The following table will show the decrease and illustrate that it has been greater during the last two decennia than in the preceding intercensal periods:

Year.	Number of Blind.	Blind per Million Enumerated.	Persons Enumerated to One Blind Person.
1851	18,306	1,021	979
1861	19,352	964	1,037
1871	21,590	951	1,052
1881	22,832	879	1,138
1891	23,467	809	1,236

This decrease can scarcely have been brought about by other causes than the increased precautions taken to prevent the ravages of purulent ophthalmia, a more widely diffused knowledge of, and improvements in, the treatment of eye-affections, and diminished prevalence of diseases, such as small-pox, which were by no means infrequent sources of blindness. In spite of these improvements, however, a considerable proportion of blindness must be due either to purulent ophthalmia or other diseases specially affecting children, from the fact that out of the 23,467 blind persons of all ages 4005 were stated to have been blind from childhood, and of these 1188 children were still under ten years of age. The males even in childhood exhibit a greater liability to blindness than females. The disproportion is true of all ages, but its showing itself in childhood, before difference of occupation or of liability to accidents can come into operation, would indicate that it was apparently due to the greater liability of male children to diseases of all kinds. Ophthalmia neonatorum, for instance, which so largely accounts for blindness in childhood, is of more frequent occurrence and exhibits a larger proportion of males blind from it than of females.

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[1] Census of England and Wales, 1891, vol. iv., General Report, with Summary Tables and Appendices, p. 70.

Blindness from childhood is a serious impediment, of course, to almost all occupations, but it appears also to operate to some extent in shortening the term of life. This is shown by the following considerations: At the age of from fifteen to twenty we find 150 per million living who have been blind from youth, while in the next period, from twenty to twenty-five years, the proportion falls to 140, at from twenty-five to thirty-five to 126, and then successive decennia, 118, 113; in the period of eighty-five and upward the rate rises to 114, but the figures are too small for proper estimation. It may be inferred, therefore, that those who are blind from youth are on the average less healthy than those who are not so afflicted. The following table shows for the last three censuses and for each sex the proportion of blind at successive age-periods per million living of corresponding age and sex:

Blind per Million of Corresponding Ages.

AGE-PERIOD.	1891.			1881.			1871.		
	PERSONS.	MALES.	FEMALES.	PERSONS.	MALES.	FEMALES.	PERSONS.	MALES.	FEMALES.
All Ages	809	874	748	879	953	809	951	1,029	[illegible]
0	155	168	142	166	172	161	185	189	180
5	238	255	220	288	312	263	306	345	267
15	370	419	322	388	449	328	404	451	[illegible]
20	385	457	321	422	491	359	451	518	[illegible]
25	585	721	460	641	800	494	680	871	[illegible]
45	1,555	1,864	1,278	1,625	1,947	1,336	1,720	2,002	[illegible]
65	4,133	4,343	3,962	6,915	6,897	6,929	5,000	5,123	[illegible]
75	9,849	9,693	9,967				11,655	11,785	[illegible]
85 and upward	19,073	17,940	19,742				24,133	21,450	[illegible]

A consideration of the causes of blindness of the inmates which have been examined by me at the Sheffield School for the Blind will show many interesting points, and will demonstrate that the causes, general and local, are numerous. It must be remembered, however, that numbers of children are rendered sightless in one eye or suffer from impaired vision in one or both eyes who would not come under observation at a blind school.

Diathesis, especially the scrofulous and the syphilitic, plays a considerable part in the causation of eye-affections in childhood. The former, together with rickets, belongs rather to general than to ocular hygiene. Uncleanliness, insanitary surroundings, and improper feeding are in many instances responsible for the onset of conjunctival and corneal diseases, and for aggravating and protracting these and other disorders. Beneficial effects are often rapidly and strikingly brought about in children of this description by a brief residence in a hospital under a suitable diet and the attentions of a nurse.

General disease, and especially brain-disease, are responsible for a large amount of blindness in children. Out of the one hundred and seventy-three tabulated cases from the Blind School no less than thirty owed their blindness to meningitis. Twenty-five were apparently idiopathic, two followed whooping-cough, and in three the optic atrophy and apparently preceding meningitis occurred after injuries to the head.

The exanthemata also furnish a series of cases of blindness. Scarlet fever and measles are responsible for about two per cent. of the instances of blindness in our table. The majority of these resulted from ulceration of the cornea.

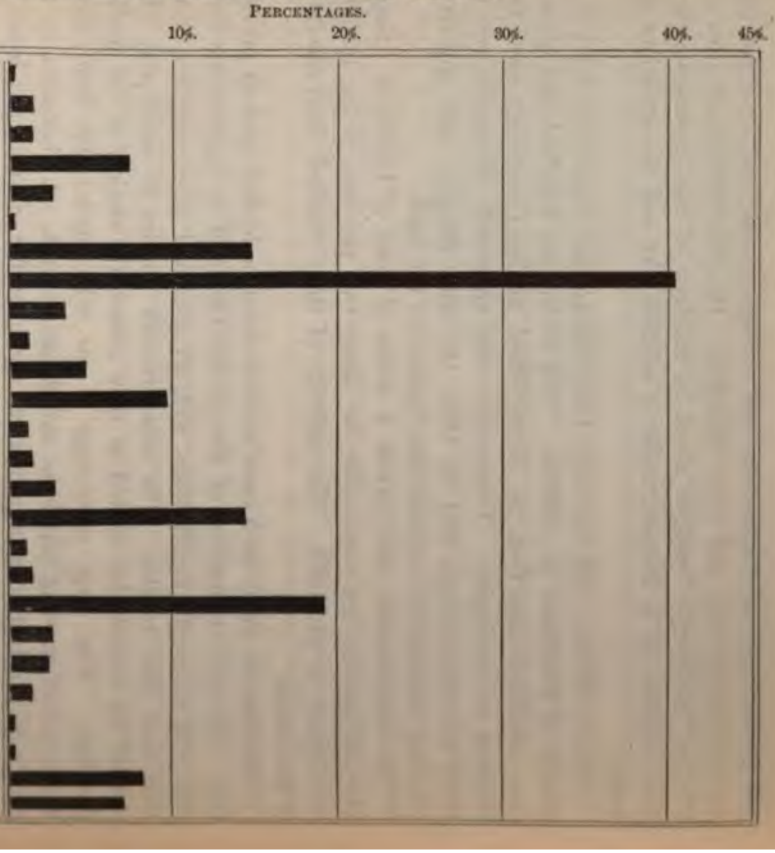
Blindness in many such cases as these must be looked upon as preventable. An early recognition of the eye-mischief and its appropriate treatment with antiseptic applications, with atropine and other remedies, would avert such dire results. The same remark applies to small-pox. The onset of the eye-trouble should at once arouse the anxiety of the medical attendant. The large percentage in our table under this head shows what a potent cause of blindness this disease is still. A discussion on the efficacy of vaccination as a preventive and modifier of small-pox is out of place in such an article as this, but mention may be made of the fact that investigation of the instances of blindness from variola shows that very few had previously been protected by vaccination. Nor is this observation confined to children. It may be safely stated that as the worst examples of variola are to be found among the unvaccinated, so also will the eyes among the unprotected be more frequently and more seriously affected.

Scarlet fever and measles also injuriously affect eyesight by inducing optic atrophy consequent on meningitis. Enteric fever also is on the list as a cause of optic atrophy.

Injuries in children are a very frequent cause of blindness or impaired vision. Many accidents result from permitting even little children to use sharp-pointed articles as playthings. Thus it comes to pass that knitting-needles, scissors, and penknives are frequently the means by which the injury has been inflicted. The employment of a fork to undo a boot-lace is another way in which, in the writer's experience, serious injury is often wrought. The child is stooping down, the fork slips, and a prong or two penetrate the cornea or the sclerotic, occasioning grave injury to or loss of the eye. In the table seven and six-tenths per cent. owe their blindness to sympathetic ophthalmitis. With one or two exceptions, in the thirteen instances out of the one hundred and seventy-three inmates of the Blind School it resulted from injury. In several the trauma was inflicted in some

TABULATED REPRESENTATION OF THE CAUSES OF BLINDNESS, WITH THE PERCENTAGES, IN ONE HUNDRED AND SEVENTY-THREE SCHOLARS AT THE SHEFFIELD SCHOOL FOR THE BLIND.

			Cases.	%.
CONGENITAL.		Anophthalmus	1	.58
		Microphthalmus	3	1.7
		Hydrophthalmus	3	1.7
		Congenital cataract	13	7.5
		Optic atrophy	5	2.9
		Choroido-retinitis	1	.58
		Total	26	15.20
IDIOPATHIC DISEASE.	Conjunctiva.	Ophthalmia neonatorum	69	40.1
	Cornea.	Cornea—diffuse corneitis	6	3.4
		Cornea—abscess	2	1.15
		Cornea—ulceration	8	4.7
		Total	16	9.42
	Iris, choroid, and retina.	Irido-choroiditis	2	1.15
		Choroido-retinitis	3	1.7
		Total	5	2.9
	Optic nerve injury	Optic atrophy (due to meningitis or brain-disease)	25	14.95
		—after whooping-cough	2	1.15
		—after injury to head	3	1.7
		Total	30	19.5
EXANTHEMATA.		Small-pox	5	2.9
		Scarlet fever	4	2.3
		Measles	3	1.7
		Measles, German	1	.58
		Enteric fever	1	.58
		Total	14	8.24
		Sympathetic ophthalmitis	13	7.6



such way as already mentioned. Something can perhaps be done by degrees in spreading a knowledge of the dangers attending the intrusting of sharp-pointed tools or instruments to children. No injury to the eye of a child should be neglected. All perforating wounds of the eyeball are more prone to induce sympathetic ophthalmitis in the fellow-organ than in the case of an adult. After an injury the eye should be rendered as aseptic as possible with the least delay. Any wound should be thoroughly cleansed by an antiseptic lotion (sublimate answers the purpose well), and if the injury be corneal it should be freed from any entanglements of iris. This is of the utmost importance. The portion of iris which is caught at the wound in many instances can, if the case is seen sufficiently early, be released by a spatula. Should it not be possible to push it back, the incarcerated portions of iris should be excised, the object being to leave the wound as free as possible from entanglements, so that close apposition will facilitate speedy union without iritic adhesions. All perforating wounds of the globe are liable to be followed by sympathetic ophthalmitis of the fellow-eye, but those of the ciliary region are especially dangerous. An eye which is hopelessly lost is better removed. It is held by some that the wide-spread employment of antiseptics for eye-injuries has made itself felt in a diminished number of instances of sympathetic ophthalmitis. Injuries with the lodgement of foreign bodies are occasionally met with in children, but the age at which they occur with any frequency is beyond that of the class we have under consideration. It need only be said that the removal of the foreign body is indicated.

CONJUNCTIVA.

Purulent ophthalmia of the new-born contributes no fewer than sixty-nine out of the one hundred and seventy-three instances of blindness from all causes in our table. This percentage (forty) differs but little from that mentioned by many other observers. Such figures, however, give no idea of the great number of infants rendered blind by it in one eye, or whose vision in one or both eyes has been seriously impaired by its ravages.

The disease is a preventable one, and, with prophylaxis carefully and rigidly carried out, its occurrence should practically cease. It is, moreover, amenable to treatment, but neglected or unskilfully treated ulceration and destruction of cornea will speedily ensue. It usually commences two or three days after birth, and is characterized by discharge of pus from the eyes and swelling of the eyelids. Both eyes are, as a rule, affected, but one may be so earlier than the other. Inoculation usually occurs at birth when the eyes are first opened. It is more frequent in primiparæ, when the labor is often tedious. A few authenticated cases are on record indicating the possibility of pre-natal infection. Should the disease commence after the third day, infection will have taken place subsequent to the labor, possibly through the agency of the mother or nurse.

The disease and its treatment are fully described elsewhere. Here we deal only with its prevention. The observations of Neisser have demonstrated it to be associated with the gonorrhœal gonococcus, and it may be held that the disease owes its origin, therefore, to gonorrhœa. Other vaginal discharges are irritating, and it may be that the less severe instances in which the gonococcus cannot be found are not necessarily gonorrhœal. The preventive measures may be mentioned under the following heads:

1. *The Man.*—No man with gonorrhœa should be permitted to marry.

2. *The Woman.*—A woman known to be the subject of gonorrhœa should be treated before and during labor with antiseptic injections. Some have advocated the use of vaginal injections during labor for all cases, but Korn, who employed thorough irrigation before and after each vaginal examination with solution of perchloride of mercury, gradually relinquished the procedure and finally dispensed with it, as it was thought that no infection took place during the passage of the child. When no examination was made no sublimate was used.

3. *The Child.*—Credé's plan is well known and has been widely adopted. He advocated that immediately after the cord was severed the eyes should be cleansed with tepid water, and that a two per cent. solution of nitrate of silver should be dropped between the eyelids. By this means he succeeded in reducing the percentage of the disease from thirteen to one per cent., and later to even less. This may be regarded as an absolute preventive of ophthalmia in infants. Simple washing of the eyes in tepid water immediately after the passage of the head, and before the infant opens the eyes, has been held by some to be sufficient. Instructions of this nature issued to the midwives of the Hospital for Women in Sheffield led to the practical abolition of the disease. The midwives received directions that as soon as the head was born attention was to be given to the baby's eyes. Then with little pieces of lint moistened in tepid water the eyes were to be carefully washed, as well as the eyelids and adjacent parts. Subsequently in washing the child care was to be taken against reinfection. Directions were also given that should a child's eyes look red it was to be taken at once to the hospital for nitrate of silver solution to be dropped into the eyes. This simple method can be carried out by any midwife, but it would be well to replace the tepid water by some antiseptic solution, such as sublimate. Some would perhaps hesitate to intrust to a nurse the use of such a strong solution of nitrate of silver as Credé suggested, but the method would hardly suffer from the employment of a weaker one.

4. Measures should be adopted to combat the ignorance which is prevalent as to the terrible results ensuing from the disease. Up to a very recent date obstetric works, and books written for midwives, made little or no mention of the disease, and omitted to enjoin the use of prophylactic means. All works either for the medical student or for the midwifery nurse should teach that the disease is a preventable one, and should set forth the measures necessary to obviate its occurrence, as well as its treatment when

it has arisen. The efforts which have been made in England to get the government to assist, through the agency of the registrars of births, in a diffusion of a knowledge of the disease and its ravages have not been fruitful. In many other directions, however, exertions have been made to spread information on the subject. For years past every parent bringing an infant suffering from ophthalmia neonatorum to my clinique has been presented with a card bearing the following inscription:

> "**Important.**—If a baby's eyes run with matter and look red a few days after birth, take it at *once* to a doctor. *Delay* is dangerous, and one or both eyes may be lost if not treated immediately."

In some parts the voluntary assistance of the registrars has been enlisted to distribute with the birth certificate a slip bearing information as to the disease and the necessity for its prompt treatment. In other places the sanitary authorities have undertaken in different ways to act with the same objects.

5. In Switzerland and in many of the States of America laws have been passed making it a punishable offence for a midwife or other person having charge of an infant to treat the disease, and necessitating the calling in of medical aid.

Purulent ophthalmia may also be caused at any period of childhood by an eye similarly affected, or may be occasioned by gonorrhœal infection. From about the age of two to that of ten years purulent conjunctivitis may be met with in girls with vaginal discharge. It is not necessary to regard all such cases as gonorrhœal, and the less grave instances of ophthalmia may be occasioned by infection from non-specific discharges. In severe cases, however, the gonorrhœal origin may be more than suspected. A correct diagnosis is possible only by means of a microscopic examination of the discharge and the finding of the gonococcus of Neisser. Inquiry will often elicit some way in which the vaginal discharge has been induced. A history, for instance, will be ascertained of the child sleeping with a mother, sister, or maid with a vaginal discharge, or the communication of the infection direct by a sponge or cloth to the eyes will be made out. The possibility of the child having been raped must not be overlooked. These inquiries are necessary to aid in preventing a spread of the disease. The danger of infection must be pointed out to those in charge of the patient, and absolute cleanliness as to their hands, etc., insisted upon. Should only one eye of the child be affected, protection must be afforded to the other by closing it with a watch-glass surrounded by plaster, or by some other method which will effectually guard it against the entrance of pus from the diseased eye.

Acute catarrhal ophthalmia may occur at any period of childhood. It not infrequently affects several inmates of the same house or family, and it may assume the aspects of an epidemic. Both eyes are usually affected, one commencing shortly before the other. The disease is especially prevalent in spring and when catarrhs of the air-passages are common. Its characteristics are redness of the eyeball, photophobia, lachrymation, a sensation as if sand was in the eye, with yellowish flakes of mucus sticking the eyelashes together. It may become seriously complicated with ulceration of the cornea. Neglected, it may pass into a chronic intractable conjunctivitis. The spread of the disease among the children of a family may be prevented by precautions against its contagiousness, the destruction of the lint or other materials used for the eyes, the use of the towel, etc., for the particular child affected only, and isolation. The virus is probably a micro-organism, and a bacillus has been described by Weekes.

Diphtheritic ophthalmia is another disorder of which the discharge is contagious. It is essentially a disease of childhood, and is one of the most serious of eye-affections. Should one eye only be affected, measures must be taken immediately to prevent the other from becoming infected. Those attending on the case must be careful to cleanse their hands with antiseptics and to destroy the materials used for dressing the eye. It is a more frequent disease in Germany than in other countries. In England it is rare and does not contribute to our table of causes of blindness. The finding of Loeffler's bacillus will distinguish true from pseudo-diphtheritic or membranous ophthalmia.

Granular ophthalmia, or trachoma, is one of the most important eye-diseases, both from its intractability and from its serious sequelæ. It is highly contagious, and where there is crowding together of people in barracks, houses, or schools, the disease once admitted, unless preventive measures are adopted, will soon spread among the inmates. The history and treatment of the disease have received considerable attention in recent years. Our space forbids our entering upon these topics. The cause and prophylaxis of the affection only can claim our attention.

The disease is met with in greater frequency in some countries and in certain races than in others, and afflicts all ages,[1] but children and old persons are less subject to it than the intermediate ages. Its occurrence as an epidemic in schools and institutions of all kinds where children are gathered together makes it an important disease to be considered in this article. The essential characteristic of the disease is expressed by its name, granular ophthalmia. The granulations or sago-grains are chiefly found on the inner surface of the upper eyelid and in the upper cul-de-sac. There is always a certain amount of discharge present, varying in quantity according as the disease is acute or chronic. A discharge which may not be evident in the daytime may often be detected early in the morning adhering to the eye-lashes before the patient has been washed.

Epidemic ophthalmia may be regarded as comprising all conditions of the conjunctiva attended by discharge. In epidemics which have usually been described the form, however, has been trachoma. Once introduced into an institution, the disease will spread like wildfire. A single case will rapidly be the source of many others. The virulence depends largely on the amount of the discharge. The older theory that the disease might be conveyed through the atmosphere has, especially recently, been giving way to a more general recognition that it is a contagious disorder and is propagated by contagion. The proper treatment of the disease and its prophylaxis are closely associated with an appreciation of its contagious nature. It may be conveyed directly by the hands or fingers, by the washing apparatus, by handkerchiefs, by bed linen or body apparel. "It may be conveyed by any article capable of being touched or used in common by sick and healthy persons." (Stephenson.) Animals may carry it, and flies in Egypt, where it is so prevalent, are credited with conveying it. Epidemics vary greatly in severity. In some throughout the instances of ophthalmia met with may be simply catarrhal, whereas in others the disease may be of such a virulent character that sight is speedily lost. As a rule, the later cases are less severe than those first occurring in an outbreak. In an epidemic the sanitary surroundings of the institution where the outbreak has arisen should be investigated, and the cubic space allotted to each child in the dormitories and the ventilation are matters which should claim attention.

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[1] From one to ten years, 6.9 per cent.; ten to twenty years, 39.5 per cent.; twenty to thirty-five years, 44.4 per cent.; thirty-five and upward, 9.1 per cent. Handbuch der Augenheilkunde, von Graefe-Saemisch, Bd. iv. S. 63; quoted by Fuchs, Causes and Prevention of Blindness, p. 154.

Follicular ophthalmia bears a close resemblance at first sight to trachoma, and it is of the utmost importance when an outbreak of ophthalmia has arisen to determine whether trachoma has to be dealt with or whether the affection is follicular. The differentiation is attended at the onset with considerable difficulty. Attention to certain points will render assistance. The vesicles in follicular ophthalmia are merely enlarged follicles, and should, as a rule, be distinguished without great difficulty from the typical sago-grains of granular ophthalmia. They will be found chiefly on the inner surface of the lower eyelid, which is also reddened, running like a chain of pearls for the greater part of the length of the lid. They seldom invade to any extent the cul-de-sac. The special habitat of the true trachoma granule in the upper cul-de-sac is not a place where these vesicles are much found. The great distinguishing feature is to be found in the termination of the two affections. Follicular ophthalmia runs perhaps a protracted course, but passes away leaving no evidences of change behind, whilst granular ophthalmia occasions cicatrices in the lids, and in more severe instances opacities of the cornea, alterations in the curvature of the lids, and displacement of the eyelashes.

Stephenson[1] has recently investigated follicular enlargement, and after a very extensive series of observations, has arrived at the conclusion that follicles are to be observed in healthy conjunctivæ, their presence being unaffected by the class of scholar or whether the school be in town or in country. He does not regard follicular ophthalmia as a separate affection, but only as an exaggeration of a normal condition. Fuchs and others hold that it is a distinct affection, whilst others regard it as leading to trachoma. My own inclination is to agree with Fuchs.

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[1] Epidemic Ophthalmia, by Sydney Stephenson. Young J. Pentland, Edinburgh and London, 1895.

Some little time since I investigated[1] an epidemic of ophthalmia which broke out in a good class boys' school. The origin was obscure. Apparently it started with catarrhal ophthalmia in one of the boys. Other boys later suffered from slight redness of the eyes with very little discharge. The cases increased in number, and an examination of the entire school disclosed the fact that a large number was suffering from follicular ophthalmia of the type which has been described. The hygienic surroundings of the school could not be seriously complained of, and it was hoped that with the measures adopted for separation of the affected from the healthy and a periodic examination of them the disease would disappear. A later thorough examination showed, however, that several of the teachers and servants who attended on the boys were afflicted in a similar manner. Ultimately the removal of the boys from the school led to the disappearance of the outbreak. The disorder throughout was of a very mild character, and taken separately hardly any case would have been regarded as worthy of much attention. Collectively, however, they led an eminent authority to look upon the outbreak as one of granular ophthalmia. This it clearly was not, as in no instance were there complications or sequelæ. The conjunctiva in all, as far as the writer knows, recovered a normal condition. Nor among the lads sent to their homes were any instances brought to light of cases arising among their families. If contagious at all, it was not so to any considerable extent. It appeared rather to depend upon sanitary conditions inherent in the premises, or upon the fact that the city sewers in immediate proximity to the school had for some time been open for alterations. Mild cases of ophthalmia prevailed in another school (for girls) with which there was some communication, and also in the city.

The essential element in dealing with an outbreak of epidemic ophthalmia is isolation. The afflicted should at once be separated and careful observation be made for the detection of other cases as they arise. They should be placed under appropriate treatment, and be allowed to mix with the unaffected only when a clean bill of health can be given them. The healthy ones should undergo periodic examination in order to deal promptly with any fresh cases of ophthalmia. The examination should include an inspection of the inner surfaces of the upper and lower lids. The surgeon should be mindful of the possibility of contagion being conveyed in the examination, and the use of antiseptics for his hands and instruments is indicated. The school premises should be inspected; the sleeping accommodation and the cubic and floor space allotted and the sanitary conditions should be looked into. The food, both as to quality and as to quantity, should be seen to; in fact, all matters that make for the health and well-being of children should receive attention. But of prime importance is the prevention of all means by which contagion can in any way be conveyed to others. This involves a stringent supervision of the lavatory arrangements. Not only should each child have a separate towel, but the towel should be hung *separately;* basins may often with advantage be abolished and washing substituted with running water from a tap.

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[1] British Medical Journal, 1894, vol. ii. p. 1104.

Enormous sums of money have on different occasions been spent either to get rid of the disorder when it has once taken root in an institution or to bring existing buildings into accord with modern sanitary requirements. In this connection I think the plan of scattered homes for children recently adopted by the Sheffield Board of Guardians is worthy of note. The primary object of the scheme was to dissociate the children as far as possible from the environment of the pauper's life and to keep them outside the walls of the workhouse, providing instead a home training and educating them at the ordinary schools attended by the children of the working classes. Scattered homes, each taking about eighteen or twenty children, have been provided in different parts of the city and neighborhood, and in 1896 these homes had nine hundred and forty-four children passing through them, and as many as three hundred and thirteen were inmates at the same time. A receiving house and children's hospital form part of the scheme. On admission each child is taken to the receiving house, there examined, detained for a time, or passed at once either into the hospital or into one of the homes, as the medical officer may decide. At a recent visit I saw five children, all members of the same family, who had been drafted direct from the receiving house into the hospital for isolation and treatment. Each suffered from muco-purulent ophthalmia, and if admitted direct into a large barrack-like institution for children they might have occasioned a serious outbreak. It appears to me that this scheme of scattered homes may be watched with considerable interest, and with a fair amount of certainty as to its effect in preventing outbreaks of ophthalmia. Each home's having such a small number of inmates must, of course, confine any outbreak, should one occur, within very small limits. A medical officer visits and reports upon each home and its inmates. Children are admitted as young as between two and three. It would be well for one of the homes to be reserved for children after discharge from hospital before allowing them to mingle with the healthy ones.

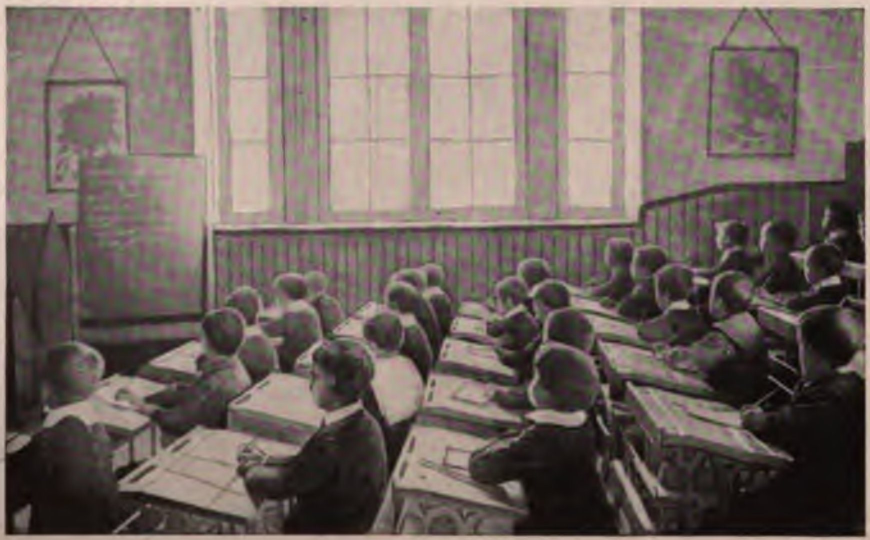
The school period has important relations to children's eyesight. It is well known that the eyes of most infants are hypermetropic, and the extra effort this condition requires for the proper focussing of near objects on the retina is liable to occasion discomfort and fatigue when the eyes are used. Fortunately, these are symptoms of which a child will often complain, and therefore, if proper advice be sought, relief will be afforded by the use of suitable glasses. Headache, so common among school-children, should cause attention to be directed to the state of the eyes. It will often be found to be dependent on overstrain due to hypermetropia or astigmatism, and the correction of the refractive anomaly will give relief.

The important condition to discuss in this connection is, however, myopia. It may be either congenital or acquired. It is the latter which chiefly concerns us now. The onset and subsequent progress of myopia bear a close relation to the conditions incidental to school life. Observation has clearly demonstrated that once developed, under the same conditions, it is prone to become aggravated. Long-continued, close application of the eyes to near-work in an imperfect light and with a stooping posture, aided often by a fatigued brain and body, are among the causes conducive to the acquirement of or aggravation of myopia. Myopia once set up reacts on itself by making further demands on convergence. Progressive myopia is often associated with organic disease of the eyes. Choroiditis, vitreous disturbances, and detached retina may be named.

The causation of myopia and its prophylaxis have been closely studied by many observers in all parts, and valuable statistics have been collected relating to its prevalence in different countries. The limits of this article permit brief reference to only a few of these investigations. Cohn, who has perhaps devoted more labor to the subject than any other observer, has shown that in Germany the number of short-sighted scholars constantly increased with the grade of the school from the lowest to the highest, and that the number of children affected went up from class to class. On repeating his observations after a few terms he found that several who were not myopic at his former visit had become so, and that others had developed a higher degree of the defect, that an actual lowering of visual power had also resulted in these myopic eyes, and that an increased number were afflicted with changes in the fundus. Erismann has recorded observations showing that the percentages of myopia increased with the lengthening of the hours of study. Those working two hours a day had a percentage of 17.7; those working four hours a day 29 per cent., and those studying six hours 40 per cent.

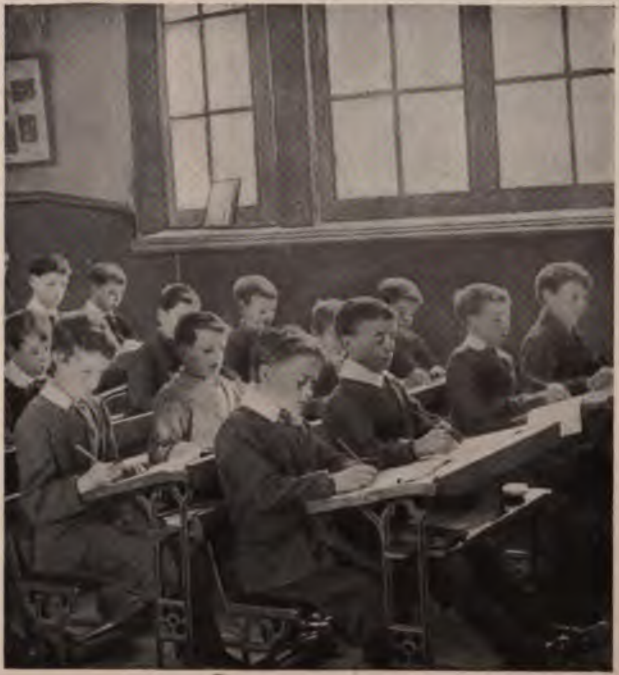
Myopia is more prevalent in some countries than in others. Germany and Russia head the list. France comes fifth, and up to the present England fares much better than other countries. Loring found in New York among children studying under the same circumstances as to hours and other respects, Germans, 23.23 per cent.; Americans, 19.35 per cent.; Irish, 14.28 per cent. Priestley Smith at Birmingham found in sixteen hundred children attending elementary schools five per cent., and among three hundred and fifty tested at a Teachers' Training College twenty per cent. In America Risley found nineteen per cent. among the pupils of the

Fig. 1.



Incorrect position of desks and light in school-room.

Fig. 2.



Correct position of desks and light in school-room.

normal schools of Philadelphia, and Randall in three thousand scholars also in Philadelphia found over eleven per cent. Dowling at Cincinnati ascertained that seventeen per cent. of all scholars were affected, but he also found that among children under nine only two per cent. were myopic, while thirty-eight per cent. were hypermetropic.

We can now pass on to consider the influences prone to act injuriously on the eyesight of school-children, and at the same time briefly to point out the measures by which they may be combated.[1]

Light, both plentiful and properly directed, is of prime importance. Indifferent or bad light necessitates a closer approach of a scholar to his book, and this it is desirable to prevent. The school may be so situated that the light is obstructed. Cohn showed that the narrower the street in which the school-room was situated, the higher the opposite houses, and the lower the story in which the lessons were given, the more numerous were the cases of myopia among the elementary classes. In France it is required that the top of a window be at a distance from the floor equal to two-thirds of the breadth of the room, and it has also been recommended that from each desk in the school there should be visible a strip of sky at least thirty centimetres in vertical extent measured from the top of the window.

The direction from which the light falls on the scholar's book is important. The left is to be preferred, and the best position for a child or an adult is that it should fall from the left, above and somewhat behind. It is often a matter merely of rearranging the seats to secure that the light shall come from the left. The right is objected to because the part of the book to which the gaze is directed will be cast into the shade owing to the position of the scholar's right arm on the desk. It may, however, supplement lighting from the left. From behind the light is insufficient unless there is side-lighting. From the front it is dazzling to the foremost scholars and insufficient for those farther back. (Figs. 1 and 2.)

In some Continental schools work has ceased when Snellen's 20-type could not be read at twenty feet. It has also been suggested that small diamond type should be legible in the parts of a room farthest from the windows. Work as far as possible, especially if the child is myopic, should be performed by daylight. When artificial light is required it should be ample and steady and come from a suitable direction.

The posture and seating of a child are of moment. It is beginning to be recognized by teachers and others interested in the education of children that "it is absolutely impossible that a tall and a short boy will both sit equally well on the same seat and at the same desk. It is just as unlikely as that the same clothes would fit the same pupils." (Snellen.) It is essential that the scholar should sit upright and that stooping should be avoided. Among the principles to be borne in mind as the requisites of a suitable desk the following points may be mentioned. A back rest, which need not reach above the loins, should be provided. The child's legs should not dangle, but should rest on a foot-board or on the floor. The height of the seat, the height of the desk, and its slope are all matters which should be observed. The slope for writing should be twenty degrees, and for reading forty degrees. The photographs (Figs. 3 and 4) show two boys seated at a properly constructed desk (Priestley Smith's), the one writing and the other reading. Contrast the posture assumed by these children with that represented in another photograph (Figs. 5 and 6), where a boy is seated on a backless bench and is leaning over a table which is too high for him. A faulty position under such circumstances is inevitable. It may be mentioned that a bad position assumed by children over their lessons is a recognized and frequent cause of spinal curvature. No child should be allowed to approach the lesson-book at a shorter distance than twelve inches. Aid must be rendered, when needed, by proper spectacles. On the Continent assistance in the same direction is obtained by employing what are called straight holders. Kallmann's face-rest is used a good deal. Cohn prefers Durr's horizontal reading support, which is a horizontal bar of iron coated with india-rubber and fastened by supports to the desk.

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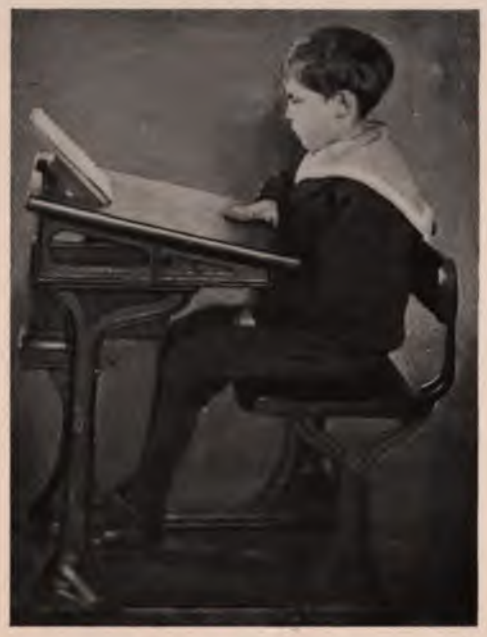
[1] *Vide* Eyesight and School-Life, by Simeon Snell. J. Wright & Co., Bristol.

The printing of school-books generally is greatly superior to that of not long since, and many of the books now placed in the hands of school-children leave little in this respect to be desired. Little children should have books with large well-printed letters which can readily be made out. All printing which is ill defined has to be brought nearer to the eye, and hence is objectionable. Cohn has laid it down that type, taking "n" as the character, which is shorter than 1.5 millimetre is injurious. The "small pica" which forms almost the standard of English books is larger than this. The printing of music should not be overlooked. Recently an eminent teacher of music drew attention to the eye-strain resulting from using the small octavo editions of Beethoven and other writers. He says "that small printed oratorios, small printed operas, frequently in two languages, present difficulties which none but those that use them in the way indicated can by any possibility imagine." (Cummings.)

The old slate once so generally in use has been rightly condemned because of the slight contrast it permitted between its surface and the pencil markings, and which was prone to become even less when the slate had become greasy from use. It has been shown that the greater legibility of black letters on a white surface from those on the ordinary slate is as much as three to four. Pens and ink have to a great extent replaced the old slate in schools.

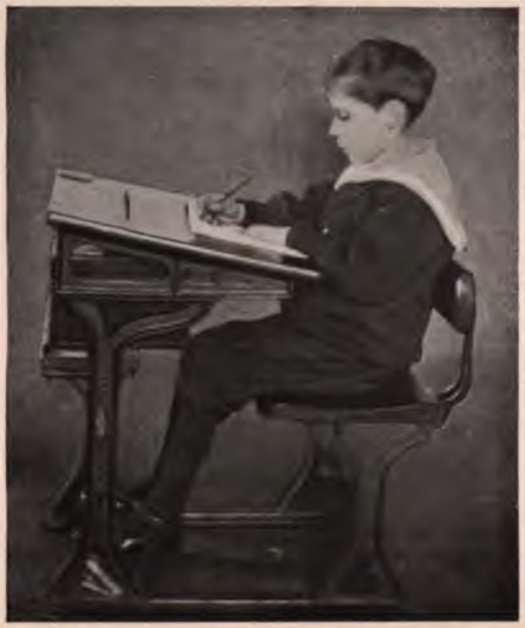
The question whether the writing should be sloping or upright has received a good deal of consideration. It appears to be more difficult with the slanting method to obtain a natural pose of body, and the writing has

FIG. 3.



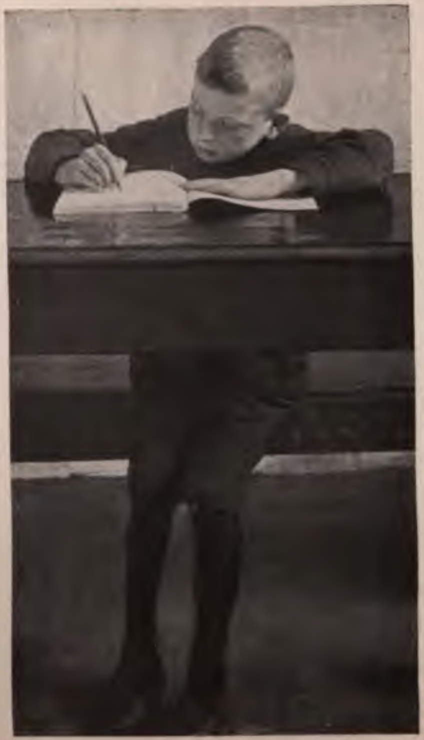
Correct reading position.

FIG. 4.



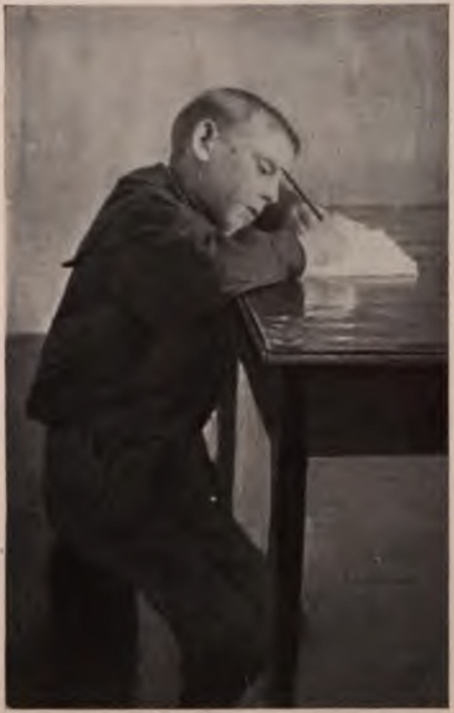
Correct writing position.

Fig. 5.



Incorrect position.

Fig. 6.



Incorrect position.

to be looked at obliquely. From an eyesight point of view there are advantages in the vertical over the sloping method. The eyes are directed straight to the copy, stooping is less likely to be occasioned, and it will be easier for the scholar to keep at a proper distance from the book.

The periods devoted to study will require careful consideration, especially if the child be myopic. In this latter event frequent breaks between the classes, even if of short duration, will be advisable. A careful adjustment of home lessons or their abandonment will also be required. Even of healthy eyes it is well to remember that a too long-continued strain at close work is often required. There is a good deal to be said for the older plan of the school hours being divided between morning and afternoon in preference to the more modern idea of a longer period at a stretch. The former allowed time between school hours for recreation, and, what is of the utmost importance, a good substantial midday meal. The encouragement of physical exercise, though pertaining more to the general hygiene of childhood, is not without its importance in considering the influences of school life on children's eyes. A great amount of good would result from the diffusion of a knowledge of school hygiene among parents, but still more benefit would accrue from enlisting the interest of the school-teachers. It should not be difficult for them to detect the grosser instances of defective sight and advise that medical aid be sought. A short-sighted child should be placed towards the front, where the task of seeing what is written on the blackboard will be easier. Snellen's test-types will enable the teacher to recognize defective sight. Mr. Priestley Smith has suggested that where a child's vision is less than 6/18 the fact should be reported to the parent or guardian, in order that proper advice may be obtained. It would be well for every child to have his or her vision tested on entering school. The condition should be noted and compared with the result when re-tested after the lapse of a year. By some such method as this defective vision in children would be detected and opportunity afforded for the parents to seek medical aid. It would disclose weak degrees of myopia, and cases which were tending from bad to worse through neglect would be recognized. It would be an additional advantage if the examination embraced a test for color-perception. Many avenues of employment are closed to those with defective form- or color-vision, and the early recognition of a child's unsuitability for such occupations could only be advantageous, and years of needless labor and expense would thus often be saved.

Many plans have been devised to prevent children from becoming familiar with the letters on the test cards. Cohn's idea may be mentioned. He has arranged E's in a square consisting of six rows of six each, making thus thirty-six E's. The branches point in various directions,—up, down, in, and out,—and, as the card can be suspended from a hook at either of the four sides, the test admits of manifold variations. The E's, for instance, may be read across from right to left, or the reverse, above downward, from either side, upward, or any line may be selected for the scholar to read. The size of letter corresponds to that of Snellen's, which the normal eye should read at six metres. This test is suitable for quite young children or for illiterates. If the direction in which the branches of the E point cannot be recognized at six metres, the child is advanced nearer to the test and the numerator correspondingly altered.

Another means which I have found convenient is to have a frame suspended on the wall with slits permitting of a line of letters being withdrawn and others substituted.

Risley has recorded some interesting observations tending to show that the care bestowed upon the correction of refractive errors in Philadelphia has led to a diminution in the percentage and a corresponding falling off in the grade of myopia.

INTRODUCTION TO THE STUDY OF NERVOUS DISEASES IN CHILDREN.

BY CHARLES K. MILLS, M.D.

IT is not intended in the pages which follow to discuss elaborately the symptomatology of nervous diseases in children, nor to describe in detail the methods of investigating the particular signs and symptoms of such diseases, but rather to present some of the general principles which should govern the physician in his examinations, and to give a few hints and suggestions arising from personal experience.

Although not a few nervous diseases may be present, actually or potentially, in the new-born, these may and usually do differ greatly in their manifestations from the symptoms of the same affections in older children. We must therefore bear in mind the age of the patient, and must not expect to find affections of the nervous system clearly manifested in those of tender years. We must, above all, be on our guard in giving an opinion at too early a date in regard to the mental or physical condition of a child in any family in which there may be an hereditary taint. Outside of its feeble movements, the condition of its reflexes, some perception of pain, and the presence or absence of gross deformities, we have very few points on which to depend in forming an opinion of the normal or abnormal state of any new-born child. The existence of paralysis at birth is not easily determined, and the forms of motor and sensory disease which are the clinical manifestations of defects in the development of the central nervous system, including certain well-recognized forms of hereditary nervous affections, such as Friedreich's disease, are often not noticed at all in the new-born infant, and may not be observed until years have passed. In these and in other cases the nervous system is imperfect at birth.

The helpless new-born infant lives largely apart from its environment; it has little power of perception, and its movements are chiefly of a reflex nature. It voluntarily extends or flexes its limbs only to a slight extent. Whilst the peripheral motor and sensory tracts are medullated at birth, and therefore capable of function, the central motor tracts are very imperfect,

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[1] The author of this paper received valuable assistance in its preparation from his colleague, Dr. Wm. G. Spiller.

and it is not until some years later that these central fibres may be regarded as fully developed. Function probably begins coincidently with the development of the medullar sheaths. The reflex movements produced by means of the sensory fibres, spinal cord, and motor fibres are present from the beginning of extra-uterine life, while those impulses which originate within the cortical cells, although in reality also of a reflex nature, must wait for their outward manifestation until the motor tracts passing to the cord are in a fit condition to convey them to the spinal motor cells.

A child is the result of hereditary laws; it is largely the reproduction in miniature of its ancestors, and is the sport of abnormal as well as of normal conditions. Inherited diseases, such as syphilis, may in some cases be detected at an early period; in other cases, however, we must wait until the gradual unfolding of the mental and physical nature of the infant reveals to us the imperfections of its nervous system. Such conditions as microcephaly, hydrocephaly, rachitis, or gross deformities may be recognized at once, even immediately after birth.

The development of a child is based essentially on principles of selfishness and self-preservation. The child recognizes first those things which contribute to its comfort; it learns to know its mother from whom it derives sustenance, and even in those children whose mental condition is of low grade the satisfaction experienced in the gratification of the instincts is frequently outwardly manifested. It is, however, at a little later period, at an age when a child begins to notice the external world and to extend its arms in order to grasp objects, that we may obtain some clue to the weak condition of the normal child. A child which at the age of two years has shown no desire to walk or to utter words is probably one that will later display more distinct evidences of imperfect development. The recognition of abnormal types at as early a period as possible is most desirable. Directions may be then given for the proper care and training of the child, or a note of warning may be raised against bringing into the world any more of the degenerated stock.

A knowledge of the most important forms of family disease, and a consideration of the age at which they are likely to be first noticed, are of the utmost value to the student of nervous diseases in children. The list of such affections is much larger than some of the many monographs and special articles in text-books and treatises would lead us to believe. It includes forms of microcephalic, hydrocephalic, and cretinoid idiocy, myxœdematous affections of peculiar type, Friedreich's ataxia, hereditary cerebellar ataxia, cerebral, cerebro-spinal, and spinal forms of spastic paralysis, other forms both of diplegia and of hemiplegia, and the family forms of progressive muscular atrophy and other dystrophies. Not a few of the forms of idiocy and imbecility, and especially those showing cranial arrest and aberration, can be diagnosticated at birth; cretinoid and myxœdematous affections may be recognized, but not quite so certainly, at a very early age. Attention is generally called to the manifestations of Friedreich's ataxia between the ages of seven and fourteen. Hereditary cerebellar ataxia usually first claims attention about the age of puberty; the forms of diplegia and hemiplegia are often discoverable at least as early as the second or third year; while the muscular atrophies and dystrophies may reveal their presence at any age from five or six years to twenty.

While the methods of examining patients suffering from diseases of the nervous system are in some respects the same for both children and adults, in others they differ, and even in the case of children the methods pursued must vary somewhat with the age of the patient. The examination for an ocular, a facial, or a limb paralysis, for marked vaso-motor or trophic disturbances, for knee-jerk or ankle clonus, for the appearances of the fundus, for the electrical condition of the muscles, for the state of the pulse, temperature, and respiration, will be on much the same lines no matter what may be the age of the patient, although even in these cases some special precautions and some modifications of ordinary methods may need to be made.

The earlier the age of the child the less will be the assistance given by subjective symptoms, and in the determination of the value to be assigned to these symptoms when present great care must be taken. The study of all the evidences of disease which can be elicited by inspection is, of course, of great value in the examination of children. Station, carriage, gait, spasmodic phenomena of various kinds, peculiarities of facial expression, and those body marks of physical degeneration which are known as the somatic stigmata are thus determined.

The child, whenever possible, should be stripped, and then should be regarded intently from top to toe. In this way much can be learned, no matter what may be its age. Every part of the body should be carefully observed before touching the patient. Charcot was very properly in the habit of speaking of the importance of a study of the nude. It is scarcely less important for the physician than for the artist to be familiar with the form of the human body, for only by knowledge of the normal conditions can he hope to train his eye to detect quickly the malformations caused by disease. Attention should be directed to the formation of the head, trunk, and limbs. Is the size of the head proportionate to the age and development of the patient? Is the anterior fontanelle closed? Is there any striking malformation in head or limbs? Are the voluntary movements of the limbs as free as experience teaches is commensurate with the age of the patient? Is the expression one of stupidity or of intelligence? These are some of the queries that the physician should address to himself.

The remarks made by Sachs in this connection are here well worthy of citation. "Never make a diagnosis," he says, "unless the child has been wholly undressed; if this is not done, a Pott's paralysis may be taken to be a traumatic myelitis, or a neuritis may be mistaken for poliomyelitis anterior. Lay the child on the table or on another person's lap in order to get a full view of it; of the relative size of head and body; of the proportionate development of arms, legs, and abdomen. Remember also that the child has heart and lungs, liver, spleen, and intestines, which, if diseased, may hold an important relation to the nervous disorder present. In proceeding to a detailed examination it is best to begin with the head, including the face; then take up the upper extremities, the abdomen, and finally the lower extremities."[1]

Knowledge of much importance may be gained from the parents. If the child was born at term we should learn whether or not the labor was difficult; whether the child was asphyxiated at birth or whether it was born with some form of peripheral monoplegia; whether the fontanelles were closed and whether the head was small at birth, or whether the child had been late in learning to walk, talk, and observe. A physician's testimony is, of course, more valuable than a parent's.

The more excitable condition of the nervous system and the greater possibility of restitution in the young may considerably modify the symptomatology. Convulsions, for example, are much more common in children, and are nearly always a bad omen. They may indicate either an organic cerebral lesion or possibly the beginning of epilepsy. On the other hand, destruction of the speech area of a child, either in the receptive or in the remissive portion, rarely causes permanent aphasia. It would seem as though the speech area of the right hemisphere, which is partially developed, even in the right-handed adult, assumes unusual functions after early destruction of the normal left speech zone, and the child with the left cortex much affected may learn to talk and understand as well as any normal one.

Let us now take up in detail the points of special interest regarding the different classes of symptoms observed in children suffering from diseases of the nervous system.

As a rule, the true insanities are infrequent in early life. The writer has elsewhere[2] called attention to some of the differences between insanity in the child and in the adult. As Maudsley has put it, where no mental faculty has been organized no disorder of mind can well be manifest. In the article referred to I have classified juvenile insanities under thirteen heads, but of these only a few are likely to claim attention,—namely, the transitory psychoses, mania, melancholia, choreic insanity, hysterical insanity, moral insanity, and obsessions (morbid fears or phobias). The transitory psychoses are of many kinds, and include several forms of mental disturbance of a fleeting character. A transient but not persisting mania may continue after the subsidence of a febrile delirium. In very rare cases children suffer from a form of agitated melancholia of transient type. Mania is the most frequent form of juvenile insanity; but it is necessary to make a distinction between it and hysterical mania. The most efficient aids to this distinction will be the discovery of some of the well-known stigmata of hysteria, although even here it must be remembered that true mania may occur in an hysterical subject. Melancholia is not likely to occur before the age of five or six years. Genuine melancholia has been reported by numerous observers, but in a child seldom assumes the extreme form observed so frequently in the adult.

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[1] Sachs, B., A Treatise on the Nervous Diseases of Children for Physicians and Students, New York, 1895.

[2] An American Text-Book of the Diseases of Children, edited by Louis Starr, M.D., Philadelphia, 1894.

While chorea is common in children, choreic insanity is comparatively rare, and yet is of sufficiently frequent occurrence and serious enough in its manifestations to make its recognition and a knowledge of its treatment important. Different forms of choreic insanity have been described. In the best known and most severe type the affection usually comes on after the motor disorder has lasted for some weeks. The patient passes into a state of maniacal excitement with extreme insomnia and uncontrollable disorders of movement. Vivid hallucinations are sometimes present.

In vol. iv. of this work the subject of hysteria has been fully considered by the writer, and it is not necessary to do more than to emphasize the fact that true hysteria may be present in children of very tender years. An important point is sometimes the differentiation of hysteria and melancholia from obsessions. Children are occasionally brought to a physician with the story that they are in constant dread of injury or accident. Almost any one of the well-known forms of morbid fear or phobia may at times be observed in them. In my own personal experience I have seen striking cases of pathophobia and mysophobia. Pathophobia, or the fear of disease, is perhaps the most common of these obsessions.

In rare cases hysterical children exhibit states of dual consciousness. A few years ago a young girl under Krafft-Ebing's care at times imagined herself to be a child four or five years younger than she really was, and while in this state she forgot the events that occurred since the time of her imagined age, and behaved as one would at the age of her assumed childhood.

Different forms of uncomfortable sensation may be described by children as pain, or may be greatly exaggerated by their susceptible and excitable brains. They are not likely to make clear distinctions between the different paræsthesias, such as sensations of burning, crawling, compression, and contraction. Almost any uncomfortable feeling may be spoken of as pain. On the other hand, a child's complaint of pain should not be carelessly dismissed. Even true migraine of severe type is sometimes observed in children of very tender years. I have seen this affection in a child three or four years old. Sensations of heat, cold, or pressure, if they are sufficiently intense, may, as Goldscheider has suggested, be equivalent to pain. The lesson to be derived by the diagnostician from these facts is not to lay either too much or too little stress upon the complaints of children as to pain, but to sift their statements with even greater care than would be used in the case of adults.

Compared with adults, it is difficult to test for sensory disturbances in children, at least up to the age of twelve or thirteen years. As a rule, young children are more responsive to painful sensations, although occasionally some unusual exception to this rule is found. The psychical response is more prompt, and although the comparatively greater suffering of children is more apparent than real, this very fact may make the value of the test less positive. That a child feels—that he is possessed of tactile sense—can usually be determined if he is of sufficient age to give intelligent replies. Many children are highly susceptible to suggestion, and the idea may take possession of a child that it does not feel, and the value of the test may thus be lessened. While the existence of the sense of pain and of temperature can be determined, the fine grades of difference may be difficult of detection, and the exact localization of limited areas of anæsthesia may be still more difficult.

It is not the purpose here to go into the exact methods of testing, which have been given in more or less detail in a previous volume, but a few suggestions will be offered. One of the best methods of testing for touch is with the finger of the physician. The degree of contact or lightness of touch can be better regulated in this way than by the use of some such object as a straw, a piece of cotton, or a feather.

The number of affections which cause sensory symptoms in children is comparatively limited, and a knowledge of these will turn the mind of the physician in the right diagnostic direction. Hemianæsthesia and segmental anæsthesia are on the whole rare up to the age of puberty, although not infrequently seen during late puberty and adolescence; but an occasional case of hysterical anæsthesia is observed even in early childhood.

Marked anæsthesia is a striking manifestation in some of the forms of paralysis following infectious diseases. Syringomyelia, which gives striking forms of dissociated anæsthesia, while sometimes a disease of early adult life, is not of frequent occurrence in childhood, although the possibility of its existence should not be overlooked. On the whole, we have little reason to expect to find in children the changes of sensation peculiar to this disease.

When pain and a paretic or a pseudo-paretic condition are present in one of the lower extremities, one's attention should always be directed to the possibility of the existence of Pott's disease, even in the absence of deformity. Pain in the trunk as well as in the limb may point in the same direction. This affection is so common in childhood that we should always be alert to the possibility of its existence. Pain or hyperæsthesia of the limbs, especially tenderness on pressure along the lines of the nerves, should arouse a suspicion of neuritis, and should lead to inquiries as to the previous administration of arsenic or other drugs or the previous existence of an infectious disease. Cases of neuritis in children caused by arsenic administered in the treatment of chorea have been reported.

In the very young it may be difficult to locate pain, for it is well known that children are frequently not able to indicate clearly to others the seat of their distress.

The examination of the pupils and of the fundus of the eye may be of great importance. It does not follow that vision is normal because nothing can be detected with the ophthalmoscope; nor is a child or an adult necessarily aware of imperfect vision even when minor changes are detected in the fundus.

In the disease described by B. Sachs as amaurotic family idiocy, the examination of the fundus becomes of diagnostic value. The examination of children with the ophthalmoscope is more difficult than in adults, for it is almost impossible to make the youthful patient "fix" an object, and one can usually hope only for an occasional glimpse of the papilla. Anomalies in the pupils and fundus are not as common in children as in adults, the two diseases, tabes and general paresis, which especially affect the pupils in adults, being extremely rare in childhood.

Probably one of the most interesting as well as important problems to the physician who sees much of the nervous affections of early life is that which concerns the patient's speech. The child does not attempt to talk at the usual age, or it does not acquire elementary speech with the facility and rapidity of other children. The solution of the problem of retarded or absent speech is by no means always easy. Many things may need to be taken into consideration, as, for instance, whether the child is idiotic or imbecile, whether it is suffering from true aphasia, or whether the affection of speech is hysterical, whether the child is a deaf mute, or whether it is simply backward in this respect, as it may be in others, and will eventually reach full stature mentally and physically. Whether or not the speech defect is associated with idiocy will be decided by other evidences of the presence of this condition. Occasionally true aphasia is of congenital origin, and, on the other hand, it may be due to acute lesions, especially to hemorrhage, although such focal disease is rare. Affections of the temporal lobe may result from the intracranial abscesses which develop in connection with purulent aural disease; and, finally, aphasia may be a consequence of hereditary syphilis. Mutism is an occasional disorder among hysterical children. True deaf-mutism must be carefully separated from all other affections of speech. It must be remembered that if a child becomes totally deaf before the age of five or six years it is very likely also to become mute, unless special means have been taken to train its powers of speech.

When, therefore, a child has made no attempt to speak, it is always well to examine into the condition of its hearing. An apparent condition of aphasia may in reality be only the result of an inability to hear. The auditory centre plays an important *rôle* in the processes of speech, and is probably the first speech centre developed. It is only after a child has repeatedly heard a word that it attempts to utter it. The functions of the auditory centre are last destroyed and first regained in lesions of the speech zone not situated in the posterior part of the left first temporal convolution. Hearing may be tested in the child as in the adult, either by approaching to the ear some instrument which produces a sound, as a watch; or by speaking to the child; or by making a noise with the hands (clapping), a method of special value when the mental condition is feeble. If the patient is old enough, a more exact examination may be made with the tuning-fork. Occasionally it happens that a young child supposed to be retarded in development or even imbecile is really suffering from the effects of adenoid growths. These, which have their favorite location in the vault of the pharynx, may not only be the common cause of the difficulties and peculiarities of speech, but may largely interfere with hearing. Whenever present they should be removed. In this connection, however, it is not to be overlooked that adenoids are not infrequently present in children who are really idiotic or imbecile.

Taste and smell are special senses less frequently disturbed in childhood. We may test them in suspected cases of infantile hysteria if the child is old enough to give an intelligent response, but, as a rule, little is to be hoped from an examination of these senses.

In the study of paralysis in children a few facts will be of importance in rapidly arriving at a correct diagnosis. If, for instance, a paralysis of the whole or part of one limb is associated with marked atrophy, with deformities at the joints, with changes in color, and with coldness, the existence of a spinal paralysis can be almost certainly declared; and if in addition to these results of inspection the muscles fail in whole or in part to respond to electrical tests, the diagnosis will be assured. Even in cerebral cases the limbs, or it may be one-half of the body, will be decidedly arrested in development; but cases of this kind differ from spinal palsies in that the paralysis is usually accompanied by spasticity, and in vaso-motor, trophic, and electrical changes not being present, or at least not of the same character. A history of convulsions is often present in the cerebral cases and rarely in the spinal. If neuritis, local, multiple, or diffuse, has been the cause of the paralysis, a history of present or of previous pain, or of hyperæsthesia and the other well-known phenomena of neuritis, are likely to be present.

If the child is old enough the movements of the face may be observed, and paralysis in the muscles innervated by the seventh nerve, if present, may be detected. Cases of facial paralysis occurring soon after birth have been reported, but they are rare. Congenital ptosis is occasionally observed, as is also recurrent oculo-motor paralysis in children. A case of this kind, evidently due to malaria and treated with quinine with great success, has recently been reported.

Much can be learned from the gait. When a child does not begin to walk until it is four or five years old, we have good reason to suspect some serious organic affection. One of the most common causes is imperfect development of the central nervous system. If the gait is spastic the child drags its toes; if suffering from idiopathic muscular atrophy it waddles like a duck (*marche de canard*); if poliomyelitis has caused the paralysis the affected limb or limbs are flaccid, and the child in severe cases can move from place to place only by the use of crutches. Of course, other signs, such as atrophy and the electrical reactions, are of assistance in making a diagnosis of the nature of the disease in any given case in which the gait is affected. A spastic gait in childhood is usually indicative of Little's disease, or congenital general spasticity.

A study of knee-jerk and ankle clonus and the other so-called deep reflexes is often a matter of considerable moment in the examination of children for nervous disease. The knee-jerk is commonly absent in diseases of toxic and infectious origin, and sooner or later in all forms of multiple neuritis. It is exaggerated in hysteria and in various forms of focal disease of the brain and local cerebral arrest. Normally it is, as a rule, a little more active in children than in adults, and more readily elicited. It may be hindered, however, by involuntary cerebral inhibition, the child, so to speak, holding the limb in spite of itself. It is often necessary to secure a nearly complete relaxation of the limbs before the knee-jerk can be obtained. Sometimes the child, frightened by the appearance of the percussion-hammer and by the slight blow on its patellar tendon, throws its muscles into a state of exaggerated spasticity, and a careless observer might easily be led to make the statement that the knee-jerks are absent when in reality they are very prompt. If the child is old enough, advantage can be taken of "reinforcement," as taught by Jendrassik, Weir Mitchell, and others; it can be made to clasp its hands or clasp the hands of some one near by while the knee-jerk is being tested, and in this way it can be elicited when it is otherwise difficult or almost impossible.

Ankle clonus is rarely present in the nervous diseases of children, but is sometimes noted in cases of cerebral or cerebro-spinal arrest, in focal disease of the brain, in caries with compression of the cord, and in lateral sclerosis. When present the phenomenon is readily evoked by suddenly flexing the foot dorsally while the knee is rested on the other hand. Care should be taken not to frighten young children by examinations of this sort, which to them sometimes have the appearance of attempts to injure them.

The plantar reflex is the only superficial or cutaneous reflex that needs consideration in the discussion of the symptomatology of nervous diseases in children. The retraction of the foot on irritation of the sole is absent in most cases of the peripheral nerves and in those diseases of the spinal cord that are due to lesions somewhere in the course of the reflex arc which joins the periphery with the spinal centres. When the lesion is higher than the level through which this arc passes the reflex may be exaggerated, although this is not as constant as is exaggerated knee-jerk under the same circumstances. As a rule, in unilateral brain-lesions the cutaneous reflexes are absent on the paralyzed side. (Sachs.)

A knowledge of the diverse rachitic affections of the nervous system, as well as of other portions of the body, is of much practical importance. Occasionally infants or young children suffering from rickets are supposed to be the victims of much more serious disease, such as spastic diplegia and paraplegia or other forms of paralytic disease. A form of rachitic pseudo-paraplegia has been described. These rachitic patients are often unable to walk, and yet they are not in a strict sense paralyzed, as they may be able to use their limbs for various movements when in the sitting or lying position. Generally, if the possibility of the presence of rachitis has been recognized, and this condition is really present, some or many of the other signs of rickets can be discovered. Deformities of the spine, of the ribs, or of the limbs, enlargement of the liver, tenderness of the muscles, and a general appearance of malnutrition will serve to open the way to a correct diagnosis. In distinguishing between a case of pseudo-paralytic rigidity and one of true spastic diplegia it needs to be remembered that the former usually follows a prolonged illness, is often associated with evidence of rickets and of the hydrocephaloid state, exhibits spasms which are frequently confined to the hands and arms and which are intermittent and of brief duration; while the latter can be traced to birth, often to a history of difficult labor or to one of asphyxia or convulsions at birth. The spastic condition may involve both upper and lower extremities, and, while it may vary in intensity, is usually persistent. (Osler.)

In connection with the study of cretinism it is well to bear in mind that a peculiar form of fetal or congenital rickets has been observed in which the appearances are very similar to those of true cretinism. The disease is, in fact, a form of rachitic pseudo-cretinism. It has been described by some English authorities, and one or two cases have been observed by me.

The importance of the recognition of rickets cannot be overrated when it is recalled that while this affection is amenable to treatment or even practically to cure, the diseases with which it is confounded are commonly of an incurable type. These rachitic patients, retarded in powers of station and locomotion, in speech and in mentality, under appropriate treatment, which should include fresh air, abundant food, cod-liver oil, and preparations of iodine and iron, are restored to health, or rather are given health and strength which heretofore they have not possessed. Tetany in children is occasionally associated with rickets, and a recognition of this fact will lead to a proper prognosis and treatment.

The forms of nervous disease which follow or accompany infectious diseases are important in their manifestations and in their results. The fact that they are sometimes present but latent in children should never be lost sight of by the physician. A child presenting the features of a post-diphtheritic or a post-scarlatinal paralysis may apparently not have had a precedent history of infectious disease, but the disease may have been latent and thus may have escaped attention. Often inquiry will develop the fact that other children or adults of the same family, or others living in the same neighborhood, have been suffering from some form of infectious or contagious disease. Prognosis and treatment, as well as exact diagnosis, will, of course, be much assisted by attention to these facts. Occasionally the mistake is made of supposing that a child suffering from a post-infectious disease is the victim of some form of arrested development.

A few words should be said about the use of electricity. This agent has a definite, although limited, diagnostic place. While it is true that a competent neurologist, and even a good general practitioner, should in the vast majority of cases be able to make a diagnosis both of the nature and the seat of the lesion causing a disease of the nervous system, without the aid of the electrical current, nevertheless, with its assistance such diagnosis can be more quickly and certainly reached, and in some instances it is the one method of investigation which absolutely clinches an opinion in a case otherwise doubtful. In those rare cases, for instance, in which the question is whether a form of monoplegia or hemiplegia is of spinal or cerebral origin, the retention or the abolition of electrical responses will settle the doubt. In several instances I have been called upon to decide whether a little patient was suffering from an obstetrical paralysis—that is, a paralysis due to pressure, torsion, or other injury to the brachial plexus or some of its branches—or from the effects of a clot or a form of arrested cerebral development. The careful use of faradic and galvanic tests decides in a case of this kind. As an obstetrical paralysis is peripheral, the partial or total reactions of degeneration will be present in some of the nerves and muscles affected, while in the cases of focal lesion of the brain or of developmental arrest the reactions will be present, although in the latter instances they may be somewhat altered. Neuritis, acute anterior poliomyelitis, and diffuse myelitis cause marked electrical changes, and it may be total disappearance of reaction. Idiopathic muscular atrophy and other diseases peculiar to childhood also cause changes in the electrical responses of the affected muscles, the nature of which is known as partial degenerative reaction.

The greatest service rendered by electricity is the same in childhood as in adult life,—namely, in enabling the practitioner, who may be in doubt in spite of other differential points, absolutely to satisfy himself as to whether or not a paralysis is cerebral, spinal, or peripheral. Electricity is also of more or less value in the differentiation of hysterical from organic paralyses. Both in hysterical and in cerebral palsies the electric reactions are normal or only quantitatively changed, but in peripheral, and in those forms of spinal paralysis in which the nerve-roots and anterior horns are implicated, changes of the most positive character are present.

Sachs has condensed the electrical conditions present in the different affections from which children suffer. They are normal in all cerebral diseases, excepting those of cranial nerve nuclei; in the diseases of the lateral and posterior columns of the spinal cord; in functional troubles; in mild peripheral troubles; and in some forms of muscular dystrophy. They are distinctly altered (R. D.) in bulbar paralysis (acute and chronic); in polioencephalitis superior (Wernicke); in poliomyelitis (acute and chronic); in progressive amyotrophies; in amyotrophic lateral sclerosis; in myelitis, but only in muscles represented in diseased level; in gliosis and tumor involving the gray matter; in anterior root-disease (syphilis, tumor, etc.); in vertebral disease (Pott's disease, tumor); in peripheral neuritis (traumatic, rheumatic, toxic); and in some forms of muscular dystrophy.

A few hints and suggestions as to the methods to be observed in the electrical examination of children may prove of service. Even adults not infrequently look with alarm upon an electrical examination, and this is not simply because they fear pain, but because they have a real dread of the effects of electricity, probably founded upon their ideas of this force as seen in nature and in the arts. Physicians are not always careful in their methods of application. No matter what the strength of the current eventually used, application should always at first be weak, and should be increased very gradually. Abrupt transitions in the strength of the current should not be made, as these cause more pain and give more shock than strong currents slowly applied. When the galvanic current is used, it must be remembered that the sensations are of a burning character and may be especially painful because of the tender skin of a young child. Rheostats or current controllers which allow very fine gradations of the strength of the current should be used. The electrodes should always be clean. This is an important point, not only for hygienic reasons, but also because clean electrodes cause less pain. Not a little of the pain produced by a faradic or a galvanic current, and particularly by the latter, is due to the polarizing currents which take place on the surface of the rheophores. For many cases, but not for all, the faradic current will answer, and this should be used by preference in such cases, as it is less painful than the galvanic. The greatest care should be taken in the use of the galvanic current to the head and neck in order to avoid disastrous results to sight and to prevent the occurrence of syncope or other serious trouble.

AMAUROTIC FAMILY IDIOCY.

By B. SACHS, M.D.

THE above title was suggested by me for a rare disease affecting several members of the same family, and characterized by a distinct lack of mental development, by a progressive weakness of all the muscles of the body, and by a defect in vision (associated with changes in the macula lutea and optic nerve atrophy) terminating in complete blindness. The disease is generally fatal, the children dying as a rule in a condition of complete marasmus before the end of the second year of life.

History.—In 1881 Waren Tay described a case presenting "symmetrical changes in the region of the yellow spot in each eye of an infant. The child was twelve months old. It was deficient in holding up its head or moving its limbs. There was weakness, but no absolute paralysis of any part. Its cerebral development was slow and poor. At the first examination, March 7, 1881, the optic disks were apparently healthy, but in the region of the yellow spot of each eye there was a conspicuous, tolerably diffuse, large white spot, more or less circular in outline, and showing at its centre a brownish-red, fairly circular spot contrasting strongly with the white patch surrounding it. This central spot did not look at all like a hemorrhage, nor as if due to a pigment, but seemed a gap in the white patch through which one saw healthy structures." The author likened these appearances to those that one is familiar with in cases of embolism of the central artery of the retina. He believed the changes in the retina to be "possibly congenital." Five months later another examination was made, showing that the disks had become atrophied, but that the changes in the macula lutea were the same as before. In the same family, according to Waren Tay's later reports, three similar cases had occurred, each one of the children presenting ocular symptoms and exhibiting physical conditions that were similar in all respects, and all three dying before the age of two years. This peculiar ophthalmoscopic finding was noted by Magnus, Goldzieher, Wadsworth, of Boston, Hirschberg, of Berlin, and H. Knapp, the last-named describing the condition as he saw it in the first case of my own.

In 1887, without any knowledge of the cases described by the oculists, I published the history and the post-mortem record of a patient suffering from what appeared to be a peculiar form of idiocy associated with blindness. The family character of the affection was not evident until a sister of my first patient became similarly affected. In still another family I saw another instance of this affection, and received the history of three other children who had been afflicted with and had died of this disease.

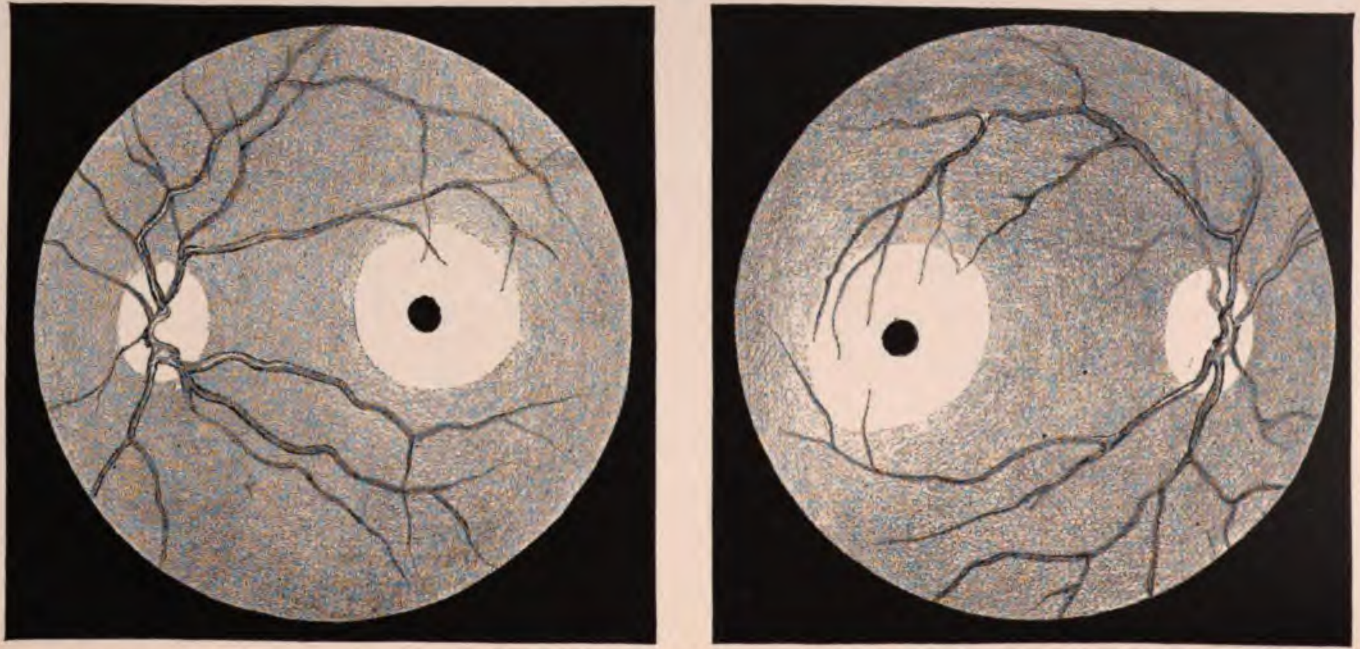
Kingdon, of Nottingham, called attention to the fact that the rare condition reported by the oculists was part of the disease which I had described. In 1894 Carter collected all cases of this disease known up to that time, and in 1896 I was able to give a list of nineteen cases, of which eight had come to my own notice. Since the publication of this last paper other cases of this sort have been described and published by Heiman, Koller, Koplik, Peterson, and Hirsch. In Europe four cases have been published by Kingdon and Russell, and one by Higier in Warsaw.

The following is a recent list of cases:

AUTHOR OR OBSERVER.	BOYS.	GIRLS.	SEX NOT STATED.
Tay	3	..	1
Sachs and Knapp	..	2	..
Koller	..	2	..
Kingdon	1	1	..
Sachs	1	3	..
Carter	..	1	..
Magnus	..	1	..
Wadsworth	..	1	..
Goldzieher	..	..	1
Hirschberg	..	..	1
Heiman	1	..	..
Koplik	1	1	..
Higier	1	..	..
Kingdon and Russell	..	..	2
Strouse (unpublished)	..	1	..
Peterson	..	1	..
Hirsch	1	..	..
Summary	9	14	5

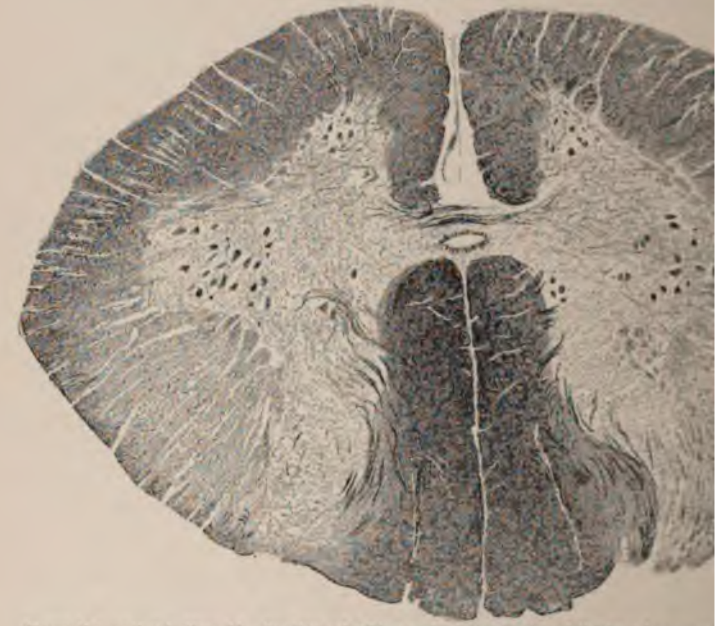
Symptoms.—The children affected with this disease are born at full term and apparently in perfect health. They appear to do very well until the end of the first three or four months of life (possibly a little later), when the parents are struck by the fact that they are listless and apathetic, move their limbs very little, and have some disturbance of vision. The child is not able to hold its head up straight nor to sit up, and as the months go on exhibits no improvement in the use of its muscles. The muscles may be either flaccid or spastic, the reflexes may be normal, a trifle subnormal, or exaggerated. In some of the cases there is unusual sensitiveness to touch and to sound, the child being made to jump by the slightest noise occurring in the room. Convulsions are rarely present, but all the functions of the body are evidently in a low state of activity, the children being subject to frequent bronchial attacks, and are harassed by unusual feebleness of digestion. An examination of the fundus reveals the peculiar condition well described by Tay, as noted above. (See Fig. 1.) There is a

FIG. 1.



Ophthalmoscopic appearances in Waren Tay's first case of disease in the region of the yellow spot in each eye in an infant. (From Frost's Atlas.)

Fig. 3.



From the author's Case II. Section of fifth cervical segment, exhibiting degener columns. (Weigert specimen.)

but this difference is due chiefly to the fact that in some instances the cortex of the brain, in others the pyramidal tracts, and in still others the cerebellar tracts are imperfectly developed.

In my book on the Nervous Diseases of Children I have on this account discussed the present disease in connection with such hereditary disorders as Friedreich's ataxia, hereditary cerebellar ataxia (type Nonne-Marie), hereditary spastic paralysis, and the like. I am firmly persuaded that its affiliations with these are very close, and it is a distinct gain to have been able to relegate a disease like amaurotic family idiocy to its proper place among a series of hereditary affections.

Pathological Anatomy.—Up to the present time our knowledge of the pathological anatomy of this disease is based upon the results of two autopsies, one by myself and one by Kingdon. (See note at end of article.)

In my first case the outer surface of the brain exhibited such abnormalities as we are accustomed to associate with brains of inferior development. I refer particularly to the confluence of the central fissure with the fissure of Sylvius and to the complete exposure of the island of Reil. The brain seemed unusually hard, the knife fairly grating as it passed through the cortex. The most important changes were those found on microscopical examination of sections taken from the frontal lobes, from the motor area, from the base of the third frontal convolution, from the first temporal, and from a part of the occipital apex of each hemisphere. The changes to be described, although a little more pronounced in the motor region, were found in every part of the brain examined. It was possible to make out the various layers of cells in the cortex, but the examination revealed the fact that in a search through innumerable sections only a few of the pyramid cells presented anything like the normal appearance. (Fig. 2.) Of well-defined processes there was scarcely a trace. The contours of the cells were either rounded or elongated, and the cell protoplasm exhibited every possible change such as we note in degenerating cells. In some cells there were a distinct nucleus and nucleolus surrounded by a granular detritus-like mass. In many the nucleus was pushed to the side of the cell. In others the nucleus and nucleolus were entirely wanting. These changes were determined by the acid fuchsin method. With the present more improved methods of Nissl and others, further changes will no doubt be made evident. In Weigert's specimens it was evident that the white fibres were but poorly developed and that tangential fibres were nowhere present. On two points there was absolute certainty,—viz., that there was no trace of any previous encephalitic process, and that the blood-supply of the cortex was entirely normal. As a result of this examination I concluded that the changes were restricted to the cells and possibly to the white fibres, and in the absence of further evidence of inflammatory changes the abnormalities of cell-structure were interpreted as the result of an arrest of development followed by degeneration.

In the second autopsy upon a case that differed from the first only in respect to the greater spasticity of the paralyzed limbs, the same changes were found in the cortical cells. There was the same absence of every encephalitic process, and there was in this second case distinct evidence of a degeneration in the spinal portion of the pyramidal tracts. (See Fig. 3.) In the pons and medulla there was no trace of any degeneration in the pyramidal fibres, nor was there any evidence of degenerative conditions higher up in the central axis of the brain. Unfortunately, the retinæ could not be examined in either case, though it is more than likely that the changes in the retinæ will be found to be developmental in character and possibly very much like the changes in the cortical cells. In my second case a very careful examination was made of all the internal organs, and the important negative fact was established that there was no evidence of hereditary or acquired syphilis.

The autopsy performed by Kingdon corroborated the findings made in my two cases, and he, too, discovered a marked degeneration in the second and third cervical segments of the cord. The question arises whether we may interpret it as a defect in development. In my own case the anterior pyramidal tracts were not affected, and Kingdon does not refer to an involvement of the pyramidal tracts in the pons and medulla oblongata of his case. While he interprets this to be a typical secondary degeneration, I cannot as yet fully endorse this view, for I believe that defective development has considerable to do with the appearance of this degeneration. According to this view, we can ally the disease with the endogenic diseases to which Strümpell refers in which degeneration is due to defective development. (Anlage.) Such developmental defect need not be congenital, but may appear at any time during life. In the disease with which we are concerned the defect is made evident in the first year of life.

Amaurotic family idiocy should not be regarded as an isolated though interesting disease, but all the symptoms and the anatomical findings suggest a resemblance to other hereditary family affections.

Treatment.—Unfortunately, little can be said in favor of any therapeutic measures. In several cases under my observation I have hoped against hope that by most careful nursing and feeding some of the children of the family in which the disease is endemic might escape, but up to the present time all such efforts have been entirely fruitless. Kingdon has suggested the use of thymus extract, but he is not yet able to state that any advantage has been gained thereby. It would be natural to infer that the wisest thing to do would be to avoid bringing such children into the world; but, as several healthy children have been born to parents who have had one or two children afflicted with amaurotic family idiocy, advice bearing upon this point can not easily be given. In conclusion, I consider it necessary to remind the oculist that these patients can no longer be regarded as afflicted with a rare ocular condition, but that the ocular condition, interesting as it is, is merely one symptom of an easily recognizable family affection.

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NOTE.—At the meeting of the American Neurological Association in May, 1898 (see *Journal of Nervous and Mental Disease*, July, 1898), Peterson and Hirsch reported upon two additional cases of this disease with autopsies. The former states that "the brain shows macroscopically and microscopically a condition of defective development; in his case the pathological conditions were limited to the nerve-cells of the cortex and medulla." Hirsch, whose examination is the most complete yet made, corroborates the findings of Sachs and Kingdon, and shows in addition that there may be an affection "of all the nerve-cells of the *entire nervous system*, the main features of which are a condition of chromatolysis and other degenerative processes of the protoplasm, combined with considerable swelling of the cell-body and displacement of the nucleus towards the periphery of the cell." The neuroglia and blood-vessels were normal (see also Holden's account of the condition of the eyes of Hirsch's patient, same number of *Journal of Nervous and Mental Disease*).

INTRACRANIAL TUMORS.

By M. ALLEN STARR, M.D., Ph.D.

In the original article upon intracranial tumors (vol. iv. p. 551) statements were made with regard to the relative frequency of the different varieties of tumors of the brain, their most common situation, their etiology and pathology. In regard to these subjects no new facts have been elicited during the past few years, and the statements there made have merely received confirmation. It is not necessary, therefore, to make any restatements of these subjects.

Regarding the symptoms of brain tumor, it is now possible to distinguish sharply between *general symptoms*, such as headache, vomiting, vertigo, double optic neuritis, mental dulness, changes in disposition and power of attention, general convulsions, restlessness and insomnia, irregularity or slowness of the heart-action and of the respiration, polyuria, glycosuria, and general exhaustion, which are produced by tumors in any location in the brain, and *local symptoms* peculiar to tumors in definite regions. Attention may be called to a general symptom which has been described by Macewen, and which appears to be of some value, as it has been confirmed by other authors,—namely, a difference in the percussion note on the two sides of the head as elicited by auscultatory percussion. Macewen states that on the side of the tumor there is sometimes found a clearer, higher-pitched, and more resonant note than upon the opposite side. This is to be elicited by percussion with a rubber hammer made at symmetrical points upon the skull, the stethoscope being applied over the forehead in the middle line, or upon the scalp when it is shaved at any point in the middle line. It is exceedingly difficult to compare the auscultatory percussion note of one side with that of the other if the stethoscope is shifted or is applied to the hairy scalp, and it is to be remembered that in children the percussion note will differ very much if the child's head is supported upon a pillow from that obtained when it is in an upright position unsupported. Hence great care must be used in eliciting this symptom, and a great deal of reliance is not to be placed upon it.

It has long been known that over the situation of a surface tumor tenderness to percussion can sometimes be elicited. Horsley has shown that pressure upon the head will often produce pain on the side of the tumor and in its vicinity; but this also is not an absolutely characteristic symptom. Many tumors produce a thinning of the skull in their vicinity, and such a thinning may account for the existence of tenderness. Thus, in a case recently seen with Professor Peabody the parietal bone was found to be reduced to a translucent thinness over an area an inch in diameter, although the tumor in the brain lay an inch deep beneath the apparently normal convolution of the superior parietal lobule and nowhere touched the dura. It is difficult to explain the effect of such a tumor upon the bone, but, inasmuch as the thickness of the bone everywhere else was normal, the relation could hardly have been an accidental one.

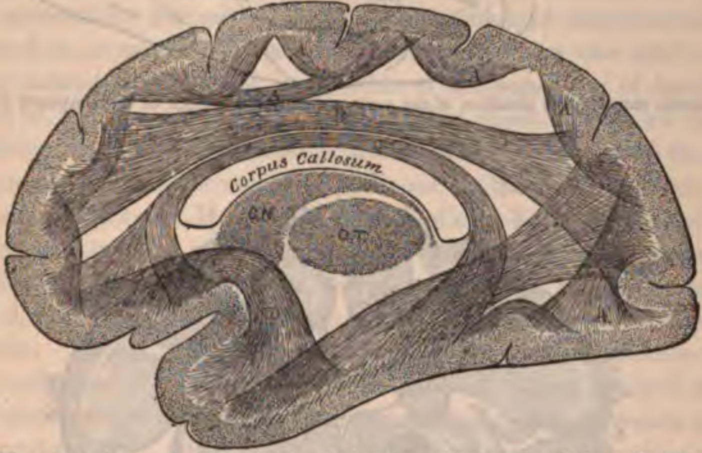
It is to be noted that the situation of the headache bears very little relation to the situation of the tumor. Thus, in a case of well-marked cerebellar tumor, the tumor lying upon the base of the cerebellum upon the right side, the situation of the headache was uniformly frontal and bilateral, and in a case in which the tumor was removed successfully from the frontal region the headache was more commonly occipital than general. The degree of the headache really depends upon the degree of distention of the lateral ventricles with fluid; for in two cases in which the ventricles were tapped the headache, vomiting, and spasms ceased, though the tumor was not removed.

A staggering gait has until recently been supposed to be a local symptom of cerebellar disease. It has been found, however, to occur in tumors of the corpora quadrigemina and of the pons Varolii and of the crura cerebri which involved the red nucleus of the tegmentum. It has also been noticed by Bruns in cases of tumor of the frontal lobes, and this fact has been confirmed by Senas, Dinkler, and Hermenidas, and by myself in three cases. In a recent monograph on brain tumors Bruns has pointed out the differential points between ataxia of cerebellar origin and ataxia due to tumors in the frontal lobes. He shows that in cerebellar ataxia hemiplegia very rarely develops, and if it does it usually is associated with some disturbance of the cranial nerves on the opposite side from the hemiplegia. There is no tenderness over the frontal region to percussion or pressure. Optic neuritis with hemorrhage into the retina occurs very early in the course of the disease and is attended by blindness. Vomiting, vertigo, nystagmus, and slowness of pulse develop early in cerebellar disease, while mental symptoms, such as dulness, apathy, and emotional disturbance, come on late. In ataxia or staggering gait due to frontal lobe lesion hemiplegia or monoplegia occurs early, and mental dulness or emotional disturbance may be among the first symptoms to appear. Ataxia or staggering gait, therefore, must be considered a general symptom of brain tumor, and its localizing value must depend largely upon its combination with other general or local symptoms. Another fact of interest in regard to the general symptoms of brain tumor which has been elicited by a more careful study of these cases within the past few years is their great variability; the attacks of vomiting and vertigo are always intermittent and occasional. The headache may also disappear for a considerable time, and often the optic neuritis may come to a stand-still for several weeks. The mental condition also varies from time to time. It has been supposed that this variability in the general symptoms is due to variations in the circulation in the brain or to a varying degree of cerebral œdema produced by the pressure of the tumor. I have noticed at operations that in some cases there was a great accumulation of serous fluid beneath the pia in the vicinity of, and at some distance from, the brain tumor, while in other cases the brain was remarkably dry and showed no evidence of œdema. The same fact has also been noticed at autopsies. It is, therefore, evident that from unknown causes tumors may produce a variation in the degree of serous effusion within the meninges and in the ventricles, and the most reasonable explanation, therefore, for this variability of the general symptoms is the varying condition of serous effusions within the cranium. It is well known that the general symptoms in tumors of the posterior cerebellar fossa are more intense and rapidly developed than those of tumors in the frontal lobes, and it is also known that distention of the ventricles by serous fluid develops earlier in such cases.

For a study of the local symptoms, spasm and paralysis, alterations of reflex action, paræsthesiæ, disturbances of touch, pain, temperature, and muscular sense, ataxia, hemianopsia, double vision and ocular paralyses, disturbance of equilibrium, and of the acts of swallowing, respiration, and articulation, vaso-motor disturbance, polyuria and glycosuria, defects of language and of memory, and symptoms referable to one or more of the cranial nerves, the reader is referred to the original article (vol. iv. p. 561). The statements in that article regarding local symptoms of tumors in various regions of the brain have received ample confirmation during the past few years, but it cannot be said that many new facts have been elicited. But little progress has been made in the local diagnosis of cerebral disease in the past eight years.

Two local symptoms not formerly described have been recently recorded. In tumors of the temporo-occipital region near the base of the brain lying just above the petrous portion of the temporal bone the disease destroys the association tract between the occipital and temporal regions. This produces a form of aphasia which has been called optical aphasia by some and visual amnesia by others. It is characterized by the following condition. The patient can understand what is said to him and can talk freely, though he avoids proper names and nouns, giving descriptions of things instead of naming them. Thus, he describes a knife or scissors as a thing you cut with. He is able to read and write, but when an object is shown him he is not able to give its name, although he recognizes the name when it is spoken. If an object is named he is unable to call the picture of it to his mind, though he recognizes the picture as distinct from other pictures when it is shown him. Thus, if a watch is shown to the patient, he is unable to remember the word watch, but will say "no" when asked if it is a key or a chain, and "yes" when asked if it is a watch. If he is asked to call to mind a vase he may be unable to do so, but if asked whether it looks like a clock or a match-safe he will say "no," and when a vase is pointed to he will assent, recognizing that as corresponding to the object named. This symptom implies a break in the association tracts between the occipital and the first and second temporal convolutions, and hence implies a lesion within the white matter of the temporo-occipital region upon the left side in right-handed persons, and upon the right side in left-handed persons. It is a symptom which has been more frequently noticed in abscesses of the brain secondary to ear disease than in tumors. This tract is shown in Fig. 1, which demonstrates the association tracts in the brain. It is marked *E*.

FIG. 1.



The association tracts of the brain. A, between adjacent convolutions; B, between frontal and occipital lobes; C, between frontal and temporal lobes, the cingulum; D, between temporal and frontal lobes; E, between occipital and temporal lobes; CN, caudate nucleus; OT, optic thalamus.

The second local symptom which I desire to record is a peculiar type of spasm occurring in cerebellar disease. I have noticed it in four patients who have had cerebellar tumors. The spasm is not a general convulsion, though there may be sudden twitching, symmetrical, bilateral, of arms and legs of slight extent, and a rigidity of the spine. It may come on in bed, and is more frequent early in the morning on waking. If it occurs when the patient is standing it results in a sudden collapse and fall, and the patient is unable to pick himself up, there being apparently a sudden suspension of the general sense of equilibrium. A spasm lasts two or three minutes and then subsides gradually with subsequent feeling of weakness and prostration which may last for several hours. Usually it is not attended by loss of consciousness, and it has never been preceded by any aura.

The diagram Fig. 2 shows the situation of the various functional regions of the convexity of the left hemisphere of the brain so far as they are at present determined. The diagram Fig. 3 demonstrates the tracts passing from the cortex downward to the internal capsule and to the spinal cord, the lesions of which produce various local symptoms, as described in the original article.

FIG. 2.

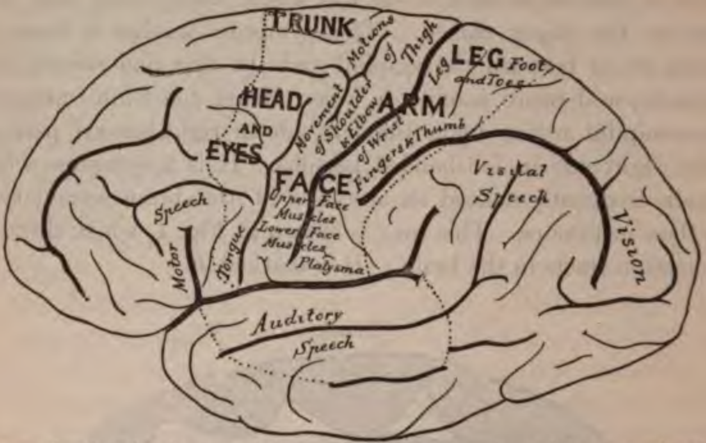


Diagram showing the situation of the various functional regions of the convexity of the left hemisphere of the brain.

FIG. 3

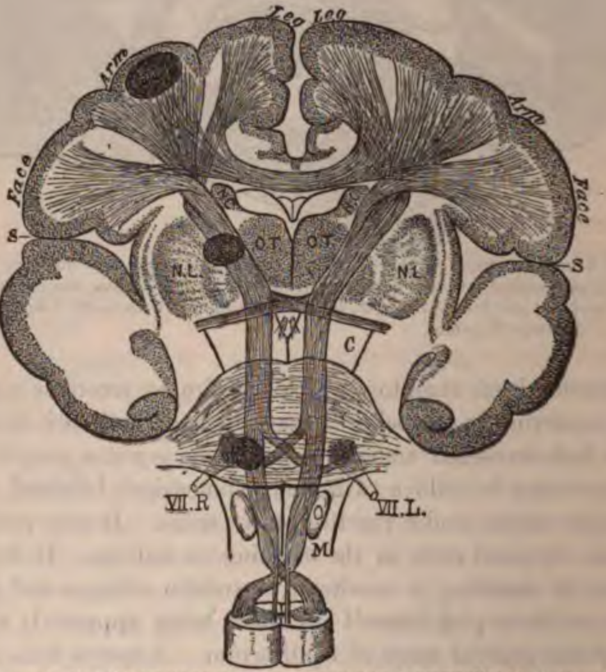


Diagram demonstrating the tracts passing from the cortex downward to the internal capsule and to the spinal cord.

The facts known with regard to local diagnosis have been utilized as guides to the surgical treatment of the disease. And it is particularly in regard to treatment by surgical interference that the original article published in 1890 requires additional statements.

In a disease so uniformly fatal as brain tumor any measure which offers the slightest hope is to be considered with favor, and although it is an undoubted fact that the experience accumulated during the past fifteen years, since the first tumor of the brain was removed, has taught great caution in giving prognosis regarding the result of such operations, yet it must be stated that in every case of brain tumor it is necessary to consider the question of the possibility of surgical interference.

It must be admitted that an operation is possible in only a small percentage of tumors of the brain. From a large collection of cases I think it may be stated with precision that about seven tumors out of a hundred can be successfully removed. There are a number of surgeons who have advocated the removal of a part of the skull in every case where a diagnosis of brain tumor is made, irrespective of the possibility of locating the position of the tumor or of removing it. The object of such an operation is to relieve the intracranial pressure produced by the growth of the tumor, and thus to mitigate the suffering produced by the headache and general symptoms. Park and Horsley, Norton, McCosh, Keen, Weir, and others have recorded cases in which a great amount of relief from these symptoms was obtained incidentally to an operation in which the tumor was not removed, and I can myself confirm this statement in several cases which I have seen. I should hesitate, however, to advise such an operation merely for the relief of symptoms, for it is not without danger in itself, it is not sure to decrease the amount of suffering, and its ultimate result can only be to prolong life. Diller and Albert have operated under these circumstances without producing the desired result. It has been stated by Horsley that such a relief of intracranial pressure may result in the cessation of the growth of the tumor, and he has recorded two cases in which the operation was followed not only by a subsidence of the symptoms, but also by a progressive and permanent recovery. This experience has been confirmed in a case of cerebellar tumor by Wilson.

The cases in which an operation is to be undertaken without hesitation are those in which the diagnosis both of the existence of the tumor and of its location in an accessible position can be positively reached. All cortical tumors, therefore, of the hemispheres are open to operation, and inasmuch as the local symptoms of tumors in the frontal, central, parietal, occipital, and left temporal convolution are comparatively clear, there is no reason why in any case in which a tumor is thought to lie in these parts an operation should not be undertaken. The local symptoms of brain tumor should, therefore, be very carefully studied in any case where a tumor is suspected, and for a consideration of these symptoms the reader is referred to the original article.

Granting that the tumor has been properly diagnosticated and properly located, the chances of a successful operation require consideration. There is no question that such operations are more liable to be successful in the hands of surgeons who have had some experience in dealing with the surgery of the head; thus the largest percentage of successful operations and of recoveries has been recorded by those who have had the largest experience. This might appear to be a truism, and yet in an operation whose delicacies are great, and whose dangers are many, it is a fact which cannot be too fully emphasized. The kind of operation undertaken in any case is a matter of importance. The American and German surgeons appear to have adopted the method of Wagner of opening the skull. This method consists in making a horseshoe-shaped groove with a small gouge in the skull after a similar incision through the soft parts, and then cutting through the skull along the groove with a sharp chisel, finally raising this flap of scalp and skull together by fracturing the short limb of bone between the ends of the incision. This method gives access to a large area of brain, and, inasmuch as many tumors are found to be greater in extent than was at first suspected, its convenience is evident. As the flap can readily be replaced and easily unites after the operation, no defect is left in the skull subsequent to the removal of the tumor. The time taken up in such chiselling of the skull is not longer than that required for a trephine opening and subsequent enlargement by rongeur, though this latter method is preferred by some operators. Horsley and some English and American surgeons prefer to remove a considerable square section of the skull by means of saws. Some surgeons have used the dental engine for this bony incision. If the bone is removed by trephine, by rongeur, or by saw, it is not replaced after the operation, and hence a defect in the skull remains, which is, however, as a rule, concealed by the growth of hair.

The chances of success in removal of tumors are largely influenced by the variety of tumor which is found. Sarcomata which are encapsulated and which are attached to the dura and simply compress the brain are those which are most easily removed, and in the list of successful operations where the patient has recovered entirely from the symptoms this has been the kind of tumor which has been taken out. Gliomata are occasionally encapsulated, as are also glio-sarcomata, and when the limitation between the tumor and brain tissue is quite distinct success has attended their removal. In many cases, however, there is no line of demarcation between tumor tissue and the brain in these two varieties of tumor. Under these circumstances it is sometimes possible to cut out a wedge-shaped section of the tumor, but in other cases where the vessels throughout the new growth are very numerous and very brittle it is impossible to attempt its division. Thus in two cases in which I have directed an operation it has been evident on exposing the brain that there was present a diffuse infiltrating and very vascular glioma without capsule, and in both these cases the hemorrhage consequent upon the manipulation of the surface of the brain has been alarming, and it has been impossible, on account of the hemorrhage, even to attempt a removal of the growth. Cystic tumors are easily evacuated, but unless the evacuation of the cyst is followed by an extirpation of its wall nothing permanent is accomplished. There are often sarcomatous elements in the wall of a cyst, and therefore recurrence of the tumor is to be expected unless the entire cyst-wall is removed. This operation is sometimes difficult on account of the hemorrhage. A few cases of angioma have been successfully treated by operation: all the vessels leading into the tumor have been tied, and then the tumor itself removed. Tubercular tumors are not frequently operated upon, as the prognosis for ultimate recovery is so bad, and it is not uncommon to find secondary tubercular meningitis or more than one tumor in such cases. Gummy tumors can often be absorbed by antisyphilitic treatment, and, as this treatment is usually given a trial before any operation is thought of, such tumors rarely require operative interference. A gummy tumor is generally so diffuse that its removal is particularly difficult. It is evident, therefore, that the prognosis in any case of operation for brain tumor cannot be positively given until the variety of the tumor is ascertained at the operation, and it is also evident that, but a small percentage (about one-sixth) of the tumors being of the sarcomatous variety, it is in but a few of the cases that success is to be expected.

In regard to the results thus far obtained from operative treatment of brain tumors, the following table displays the recent statistics:

Table of Results of Operations for Brain Tumor.

	CEREBRAL.	CEREBELLAR.	TOTAL.
Total number of cases operated upon for tumor of the brain	158	29	187
Cases in which tumor was not found	46	11	57
Cases in which tumor was found but removal was impossible	5	2	7
Cases in which tumor was removed but patient died	35	8	43
Cases in which tumor was removed and patient recovered	72	8	80

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ARSENICAL POISONING.—LEAD POISONING.

By J. J. PUTNAM, M.D.

Definition.—This review will represent the facts on the subject of chronic arsenical poisoning which have accumulated since the publication of vol. iv. of this work.

History.—The only portion of the history of arsenical poisoning which is of actual importance in this connection is that which relates to poisoning by this substance when used as a medicine and in the domestic arts. In the first edition references were given to the more important contributions, coming mainly from Swedish, English, and American sources, showing that all our accurate knowledge on these subjects has been accumulating during the past thirty years. We are fortunately able to add that the dangers there alluded to have considerably diminished within the past few years in consequence of the spread of knowledge and interest in the community and among manufacturers. This is especially true as regards the danger of poisoning from wall-papers. Cases of this sort, however, are still published from time to time, and it is to be recorded that a contribution of great importance has been made to our knowledge of the way in which this sort of exposure leads to harmful results through the researches of a number of chemists, especially Hamberg, Gosio, and Sanger. These researches have made it evident that volatile arsenical compounds, the existence of which had long been suspected, may actually be formed from arsenical wall-papers under the conditions in which they are found in our houses.

Etiology.—Besides the articles mentioned in the first edition of this work as being sources of possible arsenical poisoning, it is worth noting, if only for completeness's sake, that coal-dust and even the dust of streets contain minute quantities of arsenic. It is very improbable that actual poisoning occurs from this source, but it may be that this exposure helps to account for the frequency with which traces of arsenic are found in the urine of persons in health.

It has always been considered a mystery how any one breathing intermittently the air of a room hung with wall-paper containing a few grains of arsenic to the square yard, and that, perhaps, in such a form that the surface could not easily be detached, could absorb enough of the poison to be seriously affected, and the difficulty of forming an adequate explanation, coupled with the vague character of the symptoms in many of the recorded cases, has led many excellent observers to scout the idea that the interpretation offered was the correct one. Nevertheless, the circumstantial evidence seemed fairly conclusive, and the suggestion was heard from several quarters that we had, perhaps, in these cases, poisoning with a form of arsenical compound hitherto but little known; though, in fact, the researches of various chemists previous to 1892 had seemed to diminish rather than increase this probability. In 1892 Gosio published a series of experiments showing that various common moulds may grow on arsenical culture grounds and may set free arsenic in a volatile form. These researches were taken up afterwards by Professor C. R. Sanger, now of St. Louis, and his results were published in the Proceedings of the American Academy of Arts and Sciences, vol. xxix., 1894. The monograph contains a complete review of the previous work done on this subject, the more important part of which is also referred to in a paper by Dr. F. C. Shattuck, published in the *Boston Medical and Surgical Journal*, vol. cxxviii. p. 540, which gives an excellent review of the whole subject from the stand-point of the practical physician.

In brief, the facts as developed by these investigations are as follows: A variety of common moulds (Penicillium brevicaule, Mucor mucedo, Aspergillum virens, and Aspergillum glaucum) find conditions favorable to their growth on organic substances containing arsenic in small or moderate quantity in the presence of moisture and of a temperature of from 60° to 95° F. Under these circumstances a volatile compound of arsenic is set free the nature of which is so far unknown, though it may be a derivative (arsenic pentoxide).

It is not claimed that these investigations set entirely at rest the doubts as to the occurrence and exact character of wall-paper poisoning, but it seems not improbable that we cannot directly transfer the knowledge of the absorption, elimination, and action of better known arsenical compounds to the case of the comparatively unknown volatile substance. A possible explanation is also afforded by this discovery of the fact that papers containing small quantities of arsenic sometimes appear equally important as sources of poisoning with those containing larger quantities.

The more recent facts with regard to arsenical medication as a cause of poisoning will be referred to under Symptomatology, in so far as they concern the cases of children.

Symptomatology.—In spite of the diminished danger from poisoning by wall-paper and fabrics, due to increased care on the part of the manufacturers, new cases are still from time to time reported. Such a case is described by Henrik Berg.[1] The patient was a male infant of ten months, who, in spite of good care, suffered from progressive impairment of nutrition, emaciation, thirst, and had foul and frequent discharges. Sores appeared around the mouth. The wall-papers were found to be strongly arsenical, but in spite of the fact that the family moved out when this discovery was made, the child grew worse and died. No autopsy was allowed. The mother, father, and a sister of the mother are said to have presented symptoms which could be explained by chronic arsenical poisoning and seemed to be related to occupation of the suspected room.

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[1] Eira, xvi. 10, 1892. Abstract given by Walter Berger, Jahr. f. Kinder., Psych. Erziehung, Bd. xxxv., 1893, S. 339.

Dr. F. C. Shattuck, in the paper above alluded to, cites a series of cases observed in an infant asylum by Dr. C. P. Putnam. The symptoms consisted of progressive anæmia, suppuration of the fingers, slight discharge from the ears, and bronchitis, ending in two cases in death. Sores also occurred on the fingers or elsewhere among the nurses. It was finally discovered that all the nurses in the asylum were wearing dresses made of a blue cloth provided by the institution. This cloth was found to contain much arsenic, and the dresses were thoroughly washed and then resumed, but symptoms similar to those described, which had diminished, again reappeared. The dresses were finally discarded, and the symptoms disappeared and have never since returned.

Dr. Shattuck also cites the cases of two vigorous boys, brothers, who had been confined to the house by slight illnesses shortly after their return from the sea-shore. Immediately after this they began to have recurrent attacks of nausea and vomiting, as did also another brother who had been previously well. Arsenic was found in considerable quantities in the wall-paper of the hall, entries, and stairways on every story, and also in that in five of the chambers, though not in the one commonly occupied by the boys. Recovery soon took place after removal of the paper.

Lancereaux[1] reports the case of a young girl of thirteen who took medicinal doses of arsenic under the form of either Fowler's solution, or sodium arseniate, for three years as a part of the treatment of a universal psoriasis. Towards the end of this time the following symptoms set in: a sense of fatigue, fever with evening exacerbation, and numbness of the toes, and finally paralysis associated with pains of neuritic character. At the same time the appetite failed, emaciation began, and the skin lost its healthy tint and became gray, wrinkled, and scaly. Finally the legs became semi-flexed on the thighs, the toes on the feet, the feet themselves becoming œdematous, and the nails brittle and thick. The other extremities were very much less affected, but showed, nevertheless, signs of neuritis. There were no serious digestive symptoms beyond the failure of appetite. After appropriate treatment her recovery occurred. Lancereaux remarks that cases of arsenical poisoning with fever are very rare, but I have recorded several such as occurring among wall-paper cases.

These cases indicate very correctly the range of symptoms observed in chronic arsenical poisoning, and it is only necessary to refer in addition to the general classification of symptoms given in the previous edition of this work.

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[1] Dangers de la Médication par l'Arsenic, La Médecine Moderne, 1896, No. 60. Abstract by Gaston Bresson in Revue Neurologique, October 30, 1896, p. 622.

It is well known that typical instances of poisoning and even neuritis are occasionally seen among children treated with arsenic for chorea. Fortunately, these cases are rare, even when large doses are employed, as is shown, for example, by the statistics of Comby.[1] It is said that larger doses are borne when the arsenic is given by subcutaneous injections than when taken by the stomach; but if we accept the view that the digestive disorders are due to chemical irritation, it is difficult to believe that this can really be the case.

Prognosis.—The prognosis of chronic arsenical poisoning is good if the patient is removed from exposure. A severe neuritis, however, is always recovered from very slowly.

Treatment.—It is unnecessary to make any further statements in this regard than those given in the first edition. The essential indications are rest, good hygienic measures, and the use of the treatment suitable to neuritis in general if this is present.

LEAD POISONING.

Definition.—Chronic poisoning by lead or its salts.

History.—It is not necessary in an article coming within the scope of this work to review the large subject of the history of lead poisoning in so far as it relates to adults whose occupations expose them to the action of lead. Since the first edition of this work was published a number of important cases of lead poisoning in children have been reported, the references to which will be given under Symptomatology, and by means of them our knowledge of that subject has been considerably enriched. The more important series of facts mentioned in the first edition are the following:

1. Constantine Paul reported in 1860 a number of facts showing that the children of lead-workers are subject to various forms of nervous disease, and that infants are prone to die early in convulsions, or to show evidences of seriously impaired nutrition. Later Roque and Berger and others confirmed these statements, and there has been some evidence that even where parents are poisoned with drinking-water, the same result may occasionally ensue.

2. It was pointed out that children enjoy a certain degree of immunity from lead poisoning, as compared with adults, perhaps because they eliminate more rapidly, and that typical paralysis is comparatively rare among them as compared with cachexia, convulsions, and other nervous disorders.

3. It was also noted as probably true that when paralysis does occur the legs are quite as likely to be affected as the arms, and may be attacked earlier and more severely, thus giving a clinical picture which is extremely rare in adults. Later observations have not only confirmed this statement,

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[1] Congress of Medicine, Nancy, 1896.

but have shown that this form of paralysis is typical and almost invariable.

Etiology.—This part of the subject was quite fully treated in the earlier edition. The most fruitful source of harm is still the careless use of lead service-pipes for drinking-water, in spite of the frequency with which public attention has been called to this danger. A number of well-marked cases from towns in Massachusetts have been reported to the State Board of Health within the past year, and I have personally examined several patients from two localities. It is not realized how rapidly the soft waters, and especially those containing vegetable acids, will dissolve lead from pipes, nor how long it takes to wash the lead solution out. The probability is that for a pipe of a hundred feet half an hour is not too long to let the water run. The layer of water which lies next the pipe adheres more or less strongly, and this is probably especially true where the surface is rough. Dr. C. P. Worcester, the chemist of the Massachusetts State Board of Health, has nearly completed an analysis of drinking-waters from all the towns of the State, the results of which will be made public in the next report.[1]

The Board has also published a report[2] of the analysis of the metallic stoppers of bottles in which carbonated waters and syrups are put up, its attention having been called to the fact that cases of lead poisoning were traceable to this source.

This investigation shows that these stoppers contain from three to fifty per cent. of lead. The contents of all the bottles were found to contain traces of lead, the largest amount found being equivalent to $\frac{28}{100}$ grain per gallon.

Pathology and Pathological Anatomy.—It is only necessary to add to the statements made in the earlier edition that the more recent investigations, which the newer methods of histological research have made possible, show that lead ranks definitely as one of the causes of parenchymatous degeneration of the nerve elements of which the nervous system is composed. It has also become more than probable that the disease which has hitherto been called "peripheral neuritis," due to lead, should really be considered a neuron degeneration due, in part at least, to changes in the trophic nerve-cells of the spinal cord. It is not, however, to be denied that the distal portion of the neuron may become diseased independently.

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[1] I quote the following, by permission, from a personal letter written by Dr. Worcester, February 22, 1897:

". . . It will hardly be possible to anticipate any of the figures, but in general it may be said that the commonly accepted opinion that after a short time a new lead pipe in water-service becomes coated with an absolutely insoluble protection of oxide, and that thenceforth it is perfectly harmless, must be revised.

"It is true, so far as our experience goes, that a bright lead pipe is tarnished by every natural water, the surface deposit differing in quantity and composition according to the variety of the impurities carried by the water. But that this coating is a perfect protection to the underlying metal, or that it is absolutely inert itself, and is not washed along by ordinary usage, does not prove true."

[2] Weekly Bulletin, State Board of Health, Massachusetts, December 12, 1896.

Symptomatology.—In the earlier edition attention was called to the fact that relatively few cases of typical lead poisoning had been reported among children. Since the publication of that article the following instances have come to my notice:

I. Variot and Gastou[1] report the case of a child one and a half years old who was in the habit of playing in warm water on a lead roof, and for several years suffered through the summer from vomiting, colic, and constipation. Other children who were exposed in the same way had similar but slighter symptoms. Two of the children had a lead line on the gums, and two dogs who drank rain-water running from the roof died with paralysis of the posterior extremities.

In June, 1891, the child first mentioned was attacked with abdominal pain, vomiting, and constipation, also with acute pain in all four limbs and general loss of strength. The weakness of the extensors of the thighs and legs became so great that the child could straighten himself only by bracing the arms against the thighs. A little later the arms became paralyzed to a greater or less degree. The liver was found enlarged.

II. Wharton Sinkler[2] reports the cases of three children, all from the same family, the father being a house-painter and having his shop in the house in which he lived. The parents are said to have been healthy and to have had nine children. Four of these are reported as having been strong and well, but one died of convulsions at three years of age, and another of "spinal meningitis" at five. The histories of the other children are in brief as follows:

Case I.—Girl ten and a half years old. She is said to have been well up to June, 1889, when she awoke in the morning with loss of power in the legs, associated with pain. Four weeks later the arms became weak and the muscles wasted. There was fever for a time, and some vomiting and constipation, but no colic. Under suitable treatment she recovered to a great extent, but a month later the symptoms set in again with still greater violence, and then complete wrist- and ankle-drop were found, associated with an extensive paresis of the other muscles of both legs and arms. The muscles most strongly affected exhibited the degenerative reaction on electrical examination.

Case II.—Boy six years old. He began to cut his teeth at the third month of his life, and remained apparently well until six months old, when he had convulsions which lasted three days. When sixteen months old he had enterocolitis. The more serious symptoms began in November, 1891, with fever, colic, and constipation. These were followed by paresis of the arms and legs, involving the muscles quite extensively. There was also atrophy characterized by the reaction of degeneration in the muscles most strongly affected. The patient improved, but even three months later he was far from well, and the thenar and hypothenar muscles were still atrophied.

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[1] Bull. et Mém. Soc. Méd. des Hôpital de Paris, 1891, vol. viii. p. 505.

[2] Transactions of the Association of American Physicians, Philadelphia, vol. ix., 1894, pp. 133–143; Medical News, Philadelphia, 1894, pp. 85–89. (The article contains a valuable summary of reported cases.)

CASE III.—Boy three years old. He was said to have been healthy as an infant, but not to have begun to walk until two years old, at which period it was noticed that his right leg was weak. The history was the same as that of the brother, except that the case was less serious; but even two years after the onset of the typical symptoms, and in spite of thorough treatment, the recovery was far from perfect, and the reaction of degeneration was present in the extensors of the right foot.

Dr. Sinkler remarks that neither of these children showed any blue line on the gums, and that, although they were thin and pallid, there was no distinct saturnine cachexia.

This interesting series of cases bears out the statements that the children of parents poisoned with lead start in life with impaired vitality, and also that the paralysis in children affects the lower extremities as much and as quickly as the arms.

It would be interesting, if we had the power, to make a searching investigation into the health of such patients before typical symptoms showed themselves. In my opinion, there is no doubt that the microscopic examination of the affected nerve-cells, and even fine clinical tests, would give indication of disease, and this opinion is borne out by the researches made many years ago by Gombault, who found degenerative neuron changes in guinea-pigs poisoned with lead, in spite of the fact that they had exhibited during life no signs of illness.

III. In this connection a case studied at Oppenheim's polyclinic and reported by Anker[1] is of great interest. The case was that of a boy who at the time of examination was eight years old, and was said to have developed normally up to his third year, at which time he began to show signs of progressive dementia following a fall on the back of the head. It appeared that the father, who was a type-setter, had suffered repeatedly from lead colic, but the child had not been exposed to lead so far as could be ascertained.

The observers believed that the disease was actually inherited. The chief symptoms were dementia, anæmia, and tremor of the facial muscles. The extensors of the feet and toes were paralyzed, the tibialis anticus being less affected than the extensores digitorum. The extensors of the hands and fingers were paralyzed, except the supinator longus, and also the interosseous muscles and those of the thenar eminence. The peroneal paralysis was complete and persistent, but the muscles of the arms and hands regained their power. No lead was found in the urine. The knee-jerk was exaggerated, a symptom which I have observed and reported as occasionally seen even in adult cases.

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[1] Berliner klinische Wochenschrift, Bd. xxxi., 1894. Abstract by Eisenschitz, in Schmidt's Jahrbuch der Medicin, Bd. ccxliv., 1894.

In spite of the interest of this case, it is difficult to believe, in view of the modern biological opinions, that the lesions suffered by the parents were transmitted as such, or that if the blood of the mother had conveyed the poison to the child the outbreak of the symptoms could have been so long delayed. An undetected exposure on the part of the child itself seems more probable.

IV. Newmark[1] reports the case of a girl seven or eight years old who was poisoned by white lead paint, home-mixed, covering an iron bedstead in which she slept. The paint was not varnished, and peeled off easily. The symptoms consisted at first of vomiting, constipation, colic, foul breath, sore mouth, and eventually of paralysis. She was in bed for three weeks, and at one time was unable to walk, and presented wrist- and ankle-drop on both sides, as well as a lead line on the gums. The arms, as in most of these cases, recovered before the legs.

V. Caton[2] reports a drinking-water case where the patient was a boy eleven years old. The case was in all respects typical, except that there was no lead line on the gums, and here, as in all the cases of lead paralysis in children which have been reported, the lower extremities were affected as well as the upper. Albumin and lead were found in the urine. The patient recovered under appropriate treatment.

VI. A case reported by Pässler[3] is important, because the poisoning was thought to result from the external use of diachylon ointment over an excoriated surface. The patient was a child a year and nine months old, with extensive eczema, which was treated by freshly made ointment spread on plaster. The first application was made July 4, 1893, and it was renewed on July 6. On July 7 the child seemed dull and apathetic, and the limbs were œdematous. The urine was very scanty and of a dark color. The ointment was washed off with warm water, and the applications were not renewed after this date. On July 8 the urine was very scanty and found to contain albumin, blood, and hyaline and granular casts. Stomatitis also showed itself. On July 12 the child seemed well, but the urine, though secreted in much larger quantity, still contained the same elements as before. The heart was dilated towards the left. By the end of the month the œdema had gone and the signs of nephritis were less marked, though still present. By the end of August the child was well.

I think it possible that the acute nephritis may have been due to some other cause than lead, and report a few cases where it has followed severe eczema. Nevertheless, in view of the infrequency of this sequence, the presence of the stomatitis, and the subsidence of all signs after abandonment of the diachylon ointment, I think it most probable that lead poisoning was the real cause, in spite of the fact that no lead was found in the urine.

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[1] Medical News, Philadelphia, 1895, vol. lxvi.

[2] Liverpool Medico-Chirurgical Journal, January, 1895. Abstract by Mettenheimer, Jahrb. f. Kinderheilkunde, Band xl., 1895, S. 322.

[3] Münchener medicinische Wochenschrift, Bd. v., 1894.

VII. It was noted under Etiology that a number of striking cases of poisoning from the drinking-water had been observed in Massachusetts within the past year. The most important of these, so far as children are concerned, is the following:

The patient was a girl of eleven who was recently referred to the Massachusetts General Hospital for diagnosis by a physician of a town within the State. The family history was unimportant, except that three or four brothers had died of tubercular disease.

Up to her ninth year the patient had had no important illnesses other than scarlet fever and whooping-cough. In July, 1894, she was taken with fever and vomiting and was confined more or less to her bed for three weeks. By the end of this time she had become weak, though not distinctly paralyzed, and it was thought that she had malaria. In September of the same year she had a similar attack lasting about one week, and in December a third attack in which she was feverish and vomited more or less and suffered from joint-pains of a sharp, cutting character. At this period foot-drop was first noticed, and soon afterwards the legs became so weak that she was unable to walk without assistance. The pains about the joints persisted and were not relieved until a year later, and abdominal pain was also present from time to time. Atrophy of the muscles was first noticed after the third attack.

The patient was examined by my colleagues and me at the Massachusetts General Hospital in January, 1897. She was found poorly nourished and with an expression of anxiety and feebleness. The pupils were mobile; the movements of the face normal. The arms and chest were mottled, presenting an appearance as if the blood had settled in the superficial veins. The extensors of the fingers and hands were paretic, and the fingers were habitually flexed. The thenar, hypothenar, and interosseous muscles were atrophied. The muscles of the arm were, in general, small and feeble, though all movements at the wrist, elbow, and shoulder were possible to some extent. The lower part of the chest was flattened from side to side, and the sternum was somewhat prominent. The legs were thin and cold. Movements at the hip- and knee-joint were possible but feeble. The feet were fixed in the equinus position, so that even passive dorsal flexion of the foot and toes was impossible.[1] Plantar flexion, though feeble, could be performed so far as the rigid condition of the tissues about the joint would permit. The feet were cold, the skin dry and mottled as on the chest. There was a strongly marked lead line upon both the upper and lower gums, inside as well as outside. The mother also had a lead line, and it was found later that the same was true of another child in the family who had suffered from ill health, though not paralyzed. Examination of the heart revealed an accentuation of the second aortic sound, and a low-pitched systolic murmur was heard over the aortic area. The knee-jerks were present but weak. The urine contained albumin and renal casts.

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[1] Eventually a tenotomy was done to relieve this contracture, and with considerable benefit.

An examination by the chemist of the State Board of Health of a sample of water which had been standing eleven hours in the pipe used by this family showed 0.295 part of metallic lead to 100,000 parts of water, or 0.17 grain per gallon. The water was supplied by the town, and iron pipes were used from the street to the house, but those in the house were of lead.

It will be noticed that in this case, as in several of the others, the symptoms were ushered in by fever.

Prognosis.—A glance over the cases here reported renders it sufficiently clear that the prognosis in severe cases of lead poisoning is by no means always favorable so far as complete recovery from paralysis and atrophy is concerned.

Diagnosis.—The diagnosis of a well-marked case of lead paralysis should be sufficiently easy to any one acquainted with the disease as it occurs in adults, among whom it is much more common than with children. On the other hand, where paralysis is absent and the lead line is also wanting, as sometimes happens, and where, in short, the symptoms are those of anæmia and prostration, with perhaps attacks of feverishness and indigestion, there may be little to call the physician's attention to the true nature of the case. Persistent colic is suggestive, but it is unquestionable that colic is a sign of neuralgia to which some children are subject independently of lead poisoning or other obvious causes.

A careful scrutiny of the conditions of water-supply made under a recognition of the fact above noted, that a piece of lead pipe must be emptied many times over before the lead water is entirely washed out, may help to arouse suspicions, and careful investigation of the health of other members of the family or neighbors using water-supplies in a similar manner may strengthen these suspicions.

It is, however, amazing how ready both physicians and intelligent laymen are to deride the significance of this danger.

Treatment.—It is not necessary to add anything to the remarks on treatment given in the earlier edition. It is, however, to be remembered that the use of potassium or sodium iodide sometimes precipitates new outbreaks of symptoms, though indeed these may occur independently of this immediate cause and after the patient has been wholly withdrawn from exposure. Iodide should therefore be used cautiously. The persistent use of strychnine, cod-liver oil, and arsenic from time to time for short periods is indicated for the relief of paralysis. Electricity, massage, and orthopædic measures are of prime importance.

MYELITIS, MENINGITIS, AND SPINAL HEMORRHAGE.

BY MARY PUTNAM JACOBI.

WITHIN the present decade the medical aspect of the above group of diseases has been changed or enriched by:

The introduction of operative measures for the relief of compression myelitis; the establishment, through clinical observation and experimental research, of the infectious origin of many cases of myelitis; the demonstrated importance of vascular lesions in myelitis, and the relations of these on the one hand to generalized infections, on the other to the anatomical peculiarities of the spinal blood-vessels; the influence of hereditary syphilis upon spinal cord disease; the demonstration of the reality of primary hæmatomyelia, and the more accurate exposition of its clinical symptoms.

The first operations on the spine, apart from cases of fracture, were made not to cure compression myelitis, but to evacuate tubercular abscess, or to remove carious sequestra from the accessible posterior regions of the vertebral column, the spinous processes and laminæ.[1]

In 1882 Jackson performed this same operation for a different purpose, —namely, with the hope of freeing the cord from compression presumed to be exercised by confined masses of caseous tubercle, and to which should be due a flaccid paraplegia. No compressing mass was found, and the result was imperfect.[2] But between this date and 1890 thirty-five cases of laminectomy for paraplegia were published and summarized in Chipault's essay.[3] Among these are seventeen cases of Pott's disease in children.

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[1] Lannelongue, 1878, Bull. Soc. Chir.; Boeckel, 1880; Polaillon, 1883, Union Médicale; Reclus, 1884, Cliniques Chirurgicales, and Gaz. Hebd., 1882–1887; Delorme, 1887, Bull. Soc. Chir. (All quoted by Chipault, Arch Gén, Paris, 1890.)

[2] Brit. Med. Journ., 1883, vol. i. p. 812.

[3] Loc. cit. These are: Macewen (five cases), Brit. Med. Journ., 1888; Southam, ibid., 1888, ii. p. 665; Wright, Lancet, July 14, 1888; Duncan, Edinburgh Med. Journ., 1889; Deaver, Am. Journ. of the Med. Sci., 1888; Abbe, N. Y. Med. Record, July 26, 1890, eight cases of spinal surgery for fractures or tumors, and two for intractable neuralgias; Gerster, ibid.; Horsley, seven cases, unpublished (quoted by Chipault); Chipault, two cases, loc. cit.; Lloyd's essay on laminectomy, in Am. Journ. of the Med.

In 1891 Lane published the records of eleven cases, of which seven were in children.[1] In 1894 Parkin published six other cases, all in children.[2] These recent reports, therefore, include thirty cases of laminectomy in children for paraplegic caries, to which we may add the four cases in adults operated on by Lane for the same condition, thus thirty-four in all.

Of these cases twenty recovered completely; in three a relapse of the paraplegia occurred in from two to four months; in seven cases, although the patients survived the operation, the paraplegia was little or not at all benefited; four cases died, shortly after the operation, from exhaustion, asphyxia, or pulmonary congestion.

In six cases abundant caseous material was removed; in nine cases pus was evacuated; in six, solid fibrous or leathery masses were raised from the cord they compressed.

Surgeons hesitate to perform laminectomy in vertebral caries until every other resource shall have been exhausted, and yet review of the operations already performed suggests that an early evacuation of a tubercular abscess would avert the development of a compression myelitis, or arrest it before it had progressed so far as to be incurable. As the profession becomes more familiarized with the operation, this is the practical conclusion that will probably be reached.

INFECTIOUS MYELITIS.

Until recently acute infections invading the nerve-centres were supposed to expend themselves principally upon the meninges. In 1895, however, at the Congress of Internal Medicine held at Bordeaux, Grasset made an elaborate report upon infectious myelitis. The occasional occurrence of myelitis as a sequel to infectious diseases had already been noted. In 1827 Ollivier d'Angers reported a case after pneumonia, and Gull reported another in 1856. Gubler in 1860 reported several cases of myelitis occurring after various forms of infectious disease. In 1867 Delioux de Savignac observed two cases after dysentery; Roger and Damaschino, two cases after variola and dysentery; Oertel, cases after diphtheria; Jolly, after erysipelas; Westphal, after pneumonia and variola.[3]

But the elaborate report by Grasset has formally introduced the subject to the profession, and given an aspect to acute myelitis which especially affects cases occurring in children.

Sci., 1891, relates to traumatic cases; Demons, Clin. Chirurg., Bordeaux, 1889; White, Annals of Surgery, 1890; Thompson, Lancet, 1889, ii. p. 315; Burrell, Boston Med. Journ., October, 1889, p. 405; Lane, Brit. Med. Jour., April, 1889, i. p. 884; Ibid., Lancet, July, 1890, ii. p. 11; Wyeth, N. Y. Med. Journ., March 8, 1890; Colman, Lancet, February 22, 1890; Kraske, four cases, Congrès Chirurgic. Allemande, April 12, 1890.

[1] Trans. Lon. Clin. Soc., 1891.

[2] Brit. Med. Journal, 1894.

[3] J. Crocq fils, Presse médicale Belge, Mai 31, 1896.

The cases quoted by Grasset may be summarized as follows:

After variola	7
After typhoid	1
After gonorrhœa	2
After erysipelas	2
After dysentery	5
After pneumonia	5
After influenza	2

In the variola, dysentery, and influenza cases the myelitis, diagnosed clinically, was demonstrated by autopsies.

Gowers ascribes to influenza various cases of acute myelitis, transverse, disseminated, or limited to the anterior horns.[1] Such a case is related in detail by Sansom. The patient suffered from motor paresis of all four limbs, tumor of hands, incoördination of upper but not of lower extremities, some wasting of hand muscles. No tenderness, no rigidity, no alteration of sensibility. Preservation of superficial reflexes, and fair amount of faradic contractility, diminished reaction to galvanism, exaggeration of deep reflexes. The patient recovered after treatment by arsenic and bromide and iodide of potassium.

Mackay reports a case of cervical meningo-myelitis following influenza and demonstrated by autopsy, death occurring after an illness of four months. There were motor paresis of all four limbs, uniform wasting, especially in upper extremities; diminished faradic and galvanic irritability, especially in arms; absence of rigidity, but ankle clonus and increased knee-jerks and plantar reflexes; pain in neck, increased on movement; numbness and some tactile anæsthesia in tips of fingers, and analgesia throughout hands; scattered patches of hyperæsthesia over limbs. At the autopsy the cervical dura was found adherent to the vertebral bodies and covered with a fibrinous exudation one-sixth inch thick. The cord was too soft to be hardened, but the softened tissue contained the usual myelitic débris.[2]

In contrast with this acute infectious softening are the cases of spinal sclerosis after typhoid fever reported by Marie.[3] The sclerosis is said to be due to a disseminated inflammation of blood-vessels which is the primary lesion caused by the infection. Spinal sclerosis so caused cannot, observes Marie, be properly considered a disease of the nervous system; it is the medullary localization of a generalized vascular infection.

Infectious alterations of blood-vessels have been studied in the brain more than in the spinal cord, but the process is undoubtedly the same in both parts of the cerebro-spinal axis. Irritating substances are carried by the blood-current from the focus of the primary disease and become accidentally attached to the walls of some spinal blood-vessels.[4] In fatal cases of typhoid fever Popoff found accumulations of lymphatic cells in the perivascular spaces of the cerebral blood-vessels, and similar lesions in the cord may be supposed to explain the symptoms of typhoid myelitis.

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[1] Lancet, July 8, 1893.

[2] Lancet, August 1, 1891.

[3] Progrès Méd., 1884.

[4] Ribbert, Virch. Archiv, 1882, Bd. xc.

The spinal symptoms may appear at some distance from the *début* of the fever, thus during convalescence, even when this has been delayed for three months.

Although Ollivier d'Angers had reported his case of pneumonia myelitis as early as 1827, the observation did not lead to any general theory of infectious myelitis, nor could it do so until pneumonia itself had been ranked among the microbic diseases. Grasset's voluminous text-book on nervous diseases, published in 1886, omits all infections but syphilis from the etiology of spinal cord disease. The individual cases which I have found reported since infectious myelitis has been definitely recognized have all been in adults. But in 1895 Sachs refers to the "myelitis developed in cases of a toxic character;"[1] and in 1897 Emmett Holt observes that "myelitis in children, a rare disease apart from compression myelitis and acute poliomyelitis, usually results from injury, but may occur as a complication of any of the acute infectious diseases, especially typhoid or scarlet fever and diphtheria."[2]

Thus infectious myelitis has fairly entered the domain of the systematic text-books, so much so that a certain doubt attaches to any cases alleged to be "idiopathic."

Within the last few years numerous experiments have been made upon animals submitted to the influence of microbic toxins directly injected into the blood. In these experiments myelitis is induced with such facility that we must infer a much greater susceptibility to the poison in these lower animals than in man.

Injections have been made, first, with completely aseptic emboli; second, with non-specific pathogenic organisms; third, with cultures from various infectious diseases.

Lamy[3] experimented on the dog and the cat. He used two kinds of powders,—the finest lycopodium, capable of penetrating the smallest arterioles, and a coarser powder, vanilla grains, able to block the principal spinal arteries. These powders were suspended in artificial serum, sterilized, and injected into the aorta. The finest proved incapable of producing profound or durable lesions. The coarser powders engaged chiefly in the arteries of the anterior system, and when the embolism was massive caused a corresponding focus of softening in the cord. If the embolisms were discrete, lesions were confined to the ganglionic cells. These in a few hours after the injection had in great numbers become unrecognizable. After twenty hours they had shrivelled and were greatly diminished in volume. Coloration by Nissl's method with methylene-blue showed a complete alteration of structure. The chromatophilic corpuscles which normally surround the nucleus had disappeared. In their place the centre of the cell was occupied by a sharply tinted homogeneous mass, irregular in outline, and apparently constituted by an agglomeration or precipitation of chromophilic elements. The periphery of the cell was paler than normal, and in many places the cell-substance had disintegrated, leaving vacuoles. The nucleus was quite destroyed, the cell-prolongations broken. These experiments are essentially analogous to those in which the vascular supply of the cord is interrupted by ligature of the aorta. Sprouck[1] passed a curved needle carrying a ligature through the intact skin of a rabbit around the aorta and the spinal column. The ligature was left for an hour, and the animal allowed to live twenty-four hours or longer. A very rapid effect was produced on the ganglionic cells, and also on the myeline fibres of the anterior horns. The fibres of the posterior horns remained intact, because the fibre degeneration was always secondary to that of cells, and the trophic cell-centres of the posterior horn fibres lying in the posterior spinal ganglia remained uninjured, owing to the freedom of the collateral circulation.

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[1] Nervous Diseases of Children, B. Sachs, p. 320.

[2] Diseases of Infancy and Childhood, L. Emmett Holt, p. 766.

[3] Comptes Rendus Soc. de Biol., Juillet 25, 1896.

The affected cells lose their protoplasmic prolongations (dendrons), while Deiters's prolongation (the neuraxon) persists to the last, first becoming varicose near the body of the cell. The nucleus wastes, then the mass of the cell protoplasm becomes a heap of fine granules, and finally disappears as if wiped out by a brush. The axis cylinder of the nerve tubes in the white matter swells and becomes varicose; an active proliferation of neuroglia cells occurs.

This class of experiments, which show the marked effect on the nutrition of nerve elements of even a transient interruption to their blood-supply, must be carefully considered in estimating the influence of toxic injections. For if the latter exercise a specific chemical action, they are also liable to interfere with nerve nutrition by inducing mechanical obstructions in the circulation.

The second class of experiments have been made with cultures from specific infectious diseases. Vincent[2] inoculated the ear of a vigorous rabbit with three-fourths of a cubic centimetre of a fluid containing the typhic bacillus of Eberth and one-third of a cubic centimetre of an unknown bacillus obtained from the spleen of the same subject. During the following days the animal suffered from intense diarrhœa, complete anorexia, stupor, and high fever,—a temperature of 41.9° C. Began to recover on the tenth day; was completely well in a fortnight. *Then* appeared a progressive weakness of the hind limbs, with atrophy of their muscles, extending anteriorly. There was great diminution of faradic contractility, with persistence of galvanic contractility. The rabbit died in two weeks. At the autopsy was found no visceral lesion, nor macroscopical alteration of the cord, but microscopic examination demonstrated an almost complete disappearance of the cells of the anterior horns, less in the posterior.

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[1] Arch. de Phys., 1888.

[2] Archives de Médecine Expérimentale, 1893.

Many that remained were extensively vacuolated, or showed a protoplasm retracted around the nucleus. The cylinder-axes of the nerve-fibres in the anterior columns were pale and tumefied. The neuroglia cells were multiplied; the nuclei in the endothelium of the capillaries proliferated. There was no exudation.

The maximum alterations were in the lumbar cord. There was a proliferation of nuclei in the neurilemma of the anterior roots. No alteration in the cells of the spinal ganglia. There was slight neuritis of sciatic nerves; loss of transverse striation in the atrophied muscles.

This experiment was very successful, but other experiments, exactly similar except for some unknown factor of organic resistance on the part of the animal, failed.

In the successful experiment no microbes were found in the cord, and the myelitis could not, therefore, be attributed to a multiplication *in situ* of the inoculated bacilli, but rather to a late poisoning by the soluble poisons secreted by the inoculated germs.

Bourges[1] determined an acute myelitis by inoculation of a very attenuated culture of erysipelas cocci.

Crocq[2] inoculated rabbits with the Loeffler bacillus of diphtheria and found degeneration of the spinal ganglion cells and sclerosis secondary to that. White substance rarely affected.

Enriquez and Hallion[3] made upon three dogs a subcutaneous injection of filtrated diphtheritic bouillon, in the proportion of one and a half to two cubic centimetres per kilogramme weight of animals. The dogs succumbed in ten days, and multiple foci of myelitis were found in their spinal cords. Thus the effect of the bouillon was much more pronounced than that of the pure Loeffler bacillus.

Vidal and Bezançon[4] inoculated one hundred and sixteen rabbits with streptococci cultures derived from patients suffering from various infectious diseases,—erysipelas, variola, puerperal fever, diphtheria. In addition some streptococci were taken from the normal mouth. The cellular tissue of the ear was inoculated with one and a half cubic centimetres of the culture fluid. Of the one hundred and sixteen animals, seven, or six per cent., became paralyzed at periods varying from seven days to two months after the inoculation. Death followed the paralysis at periods varying from thirteen to seventy days. The paralysis was sometimes flaccid, sometimes attended by muscular rigidity and contractures. Muscular atrophy was constant. Four autopsies were made, and discovered lesions of cells and nerve-fibres, while the neuroglia remained unaffected. The cells were hypertrophied, rounded, granular, with their processes broken off, or else in vitreous or vacuolar degeneration, all finally atrophying. The blood-vessels were engorged, especially in the anterior horns. In the white substance the myeline degenerated, and the axis-cylinder showed degenerative hypertrophy. Large granular bodies charged with myeline were scattered through the tissue.

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[1] Quoted by Vincent, loc. cit.

[2] Centralblatt für allg. Pathol., November, 1896.

[3] Revue Neurologique, 31 Mai, 1894.

[4] Annales de l'Institut Pasteur, 1895.

Only two lesions were lacking to complete the picture of acute diffuse myelitis,—namely, a perivascular diapedesis of leucocytes and hypertrophy of the neuroglia. No microbes were found in the cord; the lesions were produced by soluble toxins.

The results of Claude's subcutaneous injections with bouillon of mixed strepto- and staphylococci were a little different from the above, in that vascular lesions seem to have been much more conspicuous. Minute hemorrhages were seen with the naked eye to be scattered all through the cord. Under the microscope appeared a generalized hæmatic infiltration, especially in the anterior horns. The small vessels were dilated and filled with blood, their walls infiltrated with round cells. The lesions of the ganglion cells were such as have been described by other experimenters, with the addition that the diseased cells were found to be surrounded with round cells, mononuclear leucocytes, which penetrated into the ganglion cells, filled them, and caused them to disappear completely. The lumbar cord was the most affected, the dorsal region the least.[1]

The guinea-pig had been inoculated on the 28th of February; illness began on the 14th of March; on the 16th paralysis set in, with rigidity of the hind limbs and retention of urine; on the 17th extension of paralysis to the fore limbs and death.

SPINAL HEMORRHAGE.

It still remains true that the majority of cases of hemorrhage into the cord or the meninges are observed after traumatism. "Hemorrhage into the cord (apart from traumatism) is one of the rarest pathological events," declares Leyden.[2] Out of two hundred and forty-five cases of organic disease, Krafft-Ebing found only three in which hemorrhage was diagnosed either from the symptoms or from the autopsy. Outten[3] tries to prove, as Hayem did many years ago, that primary, uncomplicated spinal hemorrhage never occurs. Pfeiffer, however, does not assert that traumatism is the cause in more than ninety per cent. of the cases.[4]

The traumatisms necessary for hemorrhage are of very different degrees of severity. Parkin's seven cases were all associated with vertebral fracture after severe falls.[5] All but one proved fatal. A blow upon the head caused paraplegia of both upper extremities, with inability to hold the body erect, no motor paralysis of lower limbs, analgesia, thermo-anæsthesia, and persistent tactile sensibility, with markedly increased reflexes.[1] The diagnosis was made of hemorrhage into the cervical cord. In one case, during the act of lifting a heavy weight a man of fifty-seven was stricken with paralysis of all four limbs, followed by extensive muscular atrophy, most marked in the lower extremities.[2] This condition was referred to a meningeal apoplexy. In another case, a man was thrown from a cart; flaccid paraplegia, abolished reflexes, and total anæsthesia as far as the nipples followed, symptoms which would seem to indicate a transverse myelitis of the lumbar cord. But after death an extradural clot was found which compressed the cord from the level of the fifth dorsal vertebra and below.[3] Another case seems also due to lifting, although the symptoms did not occur until several days later. Then the man awoke in the morning with complete paraplegia, retention of urine and fæces, and sharp abdominal pains. After six months in hospital there was sufficient improvement for the patient to return to work. But the motor force of the legs was diminished, the patient walked stiffly, had a *mal perforant* on the right great toe, developed an osteo-periostitis on the right femur, and had the symptom of dissociated sensibility. The tactile and muscular sense remained intact in the presence of analgesia and thermo-anæsthesia.[4]

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[1] Claude, Comptes Rendus Soc. de Biol., 30 Mai, 1896.

[2] Zeitschrift für klin. Med., 1888.

[3] St. Louis Alienist and Neurologist, 1896: On Primary Hæmatomyelia.

[4] Centralblatt für allgem. Path. und path. Anat., September 15, 1896.

[5] Guy's Hospital Reports, 1891; so also Spiller's case, Fracture of the Fourth Cervical Vertebra, Internat. Med. Mag., April, 1896; also Lax, Inaug. Diss., Zirndorf, 1895.

The predominance of traumatism in the etiology of hæmatomyelia suffices to render children relatively exempt, because less exposed to many forms of traumatism. Yet children of any age are liable to falls, and especially during the rougher sports of boyhood. Outten quotes a case from Sharkey of a boy of thirteen, who fell while skating, but walked home apparently unhurt, except for some pain in the shoulder and abdomen. In two hours, however, he was unable to walk, and was admitted to the hospital with paraplegia, impaired sensation to the fourth dorsal spine, a temperature of 101, pulse 144, and pains in the abdomen and legs. He died after eight days, of pneumonia. At the autopsy was found a hemorrhagic extravasation which occupied the whole transverse extent of the cord and extended from the upper cervical to the mid-dorsal region. No evidence of inflammation was discoverable by microscopical examination.[5]

A form of traumatism necessarily peculiar to children is due to dystocia. Raymond[6] describes a case observed when the patient was six years old. The mother had lost four other children during labor, the fifth at the age of nine months, probably from meningitis. The patient presented by the feet, upon which the *sage-femme* made violent traction; the child was born violet and apparently dead; treated by insufflations from mouth to mouth for three hours. Twenty-four hours after birth both arms were found completely paralyzed. This persisted six years later, when the arms were found arrested in their development, with diffused muscular atrophy and correlative lipomatosis. Sensibility absolutely intact, muscles of lower limbs rigid without paralysis. Diagnosis of hemorrhage limited to the anterior horns of the cervical enlargement, without encroachment on the central gray matter, and with descending sclerosis of the lateral columns.

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[1] Bikeles, Neurolog. Centralblatt, 1894.

[2] Gobel, Münch. Med. Wochenschrift, October, 1895. Krafft-Ebing relates a case where a young seamstress became similarly paralyzed after lifting a heavy weight, Neurol. Centralblatt, 1890.

[3] Lambert, Bulletin Méd., 1895.

[4] Neurolog. Centralblatt, 1895.

[5] Outten, loc. cit.

[6] Prog. Méd., February 15, 1896.

Hemorrhage sometimes occurs before birth in children who are stillborn, resulting in paraplegia or diplegia, unaccompanied by epilepsy or other evidence of brain-lesion. Herbert Spencer found spinal hemorrhages into either canal or cord in thirty per cent. of one hundred and thirty stillborn children.[1]

The recognition of hæmatomyelia in certain cases, where otherwise myelitis might plausibly be suspected, has been much facilitated by the discovery that the dissociation of sensibility characteristic of syringomyelia is also produced by a hemorrhage which scoops out cavities in the central gray matter of the cord. Such cavities may even originate syringomyelia, with whose symptomatology their own naturally then coincides.[2]

In 1892 Minor, in an elaborate and important paper, described four cases of central hæmatomyelia, in all of which this dissociation of sensibility was the prominent symptom.[3] There was hyperæsthesia on the nonparalyzed side, and on the paralyzed side analgesia and thermo-anæsthesia, while tactile sensibility was preserved. Minor then stated that this syndrome was often described as characteristic of syringomyelia or central gliomatosis, but nowhere as connected with hæmatomyelia. Since the publication of Minor's paper this syndrome is regularly sought for in all cases of suspected spinal hemorrhage.

In 1888 Leyden pointed out the marked tendency of hemorrhage to concentrate upon the gray matter of the spinal cord, and especially at its central part. "The original seat of intra-medullary hemorrhage is always the gray matter, central or lateral."[4] The symptom of sensibility dissociation is only observed when the hemorrhage occupies the former locality.

In the case described by Spiller[5] fracture of the fourth cervical vertebra had caused an extensive hemorrhage into the fifth cervical segment, but it was limited to the posterior and anterior horns, leaving comparatively free the commissures and the white matter.

Among Parkin's seven cases, with six autopsies, the hemorrhage was limited to the gray matter in three, in the other three diffused throughout the entire substance of the cord. "Hemorrhage in the white matter is never primary."[1] Similar localization in the central gray matter was diagnosed during life by Krafft-Ebing[2] and Raymond.[3] Minor considers the following law to be demonstrated, namely: If hemorrhage occur in the cord, it rarely spreads towards the periphery, but descends along the central gray matter, hollowing out a tubular space of varying dimension and vertical extent, either close to the central canal or limited to the anterior horns, or to these and the posterior, never the latter alone. Such limitation existed in Spiller's case, notwithstanding the extensive traumatism.

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[1] Petersen, American Text-Book of Pediatrics, Starr.

[2] Anna Bäumler, Deutsches Archiv für klin. Med., Bd. xx. Heft 3.

[3] Archiv für Psychiat., 1892, Central Haematomyelie.

[4] Leyden, loc. cit.

[5] Loc. cit.

When, instead of a tube, circumscribed cavities are formed by the extravasation, an anatomical condition is produced mechanically identical with syringomyelia and giving rise to the same symptoms. In Minor's paper four cases are related, of which one was in a boy twelve years old. The boy had fallen and hit his hand so violently that blood flowed. A month later he hit his wounded hand against his chest. He immediately felt such a weakness in his arms and legs that he could no longer work. The weakness increased till he fell; his legs did not seem to belong to him. Obstinate vomiting for three days. On the fifth day complete dilatation of right pupil; paresis of the left hand and left leg; some atrophy in ulnar fingers; hyperæsthesia of the left half of body; motility unimpaired on right half, as also tactile sensibility, but analgesia and thermo-anæsthesia; knee-reflex lost on right side, exaggerated on left. Thus, typical Brown-Sequard syndrome. Pressure painful over spines of seventh cervical and first and second dorsal vertebræ. Pupillary symptom, mydriasis spastica, indicated lesion of cilio-spinal centre; this, with paralysis and atrophy in ulnar sphere, localized lesion at roots of last cervical and first dorsal nerves. Electrical excitability especially diminished in region of left median and ulnar nerves. Three months later, paresis of left leg almost gone, as also the analgesia, but hyperæsthesia and exaggerated knee-jerk persist.

In Minor's three other cases, all adults, the symptoms developed immediately after a severe traumatism, which pointed to hemorrhage, and these symptoms resembled so closely those of the boy that hemorrhage must be inferred in his case also. The most characteristic is the special dissociation of sensibility, which previous to these observations had not been signalized as a consequence of hemorrhage, but only as a symptom of syringomyelia or central gliomatosis. From syringomyelia a hemorrhagic lesion is to be distinguished by its acute onset, by the absence of trophic cutaneous lesions, and by its tendency to retrograde in the severity and extent of its symptoms.

Minor establishes three forms of hæmatomyelia: 1, with the Brown-Sequard type of crossed motor and sensory paralysis; 2, with atrophy of arms without anæsthesia in trunk and legs, paresis without atrophy, but

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[1] Parkin, loc. cit.

[2] Diagnosis of hemorrhage into left anterior horn, very little into posterior horn, Wien. klin. Wochensch., 1889.

[3] Prog. Méd., 1896.

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teries which pass from the periphery into the lateral
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lesion was found by Riggs after death from pernicious anæmia.[5]

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[1] The above details are taken from Williamson's essay on "The Relation of Disease
of the Spinal Cord to the Distribution and Lesions of the Spinal Blood-Vessels," London
1895.

[2] Beiträge zur Lehre von der Haematomyelie, quoted by Lax, Inaug. Diss., Zirndorf
1895.

[3] Loc. cit.

[4] Arch. Gén., 1885.

[5] Journ. Mental and Nervous Diseases, 1896.

with the syringomyelic dissociation of sensibility; 3, with coincidence in the areas of atrophy and sensibility dissociation.

In all Minor's cases the paralysis was unaccompanied by rigidity or contracture.

These observations seem to show that impressions for pain and temperature are transmitted by fibres which promptly plunge into the central gray matter of the cord, while tactile impressions are conveyed by the posterior columns of the cord. Thus, Schiff's law is confirmed.

The gray matter of the cord is more predisposed to hemorrhage than the white, on account of its softer texture, which offers less support to the blood-vessels, and because of the rich abundance of these.

The central gray is chiefly supplied by the anterior median arteries given off, to the number of two hundred and fifty or three hundred, from the anterior spinal. Each anterior median, entering the anterior commissure, divides into the anterior central, which passes to the anterior horn, the posterior central, which passes to the intermediate gray matter, the neck of the posterior horn, and the posterior vesicular column of Clarke and the anastomotic artery, which bifurcates and sends vertical branches upward and downward, uniting the commissural arteries. The posterior system of arteries sends a small branch to the caput of the posterior horn; but this system chiefly supplies the white columns and the nerve-roots thus: the posterior median, giving small branches to the columns of Goll; the intermediate septal, in the septum between the columns of Goll and Burdach; the posterior radicular artery to the median side of the posterior root; and the greater part of the peripheral arteries which pass from the periphery into the lateral white matter of the cord.[1]

The rich vascularity of the anterior gray matter, which tends to concentrate upon it diffused infections, is thus also an evident reason for the predominance of hemorrhages within this area, and for their limitation to it when not too extensive.

I have been unable to find any case of spontaneous spinal hemorrhage in a child among the clinical records of the last ten years. A few cases are mentioned in adults: by Eichhorst,[2] in a previously healthy young woman; Krafft-Ebing,[3] in a laborer of sixty-four; and Hitzig,[4] in a woman with melancholia, in whom the paraplegia was preceded by prodromal pains for three days, and followed by death on the third day from the paralysis, the autopsy finding the subarachnoid space filled with blood-clots. A similar lesion was found by Riggs after death from pernicious anæmia.[5]

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HEREDITARY ATAXIAS AND LOCOMOTOR ATAXIA.

By JOSEPH COLLINS, M.D

THERE are four distinct diseases that may occur in children in which ataxia is the most prominent symptom,—hereditary spinal ataxia, usually known as Friedreich's disease; hereditary cerebellar ataxia, sometimes called Marie's disease; ataxic paraplegia; and locomotor ataxia, or true tabes dorsalis. All of these diseases except the last, which, as we shall point out later, occurs so rarely that it barely deserves mention, are frequently encountered in childhood. They are all familiary or hereditary maladies. Although it is often impossible to trace a similar affection in the immediate ancestry, the most striking characteristic of these diseases is their occurrence in different members of a family. If the ancestry does not show the particular disease under consideration, investigation of it often reveals some other form of hereditary nervous disease.

The most satisfactory conception of the hereditary and familiary diseases—a class that is every year becoming more comprehensive—is that they are in their origin congenital; that there is some inherent defect in the proton of the neuron, motor, sensory, or intercentral. This defect does not usually reveal itself at birth, but at some physiological epoch in the patient's life, either a developmental or a retrogressive one. For the hereditary ataxias this epoch is the one of puberty. For another familiary and hereditary disease (Huntingdon's chorea) the epoch is a degenerative one,—at forty-five years. It is also possible to conceive that these diseases are based upon anatomical conditions which, although the proton or primary constituent was properly formed and laid down, are not possessed of the vitality to carry them through the span of life of other tissues. Thus the tissues that form the anatomical seat of the disease have a brief career, just as do all incompletely developed organisms.

The evolutionary history of the ataxias is interesting. Formerly they were all put under the one caption of locomotor ataxia, after Trousseau had expiscated the latter disease. Then, in 1861, Friedreich separated off a very distinct class of cases, now known by his name. Nearly a quarter of a century later Dana split up the remainder by calling attention to a class of cases which he designated as hereditary or familiary ataxic paraplegias. Finally Fraser, Sanger Brown, Nonne, and others further reduced the number of cases by describing a more or less uniform clinical type, to which Marie gave the name of hérédo-ataxie-cérébelleuse, or hereditary cerebellar ataxia. Thus there have been separated from the one central disease whose distinguishing feature is ataxia of locomotion three diseases, each of which has a number of features so distinctive as to be considered pathognomonic, but which have a number of clinical features in common. The activity thus displayed in the nosonomy of the ataxias has left the field made up of cases of genuine tabes in children very small,—so small, indeed, that it can scarcely be found.

Of the three ataxias mentioned, hereditary spinal ataxia has been differentiated longest and is best known.

HEREDITARY SPINAL ATAXIA (FRIEDREICH'S DISEASE); JUVENILE ATAXIA.

Friedreich's ataxia is either a degenerative disease or a lack of development of the peripheral sensory neuron and the central motor neuron in their spinal course. Posterior and lateral sclerosis of the cord is the anatomical condition on which the disease rests. It is a rare disease of childhood, characterized clinically by motor incoördination of the extremities and of the trunk, loss of the tendon jerks, disturbance of enunciation and articulation, nystagmus, and deformity of the spine and feet. In its course it is characterized by chronicity, being the least rapidly progressive of all spinal diseases, and by its uniform unamenability to every form of therapy.

History.—Hereditary spinal ataxia is to-day such a well-known disease that it really is of comparatively slight importance to trace the history of its development by enumerating the men of all nations who have contributed reports of cases. Although the disease was first referred to in 1861 as hereditary or infantile ataxia by the physician whose name is eponymically associated with it, wide-spread recognition of the disorder dates from 1876, when Friedreich described three additional cases and lucidly depicted its clinical characteristics. Between the dates of Friedreich's contributions cases had been reported by Carré, Bradbury, Carpenter, Kellogg, and Dreshfeld; and although some of these, particularly a case of Kellogg, another of Dreschfeld, and another of Bradbury, are no longer considered examples of the disease, these contributions represent the beginning of the differentiation of hereditary ataxia from tabes dorsalis, under which they had until that time been considered. After Friedreich's second paper the number of published cases increased so rapidly that ten years later Soca collected one hundred and sixty-five cases, a number of which, however, did not belong to this category. There are at the present time about three hundred cases in literature published as examples of Friedreich's disease, but careful examination of the histories and judicial consideration of the evidence leads to the conclusion that many of them are not entitled to the place allotted them. This is now very apparent, since a more or less definite symptomatology has been erected for hereditary cerebellar ataxia and hereditary ataxic paraplegia.

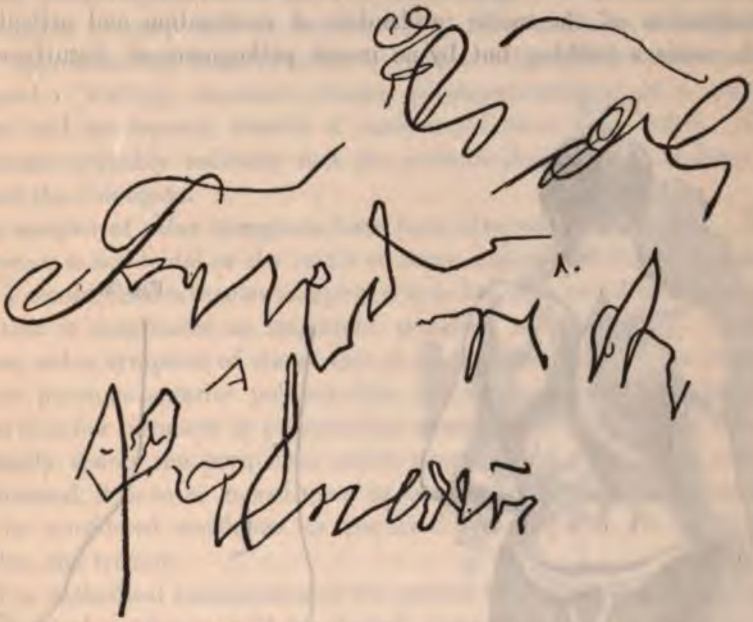
The first autopsy on a case of Friedreich's ataxia was made by Friedreich himself on one of the cases reported in his first paper. Schultze in 1877 made a very careful microscopical examination of one of Friedreich's cases, but the disease was not established on a firm pathological basis until 1878, when Kahler and Pick gave such a thorough description of the morbid changes that they tally with the findings of more accurate and refined histological methods. Americans have had much to do with establishing the recognition and description of the disease, and the contributions of W. E. Smith, J. P. Crozer Griffith, Charles L. Dana, E. C. Seguin, and Sanger Brown form an integral part of the history of the disease, both in its clinical and its pathological features.

Etiology.—The etiology of Friedreich's ataxia is enveloped in the same obscurity as all hereditary, familiary, and teratological diseases of the nervous system. If we could say why certain neurons do not reach full development, or why they die before others of similar development, we should have solved one of the most important questions with which the biologist is concerned. At the present date we can only say that these conditions occur; what the actual factors are that determine them is unknown. There are three important etiological factors to be considered. These are: the family history, the age when the symptoms first occur, and the real relationship of acute disease, which is an attributed exciting cause. The name hereditary spinal ataxia is misleading, because in at least one-third of the cases, and possibly one-half, there is no evidence whatever of immediate or remote heritage of the disease, and in upwards of ten per cent. of the cases there is no history whatsoever of pathological heritage of any kind. The most striking fact in the etiology is that the disease occurs in families, though even this is by no means discernible in all cases. It is more apt to occur in large than in small families, and at times it seems to affect the male members, while the females escape, or *vice versa*. Although all the members of a family are not affected, unless in exceptional instances where the number is very small, the remaining members may show some other form of degenerative nervous disease, and possibly a nervous disease of a teratological nature. The immediate and remote family history also may show the presence of some degenerative neurosis or psychosis, such as epilepsy, hysteria, inebriety, migraine, etc.

The disease develops, as a rule, between the ages of five and fifteen; it sometimes occurs in a recognizable form before that period, and the number of cases that have developed after the fifteenth year is not very small. I have a case under observation, which conforms clinically in every feature to the classic example of the disease, in which the symptoms were remarked for the first time when the patient was twenty-five years old. A few such cases are to be found in literature. A fact that has been noted so often that it cannot be considered accidental is that when the disease occurs in several members of the same family, it appears in the first patient during late childhood or early maturity, while in each succeeding patient it appears at a less advanced age. Friedreich's disease has this in common with some other family diseases, particularly juvenile dystrophies, in which I have more than once noticed a similar occurrence. The factors that apparently have something to do with exciting the disease, at least to such activity that it becomes recognizable, are the infectious diseases—naturally those common to childhood—and injury. These factors may be interpreted in two ways. The acute infectious diseases may have nothing whatever to do with causing the disease, except so far as they weaken the neuro-muscular system and keep the patient in bed, during which time complete coördinated movements, such as walking, running, climbing, etc., which the person may have but recently acquired, are partially forgotten. Either of these factors, or both combined, may be sufficient to make noticeable the most striking feature of a disease which existed even before the infection,—namely, incoördination. On the other hand, infectious processes and their products may act injuriously upon neurons robbed by heritage of their complement of development, and cause them to degenerate. This latter view I hold to be extremely improbable. A number of cases have been reported during the last few years in which the disease was ushered in with a simple fever,—that is, fever without any accompanying physical signs or clinical features to show its dependency upon an infectious or accidental process. What the genesis of this fever is has not been suggested, but it seems to me that the explanation of its injuriousness is the same as that offered for the infectious diseases. A number of other etiological factors of comparatively insignificant importance are the finding of the disease oftener in males than in females, oftener among the poor than among the rich, and the recording of no case in other races than the white. These facts, with the exception of the last named, are in entire accord with the teachings of other familiary and hereditary diseases, all of which show themselves more frequently in males and in peoples of the lower walks of life. The disease is met with in the poor and unenlightened because parental consanguinity, excessive fetation, and malnutrition are commoner in this class.

Symptoms.—After an acute illness, one of the acute infectious diseases, or a simple fever accompanied with pains in various parts of the body, or without the occurrence of such, it is noticed by the parents, or by the patient himself, if of an age to be observing, that there is a change in the gait and in the certainty of the movements of the extremities, and possibly also of the trunk. This uncertainty of gait and of movement may manifest itself in the beginning by awkwardness and clumsiness. It soon, however, becomes associated with weakness of the lower or upper extremities, which adds materially to the patient's disability; so that the gait which was at first merely lacking in grace, and slightly incoördinate, becomes now that of a person who is trying continually to balance himself and thus secure his equilibrium. He walks with the feet widely separated, and no sooner is the one foot down than he lifts the other heavily and puts it down in a place where it will balance as well as support him. This perpetual attempt to preserve the equilibrium in walking gives rise to a peculiar oscillating gait which, when associated with the phenomena of incoördination in other parts of the body, becomes rather characteristic. In some instances the incoördination of the upper extremities precedes and is more marked than that of the lower; usually, however, it is consecutive to the latter and less marked. It shows itself first in writing or in other highly coördinate movements. In an advanced stage it gives the handwriting a typical appearance, an example of which is shown by Fig. 1. When the patient attempts to stand, it is noticed that the body is in a state of continual titubation; first one set of muscles contract, to draw the centre of gravity towards one side, and this is soon followed by the action of an opposing set in a way that reminds one somewhat of the continual voluntary efforts of equilibrium-preservation made by the tight-rope walker. These movements are to be seen in the muscles of the trunk, in the muscles of the extremities, and in the muscles of the neck and face. They are evident when the feet are widely apart, more evident when the feet are approximated; but they are no more marked when the eyes are closed than when they are open. In short, there is static ataxia which is not exaggerated by closure of the eyes. In addition to the ataxic movements of the arms

FIG. 1.

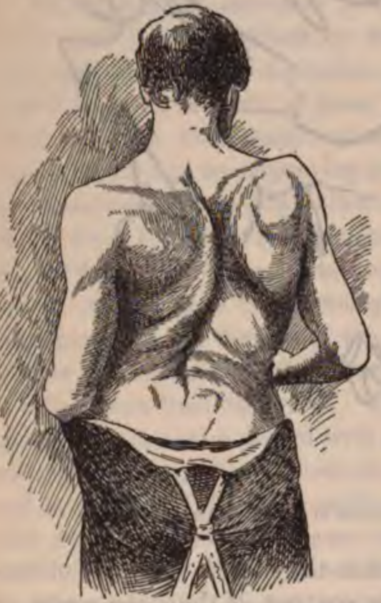


Handwriting in Friedreich's ataxia. Signature of patient. (After Besold and legs, there are sometimes continual involuntary choreic movements of the head and of the body, which are, I believe, to be explained by an involuntary attempt on the part of the muscles to maintain equilibrium.

On tapping tendons which in health respond by a quick, active muscular contraction and a consequent jerk, such as the knee-jerk and the elbow-jerk, it is found that these are diminished, unequal on the two sides, or absent, the latter being the rule. Increased myotatic irritability, exaggeration of tendon-jerks, and any form of clonus are conditions that are strictly inimical to the existence of the morbid conditions constituting the basis of this disease. The strength of the lower extremities becomes gradually more impaired, constituting mild degrees of paresis, but rarely is there anything like paraplegia.

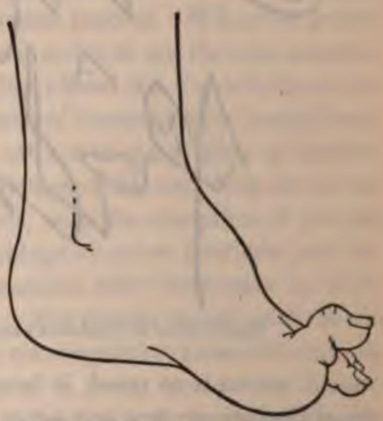
Coincident with the development of the incoördination dependent upon the loss of the sense of equilibrium in the other parts of the body there is incoördination of the motor mechanism of vocalization and articulation which causes a striking but by no means pathognomonic disturbance of speech-production. Intonation becomes irregular in rhythm, usually constant in pitch, and oftentimes nasal, while articulation is syllabic.

FIG. 2.



Deformity of spine in Friedreich's disease. (Taylor.)

FIG. 3.



Club foot of Friedreich. (Brissaud.)

As the disease makes progress two very common deformities develop, the one of the spine, the other of the foot. That of the spine is some form of rotatory or lateral curvature, most often of the cervico-dorsal and dorsal regions, while that of the foot is a hyperextension of the toes, particularly of the first and second, which produces a condition distressing to observe, although apparently not greatly annoying to the patient, and a curvature of the arch, which gives rise to a concavity of the plantar surface,—in short, to a more or less typical pes cavus. This condition of the foot does not develop until after the disease has existed for some time. Preceding it, even in the very early stages of the disease, there occurs a phenomenon which I have frequently noted. If the feet are watched, the extensor tendons going to the toes are seen to become alternately rigid, so that they stand out like the prongs of a fork, and relaxed : a part of the balancing movement that the patient makes. The deformity of the feet and the toes in an advanced degree is well shown in the accompanying illustrations.

Another symptom which develops later in the disease is nystagmus, usually of a lateral character, although the rotatory form may be elicited in some cases.

The patient himself does not complain, except of awkwardness in the performance of purposive movements. In a few instances neuralgic-like pains approximating those of tabes have been remarked, but they are exceptional. Vertigo, transient attacks of unconsciousness of a syncopal nature, and paroxysmal attacks of tachycardia have been noted. Their occurrence probably indicates that the neurotic degeneration or decay has reached the oblongata.

A number of other symptoms have been observed occasionally. Their occurrence is accidental or the result of some superadded disease. Among these I would class muscular atrophy, which has been noted by Sachs, who says that it constitutes an important symptom of the disease. It is, I believe, not a symptom of the disease at all, but the result of an intercurrent or previous anterior poliomyelitis, the existence of which has been shown in a few instances by post-mortem examination. Friedreich's ataxia habitually spares the peripheral motor neurons, and when these neurons are diseased, it is to be looked upon as accidental. Other symptoms that may be considered accidental or psychical are polyuria, tic of the face muscles, and tremor.

If a methodical examination of the patient be made, a number of negative physical conditions will be elicited, some of which are of great importance in making the diagnosis, and particularly in separating it from other diseases that have ataxia as a prominent symptom. These are absence of impairment of the pupillary reflex either in accommodation or on exposure to light, absence of insufficiencies of the ocular muscles, absence of impaired activity of the cranial nerves, integrity of the sphincters, and preservation of the various forms of sensibility mediated by the skin. The superficial reflexes are usually preserved, and trophic and vaso-motor disturbances are extremely rare.

Mentally the patient is usually without symptoms that attract attention, although a few cases of Friedreich's disease have been reported in connection with cretinoid idiocy, particularly a family reported by Nolan. As a rule, the patients are intellectually as well developed as others in their station of life. Their infirmity prevents them from going to school, and they are thus often devoid of this kind of education, which, combined with a certain apathy of facial lineament, has given rise to the erroneous impression that they are stupid or slightly imbecile.

Course, Duration, and Prognosis.—The course of the disease is a progressive one, but, unlike a disease from which it is oftentimes necessary to differentiate it,—multiple sclerosis,—it does not progress intermittently. It may safely be prophesied that each succeeding year will find the patient a little more incapacitated. There are no real remissions in the course of the disease. It is slower in complete development than any other spinal cord affection, and in a number of cases its existence does not seem to abbreviate very materially the patient's life, although he habitually succumbs to some intercurrent affection, such as tuberculosis, to which his inactivity and helplessness particularly predispose him. Thus it may truthfully be said that the disease leads to a fatal termination in from ten to fifteen years.

Diagnosis.—The distinguishing features of Friedreich's disease are six in number:

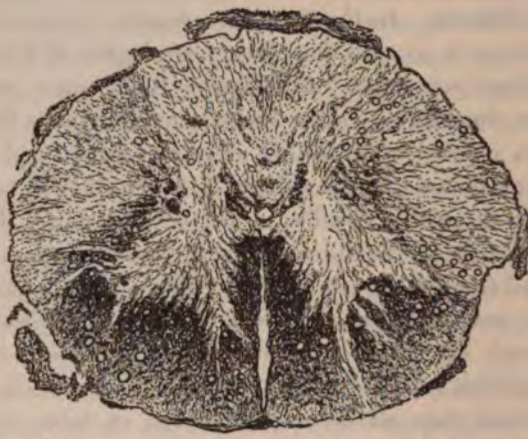
1. Ataxia, for all forms of purposive movements and static ataxia, incoördination due to loss of the sense of equilibrium, and irregular, involuntary movements necessitated by the continual attempt to maintain equilibrium.
2. Loss of the tendon-jerks, diminished myotatic irritability, and weakness which may amount to a paresis of the lower extremities.
3. Deformities of the spine and of the feet.
4. Nystagmus, static and dynamic.
5. Disturbance of articulation and intonation.
6. Absence of lancinating pains, intactness of sensibility, normal pupillary reactions, no disturbance of vision, and no disturbance of the urogenital sphere.

If these six conditions are present, and a history of familiary or hereditary occurrence can be elicited, the diagnosis is absolutely assured. If such a history cannot be elicited, and disseminated insular sclerosis can be excluded, the diagnosis of Friedreich's disease is just as certain. In parallel columns it would be very easy to detail symptom-groups, one for multiple sclerosis, the other for Friedreich's disease, so dissimilar that it would not seem possible to confound them. Nevertheless, recent experience has shown that multiple sclerosis is not a very uncommon affection in children, following the acute infectious diseases and as the manifestation of a teratological condition. Such experience has shown, moreover, that atypical forms of multiple sclerosis are as common as the typical. A patch of sclerosis in the posterior columns of the spinal cord may cause loss of knee-jerks, although exaggeration of knee-jerks is considered a prominent symptom of the disease; but deformity of the spine and feet, and particularly the deformities occurring in Friedreich's disease, rarely occur with multiple sclerosis. A patch of sclerotic tissue forming in the cerebellum may cause disturbances of equilibrium simulating those of the disease under consideration, but they are never of the titubating and perpetually balancing character of those occurring in Friedreich's disease. Then the intentional tremor, the nystagmus, the scanning speech, the spastic facies, the explosions of emotional display, are all more striking in multiple sclerosis than in Friedreich's ataxia. The course of the two diseases is essentially different: the one, Friedreich's disease, uniformly progressive; the other progressive actively for a time, followed by remittances so striking as almost to amount to an intermission. The diagnosis of Friedreich's disease, at least a more or less typical example of it, from true tabes dorsalis is not difficult. In the first place, genuine locomotor ataxia before the age of puberty is so rare that even neurologists of the widest clinical experience seldom see a case of it during their entire professional life. Secondly, tabes dorsalis posits for its existence in more than nine-tenths of all cases an antecedent syphilitic infection, while syphilis has no influence, direct or indirect, so far as has been determined, on the causation of Friedreich's disease. Thirdly, a diagnosis of tabes dorsalis without the presence of two of the three following symptoms—viz., the Argyll-Robertson pupil (loss of the iris reflex to light), perversion of function of the sphincters, and disturbance of some of the modalities of sensibility mediated through the skin—is never justified, and none of them form a feature in Friedreich's disease, and there is yet to be put on record a single instance in which they all occur. Moreover, in tabes, nystagmus, disturbance of speech, and irregular balancing movements of the trunk do not form part of the disease.

Pathology.—Friedreich's disease belongs to the combined systemic diseases of the spinal cord. The lesions found after death are invariably decay, or possibly lack of development, in the spinal portion of the peripheral sensory neuron, the columns of Goll, which are sclerosed usually throughout their entire length, and the columns of Burdach, which show the diseased processes much less extensively distributed, and which preserve one part—that adjacent to the posterior horn, the column of Lissauer—usually intact, and, in the spinal portion of the primary motor neuron, the pyramidal tracts.

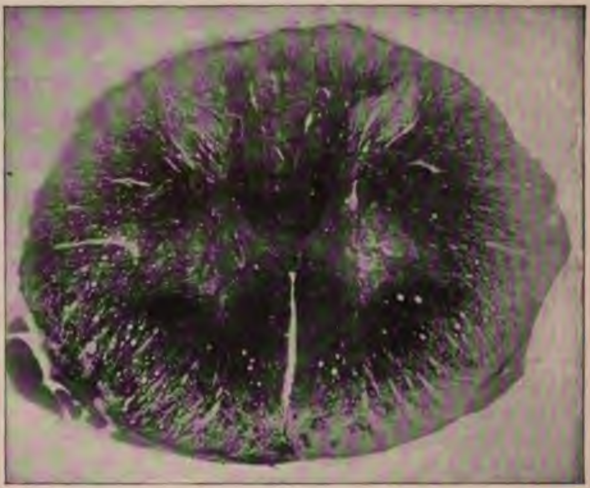
When the spinal canal is opened, the meninges usually present no abnormality. The pia is easily detached from the cord, and is not thickened except in rare instances. The spinal cord itself is almost always uniformly diminished in size, the diameter of the cord in its various levels being reduced from one-third to one-half. It has been claimed that this diminution in size, which is more striking in the dorsal cord than at any other level, is due to the contraction of the sclerotic tissue, but this seems very improbable, and it is more reasonable to attribute it to defective development of the constituents of the cord. When the finger is passed over the cord after it has been removed from the canal and laid on a flat surface the consistency is found to be increased, and this same feature is readily detected on cross-section. The sclerosis in the posterior columns and the pyramidal tracts is often apparent to the naked eye, but is particularly striking when the cord has been hardened in Müller's fluid and sections stained according to the method of Weigert or any of its numerous modifications. The loss of nerve-fibres to the posterior columns and the cross-pyramidal tracts is seen in the yellowish-colored areas given to specimens stained according to this method. In addition to the sclerosis of the columns of Goll and Burdach and the cross-pyramidal tracts, there are found in many instances changes in other tracts, more particularly in the direct cerebellar tract, which is not infrequently sclerosed from its beginning in the lower dorsal region up to the oblongata. In a few cases (Auscher's, Rossi's) the intra-cranial parts of the nervous system, including the oblongata, have been found to be much below the normal size. In others purely accidental or secondary conditions have been found, such as a tumor of the cerebellum, as in a case reported by Clarke. In other instances the antero-lateral tract or columns of Gower have been found diseased, and Dana has recently recorded an example, the illustrations of which are herewith reproduced (Figs. 5 and 6), in which there was, in addition to the changes characteristic of Friedreich's disease, a well-marked annular sclerosis of the periphery of the cord, a condition found by Schultze in one of the first cases, as well as a striking vacuolation consisting in the presence of holes which varied in size from half a millimetre to two millimetres in diameter, in which were apparently dilated vascular spaces distributed rather uniformly throughout the gray and the white matter of the cord. These are distinctly shown in Figs. 5 and 6. Other complicating lesions that have occasionally been noted are syringomyelia, periependymal sclerosis, and ancient poliomyelitis, or the spinal lesions of progressive muscular atrophy. What the

FIG. 4.



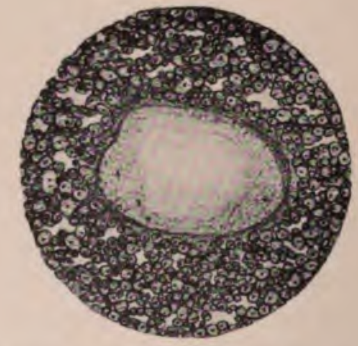
Friedreich's ataxia, showing degeneration in posterior column. (Dana.)

FIG. 5.



Friedreich's ataxia. Photograph of cervical level, showing degeneration of posterior columns, annular sclerosis, and "dilatation" spaces. (Dana.)

FIG. 6.



Friedreich's ataxia, showing dilatation. × 320. (Dana.)

pathogenesis of these lesions is can only be surmised. The view that the process is a sclerosis, as was contended especially by the French pathologists some years ago, would seem latterly to have lost favor, and the view that regards the pathogeny of the disease as a developmental shortcoming of the fibres and early decay, without consequent growth of neuroglia, has proportionately gained in favor. The idea that the lesion is a primary degenerative disease of the spinal blood-vessels, as was contended by Blocq and Marinesco, is, I believe, entirely discarded. It would seem probable that the application of the Mallory stain and of the Weigert stain, both for neuroglia, would soon settle this question of whether or not the disease is a primary sclerosis, meaning by that an overgrowth and excessive formation of glia tissue. Until such application of these stains, which were discovered but yesterday, be made, the preponderance of evidence would seem to be against considering it a sclerotic process.

Treatment.—The treatment of hereditary spinal ataxia consists in providing the patient with an intelligent attendant or nurse, who will practise daily with the patient the system of purposive gymnastics known as Fraenkel's movements,—systematic exercises for training the ataxic limbs, —which are often of material service in the treatment of locomotor ataxia. The exercises cannot be detailed here, but may be found in the original in the *Münchener medicinische Wochenschrift*, No. 52, 1890, or in the *New York Post-Graduate* for July, 1896. These, with measures to maintain the strength and nutrition of the patient, are all that can be offered in the shape of therapy. It is not probable, even if we could treat the patients at the very beginning, that medicine, such as silver, which often has a beneficent action in preventing the rapidity of certain spinal-cord degenerations, such as tabes, would be of any service in this disease. Parents to whom are born one or more children who afterwards manifest a disease of this kind should be implored to surcease procreation.

HEREDITARY CEREBELLAR ATAXIA.

Soon after the symptoms of Friedreich's ataxia began to be universally recognized as a disease with a fairly constant symptomatology, cases began to be reported which, although they had, in common with Friedreich's ataxia, the familiary or hereditary character, differed from it in many important and one or two very essential conditions, such, for instance, as the state of the reflexes and the knee-jerks. The first of such cases of which there are records would seem to be those of Fraser, which were reported in 1880. On one of his patients a careful autopsy was made; nevertheless, they were all considered anomalous forms of Friedreich's ataxia. Eleven years later Nonne, of Hamburg, described three cases, all from the same family, in which the clinical history, as well as the pathological findings in one of them, showed a very marked departure from hereditary spinal ataxia.

In the following year, 1892, Sanger Brown, of Chicago, published a paper containing the report of a large number of cases of familiary ataxia occurring in children and young adults. A number of these cases, it was recognized by the author, as well as by Ormerod, of London, and Bernhardt, of Berlin, whose critical remarks formed an appendix to Brown's article, could in no way be considered examples of hereditary spinal ataxia unless the conception of the latter disease was entirely changed. Since that time a number of cases, some typical, others atypical, have been recorded by Klippel and Durante, by Brissaud, by Londe, by Jacoby, and by myself; while cases of familiary ataxia which do not correspond with the general description of either hereditary spinal or hereditary cerebellar ataxia have been published by Menzel, by Seeligmüller, by Erb, and by Nonne.

Definition.—Hereditary cerebellar ataxia is the name given by Marie, who analyzed some of the above-mentioned cases, to a complex of symptoms occurring generally in the later years of childhood and early adult life, consisting principally of ataxia of a cerebellar character, increased knee-jerks, hesitating, abrupt, explosive speech, and mental shortcomings, the course of the disease being progressive. The name hereditary cerebellar ataxia was given to it not only because of its conformity with the name of the disease which it resembles,—hereditary spinal ataxia,—but because it indicated the most important symptom of the disease as well as its hereditary nature.

Etiology.—The etiology of hereditary cerebellar ataxia is as obscure as that of hereditary spinal ataxia. The meagre statistics at our disposal show that the familiary and hereditary features are much more constant than in the latter disease, although it is possible that further experience will show, as it has shown in Friedreich's ataxia, that this is not absolutely necessary. The familiary element is often easily made out, the hereditary element is much more uncommon. The disease first shows itself at a more advanced age than Friedreich's disease,—from ten to twenty-five,—although cases have been recorded occurring earlier. Jacoby has recently reported a most typical case in a child seven years old. Males have been afflicted more often than females. In short, the social and sex relations are similar to those of Friedreich's ataxia, and the same may be said of incidental factors, such as the occurrence of infectious diseases, blows, falls, fright, exposure, all of which have been noted more than once by different observers. If the disease is a protal one,—that is, one positing for its occurrence a lack of connate development in the constituents of the cerebellum,—as I believe it is, then none of these factors can have anything material to do with the causation of the disease, although they may bring into earlier prominence symptoms that might otherwise be delayed.

Symptoms.—The symptoms of the disease, oftentimes first discovered after some injury, exposure, or fright, show themselves usually by an impairment of coördination in walking. This is at first attributed to awkwardness, or, if it develops after acute illness, to the effects of the disease. The clumsiness and awkwardness are manifest particularly in the execution of difficult coördinated movements, such as those of climbing, dancing, etc., but soon they so interfere with the movements of walking as to be distinctly manifest in the gait, which becomes uncertain, staggering, and reeling, very like that of a person who has the motor form of alcoholic intoxication,—in short, a typical cerebellar gait. In other patients a distinctly spastic element is added to the gait, constituting a spastic cerebellar gait. The patient does not watch his feet while walking, as those with tabes do. On the contrary, the position of the body in walking is a rather striking one: the upper part of the trunk is slightly flexed, the head is thrown back, the chin is somewhat elevated, and in taking steps the patient keeps the feet wide apart, as if to give himself a broad base of support, but he has a tendency to reel in doing so. When he stands there is marked titubation of the body, but closing the eyes does not increase the uncertainty of station,—that is, Romberg's symptom is not present. Movements of the upper extremity reveal a similar incoördination to that of the lower, although the patient has more control of these movements, and the execution of highly complex movements such as writing is difficult at the beginning of the disease and impossible when the disease is more advanced. Coincidently with the ataxia of gait and uncertainty of movements of the upper extremities there develop defects of articulation and phonation. Speech becomes slow and hesitating in preparation, abrupt and explosive in execution. The voice is monotonous. Later the necessary coördination of the various peripheral parts that enter into the execution of speech becomes so defective that the patient is unable to speak so as to be understood.

The patient does not usually make any complaint of pain or discomfort, even though he be of an age to recognize the enormity of the other symptoms and the difficulty of executing coördinate movements. It may be noted by the patient himself or by the family that the memory and the faculty of association are becoming impaired.

On examination a number of very interesting features can usually be made out. In the first place, myotatic irritability is usually above normal and the knee-jerks are increased, and sometimes there has been found distinct ankle clonus. Examination of the eyes rarely fails to show one symptom,—that is, deficiency of action of the external recti muscles. Unlike Friedreich's ataxia, nystagmus is rarely or never present, although there may be slight jerky movements of the eyeballs on associated movement of the eyes. In a few advanced cases there have been noted progressive chorioiditis and optic atrophy. The patients have a peculiar facial expression which I find difficult to describe. It is not the spastic expression which one often sees in multiple sclerosis, nor is it the one of absolute repose sometimes seen in the familiary atrophies, although it is an absent expression when the child is not speaking or manifesting an emotion. The striking feature comes, however, when the child speaks, laughs, or cries, and consists in an over-action or a lack of connate action in the mimetic muscles which sometimes gives the face a peculiar dwarfish expression.

The functions of the bladder and the bowels are not usually impaired, nor is the sexual capacity abolished. Sensory shortcomings have not been recognized. There are no deformities of the spine and of the feet, such as are so striking in hereditary spinal ataxia, or at least not in the forms that are considered typical. In an atypical case recorded by me there were hyperextension of the great toe and well-marked pes cavus. In a number of instances the very interesting observation has been made that the occipital part of the head is strikingly flat, particularly that part that covers the hind-brain. This has been explained on the ground that nature builds only what she intends to fill, and as the cerebellum is small the posterior skull has developed accordingly. It is in reality an evidence of the law of consonance of development existing between the brain and the skull.

The disturbance of coördination undergoes a very striking change when the patient lies down, as would be expected, considering that the ataxia is cerebellar in its nature; the patient is then able to execute purposive movements that were very difficult or even impossible when he was in the erect position. Muscular sense, postural sense, and kinæsthetic sense do not seem to be materially affected. Trophic disturbances are in no way a part of the disease.

The course of the disease is essentially progressive, and the patient may remain for a long number of years completely bedridden, not paralyzed, but from sheer inability to utilize the power that is left to him. This condition facilitates the development of marasmus which itself leads to the death of the patient, or it makes him more susceptible to some infectious process which causes dissolution. The striking absence of subjective symptoms may in part be explained by the mental torpor, or the lack of mental awakement, or the dementia that these patients almost invariably show; but in some instances vertigo, vomiting, headache, and other symptoms associated with cerebellar disturbances have been noted.

The differential diagnosis is to be made particularly from hereditary spinal ataxia, from disseminated or multiple sclerosis, from infantile cerebral palsies of a familiary type, from chronic internal hydrocephalus, and from cerebellar neoplasm in the young. The points of differentiation from the first named are set forth in the following parallel columns. Although the differential diagnosis can and should be made, it can be done only after careful examination and study. One symptom of diagnostic importance between these two diseases should be mentioned above all others, and that is the condition of the reflexes. A patient who has present or exaggerated knee-jerks has a symptom inimical to the pathological condition which is the basis of hereditary spinal ataxia, and this symptom alone more than any other should exclude that disease.

Hereditary Cerebellar Ataxia.	*Hereditary Spinal Ataxia.*
1. Most common from tenth to twenty-fifth year.	1. Most common from fifth to fifteenth year.
2. Hereditary and familiary history can almost invariably be traced.	2. Familiary and hereditary history lacking in about one-third of the cases

Hereditary Cerebellar Ataxia.—(*Continued.*)	*Hereditary Spinal Ataxia.*—(*Continued.*)
3. Gait uncertain, reeling gait of inebriated person; occasionally slightly spastic. Patient walks with feet wide apart, body bent forward, head thrown backward, chin elevated. Does not watch the feet.	3. Gradual impairment of coördination, first in legs, afterwards in arms. Continual balancing movements both on standing and on walking. Gait paretic and ataxic, unsteady but not reeling. Balancing gait.
4. Romberg's symptom not present.	4. Ataxic station, but not usually increased by closing eyes.
5 Ataxia is very much less or disappears when patient is lying down, but incoördination can always be demonstrated.	5. Balancing movements may persist while patient is lying, but generally they disappear.
6. Over-activity of mimetic muscles when speaking or on emotional display.	6. No particular change in mimetic muscles when patient talks.
7. Speech hesitating, abrupt, explosive, ataxic, defective.	7. Affection of speech, eliding of syllables, syllabication monotonous; late symptoms.
8. Knee-jerks present or exaggerated. Occasionally ankle clonus.	8. Knee-jerks decreased or absent.
9. Irregular twitchings of eyeballs, not true nystagmus. Deficiency in action of external recti.	9. True nystagmus (static and dynamic) often develops when the disease is advanced.
10. No deformity of spine or of feet.	10. Kypho-scoliosis, pes cavus, and equinovarus.
11. Mentally often deficient up to complete dementia.	11. Mentally normal.

It is often very difficult to differentiate it from multiple sclerosis, and if islets of sclerosis develop in the cerebellum of a young person it is impossible to make the differential diagnosis except by the course of the disease, multiple sclerosis being characterized by progressions and profound remissions. The most important points in the differential diagnosis are as follows. Multiple sclerosis is neither a familiary nor a hereditary disease, although Strümpell has recently suggested that it may be due to congenital defect. Optic atrophy, particularly of the temporal halves of the retina, and true nystagmus are common in multiple sclerosis and not in hereditary cerebellar ataxia. The expression of the face is spastic in the former and ataxic in the latter. Sensory disturbances and vesical and rectal incontinence are not uncommon in multiple sclerosis, while they are decidedly uncommon in the hereditary cerebellar ataxia. Paraplegia, or paresis of a member, which is not uncommon even as a beginning symptom of multiple sclerosis, never occurs in hereditary cerebellar ataxia.

Congenital diplegia of a family type does not often require to be excluded, and when it does there is little difficulty in doing so if the time of occurrence, the attributable cause, the predominance of symptoms indicating cerebral defect, the course of the disease, the absence of the distinctly cerebellar gait, and titubation be closely attended to.

In cases of chronic internal hydrocephalus the one factor of absence of familiary history should make the differentiation, not to speak of the fact that the knee-jerks are usually lost in this condition, and that there are somatic evidences in the cephalic extremity to help make the diagnosis.

Morbid Anatomy.—Very little can be said of the pathogenesis and morbid anatomy of this disease other than what is based on analogy and theory. Autopsies have been made on a case reported by Fraser, on one by Nonne, and on an atypical case by Menzel. In Nonne's case the principal pathological findings were in the cerebellum; this was reduced to about one-half the normal size; and in the case reported by Fraser the reduction in size was even greater, the pia over the hind-brain being the seat of little cysts. Such hypoplasia of the cerebellum must of necessity be associated with imperfect development or absence of the many projection and association tracts that develop in connection with the cerebellum, and it is upon such lack of development (or decay?) that the symptoms depend. The cerebellum may, however, be of its customary size and the lesions demonstrable only on microscopical examination. These lesions consist essentially of a reduction in number and atrophy of the cells of Purkinje. Whether or not such sparsity of cells be associated with increase of neuroglia it is difficult to say. Theoretically our present knowledge of the neuroglia tissue would tend to the supposition that there should be; experience, although it is very limited, gives a negative answer.

It is possible that the various anomalous and transitional forms of this symptom-complex which have been described will be found to vary with the different parts of the cerebellum (or other parts of the brain) and association and projection tracts that are the seat of primary defect, and eventually it will be possible to place all the cases under the same category with somewhat varying symptomatology, depending upon the seat of the teratological malformation.

Treatment.—The same as for hereditary spinal ataxia.

LOCOMOTOR ATAXIA (TABES DORSALIS).

Genuine tabes dorsalis occurs so rarely in children that the subject deserves but brief consideration. I have seen but one example of the disease in a child, and that by courtesy of Dr. Joseph Fraenkel, physician in charge of the Montefiore Home for Chronic Invalids. In this case it is more than likely that the lesion was a syphilitic one, pathologically speaking, and not a parasyphilitic one, which is characteristic of true tabes. A girl eight years old, whose father and mother were both syphilitic, complained of shooting pains in the extremities, especially in the legs, and soon after showed impairment of locomotion. She gradually developed loss of knee-jerks, impairment of the vesical sphincter, and loss of pupillary contraction on exposure of the retina to light (the Argyll-Robertson pupil). Examination at this time showed, in addition to these symptoms, well-marked sensory disturbances, particularly slowed transmission of tactile and painful stimuli, and anæsthesia. The impairment of locomotion became so pronounced that she was unable to walk unaided. Later it was remarked that articulation was becoming defective, and this was the beginning of a typical general paresis which the child developed in the twelfth year. In this case the peccant activity of syphilis has shown itself in the most destructive way, causing two diseases which owe their origin, the one (tabes) in about ninety per cent., the other (general paresis) in about seventy-five per cent., to syphilis.

A very similar case, although the patient has not shown symptoms of mental decay, has recently been reported by Bloch, of Berlin. The father of the patient had syphilis, and transmitted it to every one of his children, all of whom, with the exception of the tabetic patient, were cured by a course of inunctions. In his fifth year it was noticed that the little patient had dilatation of the pupils; later there was incontinence of urine, fixity of the pupils, Romberg symptom (swaying on attempting to stand with closed eyes and with the fëet in apposition), and absence of the knee-jerks. At thirteen years of age he presented all the clinical symptoms of tabes. A search of the literature shows that there are about a dozen cases occurring before the age of puberty that will bear close analytical examination and separation from Friedreich's ataxia, although more than three times that number have been reported. In upwards of one-half of these twelve cases no history of inherited or acquired syphilis could be made out; but it is to be noted that in the cases that have been reported as ocurring in the children of non-syphilitic parents there was in one aortic insufficiency which is often due to syphilis, and in another a history of repeated abortions.

The only cases of tabes in a child in which an autopsy has been made are those reported by Gombault and Mallet and Arnold Pick. In these cases neither the symptoms nor the course of the disease were characteristic of tabes. In the patient of Gombault and Mallet the first symptoms began during the patient's eighth year, and consisted of flaccid paralysis of the legs without ataxia; later the arms became paretic and showed some ataxia on movement. Disturbances of sensibility and loss of reflexes occurred later. The patient died fifty years after the first symptoms of the disease, and the autopsy revealed lesion in the posterior columns of the cord, atrophy of the entire gray substance, and degeneration of the peripheral nerves; so that to consider this case one of true tabes would be absurd. Microscopical study of the cord from Pick's patient showed that the case was in reality one of combined systemic disease.

In one or two instances the symptoms have seemed to date from a fall; but as this is such a common attributable cause in all the ataxias, little weight should be attached to it. Moreover, in one case which seemed to develop after injury (Hildebrandt) there were a number of symptoms that are of very uncommon occurrence in tabes, such as loss of pupillary reflex in accommodation, horizontal nystagmus, kypho-scoliosis, equino-varus, insufficiency of the right abducens, and atrophy of the legs.

Some of the other reported cases of tabes that we are willing to admit to the category of tabes dorsalis, such as those reported by Freyer, Remak, Strümpell, and Kellogg, show anomalous symptoms which point to the probability of some of them being cases of spinal-cord syphilis transmitted by the parents.

In short, it may be said that genuine tabes dorsalis occurring during childhood is the rarest disease of the central nervous system. When the symptoms characteristic of this disease occur during childhood, some of them are examples of spinal syphilis, gummatous meningitis, and meningomyelitis of the posterior part of the cord, while others may be examples of true tabes,—*i.e.*, a slowly progressive decay of the peripheral sensory neurons beginning outside of the cord and extending into it, the completed process constituting degeneration of the posterior columns of the spinal cord, the degeneration being principally of certain zones of these columns, such as the zone of Lissauer, the postero-external zone, and the columns of Goll. Such lesions may produce the diagnostic symptoms of tabes,—viz., ataxia of locomotion and station, swaying on halting, pronounced and early difficulty on descending stairs, loss of knee-jerks, Argyll-Robertson pupil, impaired action of the sphincters, sensory disturbances, such as pains, anæsthesia and analgesia, particularly of the lower extremities, and optic atrophy. It should be borne in mind, however, that in every case in which such symptoms occur the chances are that the disease is Friedreich's ataxia, and that careful search will show corroborative symptoms and the familiary or hereditary history.

The prognosis of tabes is not so unfavorable as in cases of Friedreich's ataxia,—that is, there is more chance that therapy may do something to stay the rapidity of the disease. In some cases in which there has been a history of inherited syphilis benefit has been obtained by the use of inunctions; but it deserves to be emphasized that such cases are not examples of real tabes, but of spinal syphilis, and are pseudo-taboid.

If a physician should meet with a case of tabes in which no history of syphilis in the parents and no manifestations of it in the child could be discovered, the proper treatment for such a patient would be the education of the motor sensibility after the method of Fraenkel, the administration of nitrate of silver, care being taken to prevent argyria, and the application of measures to better and maintain his nutrition.

HEREDITARY OR FAMILIARY ATAXIC PARAPLEGIA IN CHILDREN.

Before concluding this chapter, I desire to say a few words on ataxic paraplegia in children. I am not at all certain that this disease is either hereditary or familiary. It is spoken of in this connection because ataxia is an early and constant symptom.

In his chapter on the hereditary ataxias of children, in the first edition of this Cyclopædia, Dr. Charles L. Dana said, "There is a primary degenerative disorder of the spinal cord occurring in children and characterized by symptoms of ataxia and some cutaneous anæsthesia and spasm. The disease develops usually about the time of puberty. It progresses slowly, and takes upon itself the characters which I have described under the name 'spastic ataxia,' but to which the name ataxic paraplegia is more often given." Concerning the occurrence of this disease there can be no doubt. What the real nature of it is, and where it belongs among the developmental or degenerative diseases of the nervous system, it is at the present writing impossible to say. We may conceive that it is a primary degenerative disorder involving the lateral columns of the cord and to a lesser degree certain of the central or peripheral neurons, or as a condition of incomplete development. The symptomatology and course of the disease are best illustrated by the citation of a very typical case, as a sufficient number has not yet been recorded to justify any stereotyped portrayal of symptoms to fit every case.

A girl fifteen years old, the fourth child of healthy parents, had been well until her thirteenth year, except for a series of convulsions in her third year, which were attributed to fright. She was in convulsions at that time for upwards of four hours. When thirteen years old she had to leave school because of vertigo, headache, and stiffness of the limbs, especially the legs, which shortly afterwards became paretic, especially the right. She was unable to walk on account of the stiffness and paresis, and remained in bed for from eight to ten weeks. The incapacity of the legs gradually disappeared under active treatment consisting of massage, galvanism to the spine and to the muscles, hydrotherapy, and a vigorous restorative treatment. She remained fairly well for about a year, although there was some uncertainty of gait. Then similar symptoms again developed. She noticed that she often fell in mounting steps and afterwards when walking on the level. Walking continually grew more difficult, until it became impossible without assistance. Examination showed pronounced spastic and ataxic gait, exaggerated knee-jerks, slight foot-jerk but no real clonus, and marked paresis of the legs. There was no disturbance of cutaneous sensibility, but loss of muscular sense and sense of position, especially in the lower extremities. Slight ataxia of the upper extremities. At this time there were no sphincter symptoms, no nystagmus, no paresis of the ocular muscles, no disturbance of speech, and no deformity of the spinal column. The feet were deformed in a way very similar to that in Friedreich's ataxia, the fingers were "crooked," the first and second phalanges somewhat flexed and the terminal phalanges slightly extended, and the handwriting was more "wobbly" than usual. Under active treatment she bettered, as before, but after a few months the symptoms returned and were much more severe and extensive than before. Examination at this time showed the gait exquisitely spastico-paretic, and the upper extremities in a similar condition but to a less marked degree. The speech became like that of a person moderately intoxicated; syllables were elided and the words jumbled together so that she could scarcely be understood.

The vesical sphincter was extremely derelict, the bowels were obstinately constipated, and there was considerable anæsthesia of the legs. The knee-jerks were markedly exaggerated, and there was now slight ankle clonus on both sides. She continued to get worse, and in a short time was completely paraplegic. When she attempted to stand she had no idea where her legs were, and it was quite impossible for her to take a step. The upper extremities became more ataxic, and she was wholly unable to feed herself. At this time vision began to fail; the ophthalmoscope showed beginning optic atrophy. There was no nystagmus nor paresis of the ocular muscles. After this condition had lasted for a few months the symptoms again became more severe and indicative of much more extensive involvement of the central nervous system. She became completely bedridden; as she lay in bed she had no idea where her legs were or what position they were in. She could not assist herself in any way with the hands, neither to eat nor to help dress herself. All the other symptoms were exaggerated. Soon after this examination of the eyes showed well-marked nystagmus, and vision became so defective that she could not tell the different edibles on her plate. Then there came another accession of severe symptoms, particularly manifest by the addition of bulbar and nuclear symptoms. Speech was so inarticulate as to be completely unrecognizable, there was some difficulty in swallowing, and there was paralysis of the seventh nerve on the right side in all its branches, without reaction of degeneration. There were paralysis of the right external rectus of both eyes and inability to move both eyelids beyond the middle line in looking to the right. When the eyes were moved to the right there was distinct nystagmus. There was marked difficulty of hearing in the left ear. Ankle clonus had disappeared, but the knee-jerks were still exaggerated, the paresis of the legs and the loss of the sense of position being as profound as ever. Titubation of the body and marked ataxia of the upper extremities were present. The optic atrophy had progressed, and the pupils were widely dilated and but slightly responsive to light. In this condition she remains. The mental faculties are intact.

The symptoms in this case indicate a gradual accession of the symptoms, each new development positing the affection of a higher level of the cord, until finally the oblongata became affected.

The treatment of this disease is not unlike that accorded Friedreich's disease. The most insistent measures should be directed to the care of the bladder, bowels, and skin. The general nutrition of the patient must be constantly bolstered, and his or her comfort contributed to by the use of orthopædic and supporting apparatus.

THE SURGICAL TREATMENT OF ABNORMALITIES OF THE BRAIN AND SPINAL CORD.

BY P. S. CONNER, M.D.

THE treatment of abnormalities of the brain and spinal cord has of late years become more and more radical. Improved technique, operative and post-operative, has secured a high degree of protection from septicity, and, as has long been known, septic infection of one sort or another has been the chief cause of death in the great majority of cases treated surgically.

Cephaloceles (both hernias and dropsies) have in a limited number of cases been removed. Though experience thus far establishes in a general way the truth of Tillman's declaration that encephaloceles and hydroencephaloceles are unsuitable for operation, yet evidence is accumulating showing that if thorough asepsis be maintained until the wound of operation is healed, the patient may be expected to recover, even though a portion of the brain has been taken away. As indicated in the original article (vol. iv. p. 741), the protrusion is at times composed of a meningeal neoplasm and not brain-substance at all, and in other cases, though brain-substance is present, it is so greatly modified that its removal will not materially affect the mental or physical condition. Occasionally, normally or practically normally developed brain-substance is present in the tumor, as, *e.g.*, the cerebellum, which Mayo-Robson "found lying in the sac and attached by a long pedicle." What the nature of the more solid protrusion is may be determined by an exploratory opening of the sac and digital and visual examination of its contents. Meningoceles may be quite safely subjected to operation, and such tumors should be so treated, certainly when large and tense. When the mental state of the child is one of decided impairment no change for the better can reasonably be expected, even though recovery from the operation itself takes place.

In removing these tumors, after skin-flaps have been dissected off the sides, a meningocele may be ligated at its base and the protruding portion cut away, or, better, flaps may be made from the sac sufficiently large to readily come together and close the opening, the skin-flaps being then united over them. When the tumor contents are solid in part or whole, the operation has been done by basal ligaturing and removal in mass, or, preferably, by opening the sac, ligaturing and removing what it was thought might properly be taken away, and returning the stump of the protrusion so far as it could readily be effected. Whatever operative procedure is adopted, it should be remembered that "unless the operator is prepared to take every pains, both in the details of the operation and in the observance of antiseptic precautions, the older rule of non-interference had better be observed." (Mayo-Robson.)

HYDROCEPHALUS.

In cases of chronic ventricular dropsy tappings have, as before, been frequently performed, but with very slight success, permanent recovery being extremely infrequent, if, indeed, it has occurred at all. Lateral tapping after the method of Keen (referred to in the original article, vol. iv. p. 747) has been done in a number of instances.

In 1893 Park operated at the base posteriorly, gouging away the bone one inch below the superior curved line of the occiput and one-half inch to the right of the median line, incising the dura mater and with a probe opening up the subarachnoid space. Observation has shown that this latter step is probably not necessary, as after the incision of the dura mater quite free sweating of the subarachnoid fluid may be expected. "Drainage when once established can be made efficient if septic infection can be prevented" (Park), and in proportion as this can be done, continuous evacuation of the fluid is better than interrupted.

Tapping the ventricle, whatever the method employed, should be done only in cases of moderate distention without much, if any, enlargement of the head, or as a desperate remedy in a hopeless condition, when the distention is great and the head enormously enlarged. In 1891 Wynter and Quincke reported upon the benefits of lumbar puncture of the subarachnoid space, and in a number of cases of chronic hydrocephalus it has been found practicable to thus drain the ventricles; not, however, in all, for at times there is without doubt closure of the communication between the ventricular and subarachnoid cavities. An ordinary aspirator-needle is passed through a lumbar interlaminar space (preferably the third, or through the lumbosacral space, which is somewhat larger) and may be entered obliquely or directly from side to side or from below. Fürbringer has shown that in infants it is possible to drive the needle through the vertebral cartilage into the aorta. Though the puncture may be made without the use of an anæsthetic, it is better to give one. The probabilities are great that lumbar puncture will, like puncture through the head, be very often followed by septic infection and death; yet it is possible to keep the wound aseptic (*e.g.*, in Park's case, recently reported).

SPINA BIFIDA.

The experience of the last few years has demonstrated the advisability of the treatment by excision of those cases of spina bifida in which operative interference should be made,—viz., those in which the tumor is (1) not small and protected by a covering of well-developed skin, (2) those in which there is not an extensive vertebral cleft, and (3) those in which there is not any associated malformation itself imperilling life. The injection treatment for a time so largely employed has been very generally given up. Almost all surgeons are now disposed to look upon the spinal hernia, so far as its radical treatment is concerned, much as upon an abdominal one: its cure to be secured, if possible, by the return or removal of such nerve-contents as may be present, the cutting away of the sac, and the closure of the vertebral opening. The chief danger is of septic infection, and it is because of the increasing probability of preventing such infection that the operation has so largely grown in favor during the last few years. However, even if a thorough aseptic operation is done, the location of the wound (generally low down in the back) makes the maintenance of an aseptic condition quite difficult, and not seldom there is an added risk of infection because of leakage of the spinal fluid along the line of junction of the flaps. In certain cases this fluid, not being able to make its way out, gravitates under the lower flap and forms a secondary tumor. Unfortunately, very often recovery from the operation does not mean recovery of the patient, a hydrocephalus being more or less rapidly developed which carries off the patient, just as takes place at times after spontaneous cure. As might naturally be expected, the prognosis of operations upon adults is less grave than of those upon infants and young children. Stated in a general way, the death-rate in the last few years has been between twenty and thirty per cent. (twenty per cent. in Mayo-Robson's twenty cases, twenty-two and a half per cent. in the thirty-one cases tabulated by Powers, thirty per cent. in Broca's ten cases, and thirty per cent. in the one hundred and three cases collected by Rhein).

The technique of the operation has varied somewhat according to the character of the tumor and the size of the vertebral opening,—in all cases, if possible, skin-flaps being dissected up on each side of the protrusion to an extent sufficient to permit of their edges being apposed. In simple meningoceles (and these constitute a majority of the cases), if the pedicle of the tumor is quite small, it may be ligated or sutured and the sac cut away; if otherwise, meningeal flaps should be formed and carefully brought together. When there is evidently a portion of the cord included, the sac had better be opened and the nerves separated and returned, if possible, into the canal, though in a number of cases on record they have been cut away without detriment to the patient, chiefly in those instances in which the cauda equina was the part of the cord involved (*e.g.*, in the case reported by A. F. Jones). When the vertical opening is other than quite small, a question arises as to the advisability of endeavoring to effect closure of it in some way additional to the application of the flaps of the soft parts. So far as can be determined at present it may be held that, while only exceptionally necessary, in a limited number of cases it is of much advantage to fill up the gap with some material taken from the neighborhood of the opening or introduced altogether from without. In the original article (vol. iv. p. 745) it was stated that Davidson had used a piece of sponge and Mayo-Robson a piece of rabbit periosteum, while Dollinger had done an osteoplastic operation, breaking down, bending in, and suturing together the everted arches. During the present decade a number of reports have been published of the use of various agents to secure and maintain closure. Among those from without may be mentioned the scapula of the rabbit, decalcified bone-plate, and celluloid, each of the organic materials probably undergoing pretty rapid absorption. Future experience will very likely show that some form of plastic operation closing the gap by tissues taken from the vicinity of the spinal opening will prove more advantageous. Reference has already been made to Dollinger's osteoplastic resection. Senenko loosened a piece of the sacrum on each side of the opening, brought the pieces in direct contact, and sutured them. Bobroff chiselled off a piece of the iliac crest (leaving it, however, attached to the erector spinæ muscle), turned it over, and attached it to the freshened edges of the gap in the sacrum. Bayer in two cases of lumbar cleft made a semilunar flap of the aponeurosis and the erector spinæ mass on either side of the vertebral opening and stitched the inner edges together, effecting firm closure, and advised the use of similarly cut aponeurotic flaps in cases of sacral opening.

Even in cases where spontaneous cure has taken place it may become necessary at a remote period to operate for the relief of conditions consequent upon pressure of the scar tissue upon the cord, as in the case of spina bifida occulta reported upon by Jones, of Manchester, England, in 1891.

In all spina bifida operations the head should be kept low and the outflow of the cerebro-spinal fluid limited as much as possible to prevent the shock (which has often proved fatal) from its too rapid evacuation. Every care should be taken to secure close apposition of the edges of the flaps, in order that, if possible, leakage may not occur, since leakage is extremely likely to prevent the maintenance of the aseptic condition which is absolutely essential to the success of the operation.

OPERATIVE TREATMENT OF THE BRAIN AND SPINAL CORD.[1]

By CHARLES B. NANCREDE, A.M., M.D., LL.D.

INCISED, LACERATED, AND CONTUSED WOUNDS OF THE SCALP.

Incised Wounds.—Because of the risks mentioned,[2] and lest the wounds of the soft tissues be only the smaller part of a severe head-injury, the following precautions should always be observed before any examination of a scalp-wound.

After thorough sterilization of the hands and instruments and irrigation of the wound, *without further disturbance*, a loose compress, wet with some efficient germicidal solution, should be placed over or lightly packed into the wound. Next the scalp should be shaved or cleared of hair by the scissors for at least one inch around the wound, preferably much farther. The scalp should then be carefully scrubbed with a sterilized nail-brush wet with something calculated to remove all oily substances, as turpentine one part, alcohol seven parts, alcohol alone, ether, or chloroform, so that germicidal agents such as mercuric chloride can act. Abundance of hot water and soap must then be employed, after which the wound must be thoroughly flushed with some efficient germicidal solution, preferably one of carbolic acid, because this agent is not decomposed by any remains of the soap, and even in the presence of oily matters acts upon germs. Prolonged, gentle friction with sterilized (boiled) water and abundance of soap and free irrigation with water as hot as can be used will sometimes secure an aseptic course for the wound when no better facilities for disinfection are accessible.

Renewed disinfection of the hands must precede this disinfection of the wound itself, which now should, for the first time, be explored by the finger or probe. If the slightest doubt exist as to the presence of a fracture, the wound must be sufficiently enlarged to determine this and secure disinfection.

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[1] When neither modifications nor abandonment of recommendations made in the first article (vol. iv. p. 748) are requisite, it must be understood that the views formerly taught are reaffirmed. Occasionally, even when only slight changes have seemed necessary, lucidity has been secured, it is believed, by rewriting the whole or part of a section.

Reference to this same article in vol. iv. p. 748 will explain the solicitude evidenced by the additional precautions recommended for the treatment of scalp-wounds.

[2] Vol. iv. p. 748.

Careful search revealing neither foreign bodies nor fracture, any hemorrhage which cannot readily be controlled by compression must be checked by twisting or tying both ends of the divided vessel. When the ends retract into the dense fibro-cellular tissue of the scalp, they should be secured by passing a needle armed with a ligature around the vessel, including some of the surrounding tissues. Ligatures will rarely be necessary in children except when the wound involves the lower part of the temporal fossa, in which case the bleeding may prove difficult to arrest. "Should the bleeding recur or become dangerous, notwithstanding all our local means, the question of applying a ligature to the external or common carotid may arise."[1] I quote the foregoing sentence not because in my practice any such contingency has arisen, but because so experienced a surgeon as Hewitt has evidently known of some such question arising.

All bleeding having been arrested except that which is to be checked by the compression of the dressings, the wound, if it has not penetrated the aponeurosis of the occipito-frontalis muscle, should be closed by fixing one end of a strip of aseptic gauze or shreds of absorbent cotton with iodoform- or ordinary collodion upon one side of the cut, when the wound can be accurately coaptated, and held so by painting the other end of the strip with more collodion. The use of adhesive plaster is absolutely contra-indicated because it soon becomes loosened by the growth of the hair and asepticity of the wound is rendered impossible.

When the wound extends through the occipito-frontalis aponeurosis, sterilized catgut, silk, or horse-hair sutures will often become necessary because of the gaping of the wound, but in accidental traumatisms or after operations where asepsis is doubtful the centre of a bunch of the finest catgut (non-chromicized) had better be secured by a suture of the same to the deepest portion of the wound, three or four strands being brought out between each pair of sutures.[2] In all varieties of scalp-wounds dusting with sterilized iodoform, laying a piece of protective along the line of the wound,—this is imperative if capillary drainage has been instituted, —and the adjustment of appropriate sterilized gauze compresses to secure contact of the flap with the deeper parts had better be resorted to, covering all in with many layers of aseptic or antiseptic gauze, the innermost of which it is safer to moisten with some efficient germicidal solution. The retention of the dressings must be secured by the application of aseptic bandages, which, if of ordinary muslin, must have been sterilized by dry[3] —not moist—heat.

Should suppuration occur beneath the aponeurosis because of neglect of proper aseptic precautions or from primary infection of an accidental wound, as capillary drainage will not remove pus, tube-drainage must be substituted, compresses being so adjusted as to prevent accumulation of the pus. Counter-openings are indicated, kept patent by the introduction of drainage-tubes, except in the rare event of some portion of the wound being so situated as to afford drainage of the most dependent portions of the pus-cavities. If the surgeon will remember that this aponeurosis is attached behind to the superior curved lines of the occipital bone, to the mastoid process of the temporal bone and the zygoma laterally, and that it becomes continuous with the tissues of the upper lids and those over the root of the nose, together with the position in which the disease will compel the patient to maintain the head, the proper places to make counter-openings can readily be determined in each case. Most strenuous efforts must be made by the use of peroxide of hydrogen and other antiseptic solutions to render the pus-cavities aseptic, and all bagging of pus must be prevented by proper compresses, for by such means oftentimes a rapid diminution in discharge, pain, and constitutional symptoms will result. Sloughing may occur, but is rarely productive of any osseous necrosis, especially if absolute or relative asepsis be promptly secured.

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[1] Hewitt, Holmes's System of Surgery, third edition, vol. i. p. 570.

[2] See vol. iv. p. 757 for proper method of employing.

[3] Not less than 150° C. for one hour

Subaponeurotic suppurative cellulitis is often mistaken for erysipelas, and doubtless may sometimes be of this nature. Although in the non-erysipelatous form some reddening of the skin may extend for a short distance beyond the points of attachment of the aponeurosis, the ears, cheeks, and face remain free. The pus can gravitate no lower than the zygoma, the upper eyelids, the attachments of the pyramidalis nasi, and the superior curved lines of the occipital bone, where it forms pouches.

Constitutional Treatment.—Regulation of the secretions by calomel and soda, followed by laxatives if the bowels do not act properly, with stimulation of diuresis by the free use of fluids, is all that is usually necessary. Nutritious, easily digested food must be given in proportion to the digestive powers. Quinine, strychnine, and stimulants may in rare instances become indicated to combat exhaustion. Iron will prove useful during convalescence. A sustained high temperature which does not promptly subside after evacuation of pus, drainage, and disinfection must be lowered by regulated bathing, not by antipyretics.

CONTUSION OF BONE, OSTEOMYELITIS, OSTEOMYELITIS FROM MIDDLE-EAR DISEASE, ETC.

While nothing additional in a general way is requisite to that given in the first edition, the proper treatment of sinus and jugular thrombosis has undergone much improvement.

A few points of technique which are applicable in whole or in part during any cranio-cerebral operation should be thoroughly mastered. Great diversity of opinion exists as to the best means of gaining access to the interior of the skull. The use of the trephine has been absolutely interdicted as a dangerous instrument by some. Others vaunt the chisel and mallet as those only to be used. Some neurologists, on the contrary, declare that the use of these instruments produces most serious immediate effects upon the brain. Others contend that some of the many forms of saws or craniotomes are the only proper instruments to employ. Such exaggerated and one-sided statements are both unwarranted and unscientific. Given certain conditions, then some one method of removing the bone *is* superior to any other, but no one method can possibly be the best for all other and often widely differing conditions. The surgeon had better never restrict himself to any one form of instrument, but employ that which seems best adapted to the special conditions presented by each case.

An essential prerequisite for many of the operations done upon the skull and brain is a reliable method of cerebral localization. This should be applicable to patients of every age, to every type of skull, and one that requires no special apparatus.

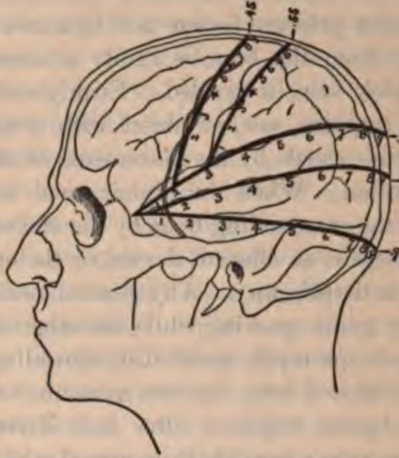
Chipault's method, now to be given, seems to fulfil the foregoing conditions better than those described in the first edition of this work, although I have as yet had but one opportunity of testing it upon the living subject.

Only three readily ascertainable points of reference must be localized,—viz., the inion, the nasion, and the upper border of the retro-orbital tubercle of the zygoma.

First trace an antero-posterior median line upon the scalp from the nasion to the inion. Then measure from before backward points corresponding to .45 of this line, which will give the pre-Rolandic point, .55 the Rolandic point, .70 the Sylvian point, .80 the lambdoidal point, and .95 the supra-iniac point. For example, to determine the upper end of the fissure of Rolando in an individual where the naso-iniac line measures .30 centimetre, multiply this by 55, and considering the last two figures of the result as decimals, 16.50 centimetres measured on the sagittal line posterior to the nasion will give—1 centimetre from the median line—the point sought. Lines drawn from the upper border of the retro-orbital tubercle to the points corresponding to .70, .80, and .95 of the naso-iniac line indicate with much accuracy the following points: the Sylvian fissure, the first temporal convolution, and the posterior portion of the lateral sinus. Connect a point on the Sylvian line two-tenths of its length posterior to the retro-orbital tubercle with that on the sagittal line corresponding to .45 of its length. This new line, starting below at the commencement of the vertical limb of the Sylvian fissure, corresponds in its upper portion to the precentral fissure. A second line extending from the junction of the third and fourth tenths of the Sylvian line to .55 of the sagittal line posterior to the nasion indicates the course of the fissure of Rolando. A division of the Rolandic and Sylvian lines into tenths will enable the surgeon to determine any other point of cerebral topography. (See Fig. 1.) Thus, as Chipault says, the foot of the second frontal convolution will be found at the junction of the third and fourth tenths of the pre-Rolandic line. If this measures 13 centimetres, then one-tenth = 1.3 centimetres; the point sought is, therefore, 1.3 × 3 = 3.9 centimetres from the inferior extremity of this line.[1]

It is requisite to be acquainted with the arrangement of the chief vessels of the pia mater, especially the larger veins. These are more superficial than the arteries, the latter, with few exceptions, lying in the depths of the sulci. The chief vein of surgical interest is that running along the horizontal limb of the fissure of Sylvius, whence it passes over the surface of the hemisphere to empty into the superior longitudinal sinus near the parieto-occipital fissure. Two branches of this Sylvian vein pass upward to empty into the same sinus, one following the course of the fissure of Rolando, the other approximating that of the post-Rolandic sulcus. A third large vein also descends from the Sylvian vein to the lateral sinus. Moreover, several large veins cross the frontal convolutions to enter the anterior segment of the superior longitudinal sinus. Prior to entering the sinuses all of these veins pass between the layers of the dura mater and form sinus-like dilatations.

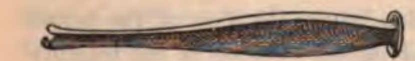
FIG. 1.



From Chipault's Chirurgie opératoire du Système Nerveux, t. i., Fig. 159.

The radicles of the veins occupy the fissures, or lie directly upon the convolutions. The inferior cerebellar veins are the only ones in this region likely to be injured in any surgical procedure; they run transversely outward to empty by two or three trunks into each lateral sinus. The arteries throughout most of their course occupy the depths of the sulci. The principal ones correspond in a general way to the more important veins, and lie from one to two centimetres deeper than these veins, being thus unlikely to be injured except in a few localities, unless the convolutions are pressed apart. At certain points the arterial trunks become more superficial because they occupy a shallow fissure or cross a convolution to dip again into the next sulcus. One large artery apt to be injured when removing the face centres is usually found lying upon the inferior fronto-parietal convolution, or on the foot of the third frontal gyrus. Another emerges posteriorly from the Sylvian fissure to reach the inferior parietal lobule or the

FIG. 2.



Craniotome of Chipault.

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[1] Mention is not made of Chiene's ready method of ascertaining the course of the fissure of Rolando, because not well adapted for the heads of children, in whom this fissure runs at a different angle.

angular gyrus. The deep parts of the anterior portion of the fissure of Sylvius are an especially dangerous neighborhood.

The control or arrest of hemorrhage during cranio-cerebral operations require careful consideration. Although a rubber tube drawn tightly around the head sometimes lessens the flow of blood, it so often fails to do so that pressure forceps and ligatures must usually be relied upon. Bleeding from the bone is rarely of consequence unless one of the principal diploic veins is divided. Firmly and repeatedly packing the groove made by trephine, saw, or chisel with antiseptic wax, which is forced into the diploic canals by the movements of the instruments, will usually check the effusion. When the diploic canal is accessible, concentric crushing into it of the surrounding bone by the end of an elevator or point of a hæmostatic forceps is an efficient device, or the opening can be plugged by a bone-chip or antiseptic wax. Any general oozing which does not cease after temporary gauze packing while the other steps of the operation are being taken can be promptly arrested by spreading a thin layer of antiseptic wax along the incised bone by firm pressure with the finger. Dural vessels should be ligated before or after their division by passing a needle through the dura mater beneath them armed with a fine gut ligature. Wounds of the great venous sinuses can be plugged with a bunch of catgut, or compression for two or three days effected by gauze packing can be safely relied upon, very little pressure being requisite. Lateral ligation or suture has succeeded, but is difficult and unnecessary. When a segment of a sinus demands excision, it must first be included between two ligatures passed by an armed needle through small incisions made in the dura mater parallel to the sinus,[1] this device facilitating the introduction of the ligatures and preventing tearing of the dura mater. Because many of the vessels of the pia mater are terminal it is important to avoid their permanent obliteration; hence the pia mater should be carefully lifted out of the sulci, incised so as to divide as few vessels as possible, and, being gently held aside, should be laid down upon the subjacent parts at the close of the operation. As has been pointed out,[2] some thrombosis may result from this procedure, but may also produce only temporary obstruction of the vessels, and this risk is better than the certainty of the permanent obliteration produced by division of the vessels. Preliminary ligation before division of the larger veins and arteries of the pia mater is advisable. Owing to the fragility of their walls and the absence of sheaths, fine catgut threads should be used, drawn only tight enough to occlude the lumen of the vessels, care being exercised when tying the second half of the knot lest the end of the vessel be cut or pulled off. The temporary application of wire serrefines will often insure the formation of an efficient thrombus by the time the wound must be closed. When bleeding recurs after the removal of the serrefines and ligatures repeatedly cut off the ends of the vessels, the reapplication of serrefines with threads attached to them to facilitate removal after their spontaneous separation at the end of a few days is preferable either to packing with gauze or leaving hæmostatic forceps *in situ*. Bleeding from vessels in the cerebral substance which does not cease after sponge pressure must be arrested by the same methods, when a ligature is employed, including, when passing the threaded needle, a little of the surrounding brain-substance. Gauze packing may sometimes be employed, but is often inefficient, and is objectionable because it prevents proper closure of the dura mater and external wound. Although closure without drainage is safe for aseptic wounds, even when considerable accumulations of blood or serum may occur, where no mechanical harm can result, yet this cannot be affirmed of many brain-injuries and operations. Moreover, intracranial infection is such a well-nigh hopeless condition, unless perchance its evils can be averted by removing its products, that in doubtful cases, especially after accidental traumatisms, all prudent surgeons will employ some form of drainage. For pus only tube drainage is reliable. Many agree with myself in draining a bullet track, certainly if it has been explored. When the brain-tissue has been much injured, so that much oozing is to be expected, in many localities drainage is the safer course, and becomes imperative if a lateral ventricle has been opened or this accident is suspected. The orifice of a ventricular wound must be lightly packed with a narrow gauze strip to prevent blood eventually reaching the fourth ventricle and producing respiratory failure; one end of the strip must be brought out through the external wound, that the gauze may be removed after forty-eight hours. If merely blood and serum require removal, capillary drainage by fine catgut is better than by gauze, unless packing is requisite to arrest bleeding, because gut is spontaneously removed by absorption.

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[1] Chipault, op. cit.

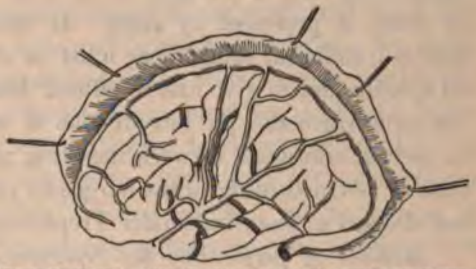
[2] See vol. iv. p. 778.

Wounds of the dura mater complicating fractures or those made to gain access to the brain must be sutured with gut passed by means either of a curved round needle or a surgical one whose edges have been thoroughly dulled up to the point by rubbing on a hone. The needles described are superior to Hagedorn needles. If drainage is indicated a portion of the dural wound should be left unsutured. Small defects of the dura may be dealt with by catching their edges with catgut sutures placed close together. When larger portions of the dura have been excised, to prevent adhesions forming between the brain and the bone or scalp, either a piece of periosteum can be sutured into the defect, or, what is in most cases better, because neither difficult nor consuming much time, a piece of heavy gold-foil should be secured between the dura mater and the bone or beneath the periosteum loosened for this purpose around the bone orifice. A celluloid plate will probably do equally well.

A consideration of the motives leading modern surgeons to operate for compound fractures of the skull will indicate certain appropriate mechanical procedures. Because infection probably has occurred, the only means of removing this or minimizing its evils are those which will enable the surgeon to remove the pressure of depressed bone or effused blood and lessen the chances of epilepsy, the former governing indications for operating. Hence the scalp incisions must be so planned as to give the freest possible access to any part, osseous or intracranial, which may have been infected. Any enlargement of the primary wound must be so effected that the line of suturing shall be as far away from the osseous opening as possible, because infection of the external portions of the wound is less apt to reach the interior of the cranium and primary union of the flaps is more probable in healthy tissues. Thus, if a tendency to hernia cerebri exists, the weight of the large flaps before healing and their prompt healing later will restrain the protrusion. Such fashioning of the flaps is often impossible, but when feasible should be adopted. Detached bone fragments should be removed, and, owing to the strong probability of their being infected, their implantation is contra-indicated. When for ulterior purposes partially or completely detached fragments are removed, these, after thorough disinfection, including, when requisite, paring with a chisel, may with reasonable safety be replaced. As later experience proves that after temporary union detached fragments of bone generally become loosened because partially absorbed, and that they may then become a source of irritation, it would seem better to employ a celluloid plate to make good extensive bony defects and prevent adhesions forming between the brain and the superjacent parts. The same remarks are applicable to decalcified bone plates,—*i.e.*, the permanent bony repair is often insignificant. Where disinfection or access to intracranial lesions does not demand removal of sound bone, only enough must be excised to permit safe elevation, or removal, if infected, of depressed fragments. Hence trephine, rongeur, forceps, chisel, or saw may each at times be the best instrument. For simple elevation of a non-comminuted depressed area, if enough overlapping sound bone cannot readily be removed by the rongeur to permit the use of an elevator, the trephine or the chisel may be employed. For cutting out infected fissures or enlarging these to permit elevation of fragments, the chisel is far superior to any saw, and certainly, when the rongeur cannot be employed, is *the* instrument for the removal of overhanging margins of sound skull which interfere with the elevation of an extensive depressed area, less sound osseous tissue being sacrificed. Still further, as operations are often performed far from the conveniences of a fully equipped hospital, the surgeon must be prepared to work sometimes with the simplest tools. While the danger of a wound of one of the great venous sinuses is greatly overestimated when the wounded part is accessible, the fear is abundantly warranted if the opening is concealed beneath solid bone, and the reader is urged to resort to what is really Percival Pott's practice as set forth on p. 756 of vol. iv. The dura mater which includes the superior longitudinal sinus must not be separated for any distance beyond the bone opening, lest the vessel be wounded at an inaccessible point. The sinus-like dilatation of the pial veins, already described as located at some little distance on each side of the superior longitudinal sinus, must also not be forgotten.

For intracranial hemorrhage, when no lesion of the soft parts is present, it is usually better to widely expose the supposed site of lesion by a temporary osteo-cutaneous flap resection, but when there is a probability of infection being present, as in compound fractures where the middle meningeal artery is the source of the bleeding, the older methods of perforating the skull are preferable because they insure freer drainage. The osteo-cutaneous flap can be cut so as to uncover the areas of distribution of the anterior, middle, or posterior branches of the middle meningeal artery, the common source of operable intracranial hemorrhage, or any two contiguous areas, according to the indications. As more practice is requisite to use the chisel safely, the trephine had better be employed by the inexperienced practitioner. If the evacuation of large clots demands removal of large areas of bone by other means than by a temporary resection, for reasons already stated a plate of celluloid, perforated at several points, had better be employed rather than reimplantation of the removed bone fragments. Drainage when used must be capillary, and the necessity for its employment will depend upon whether all the effused blood can be evacuated and whether further bleeding can be with certainty prevented. When ligation of all visible bleeding points will not arrest the oozing, light gauze tamponade may be employed. Hemorrhage proceeding from a wound of the main trunk of the middle meningeal artery at or near the spinous foramen is best arrested by packing into the foramen one end of a narrow strip of gauze by some curved, pointed instrument, as an Allis's dissector. When successful, this expedient is better than a less accurately placed larger packing or ligation of the external carotid artery. Whenever gauze is thus employed free notching of the osteo-cutaneous flap or non-implantation of the trephine button will be requisite to permit subsequent removal of the gauze.

FIG. 3.



From Chipault. The double-contoured vessels are veins, and those with transverse striations are arteries.

SIMPLE FRACTURES OF THE VAULT.

Before any operative interference with a simple fracture special care must be exercised to remove all oily matters from the scalp and to secure the longest possible contact of some efficient germicidal agent with the cleansed scalp. A soft-soap poultice applied for an hour and removed with hot carbolized water, after the more ordinary measures detailed under scalp-wounds have been employed, is the most efficient means to remove oily matters. A moist carbolized or corrosive sublimate dressing should then be applied, to remain until after the induction of anæsthesia. A large curvilinear flap, so planned as to avoid the main scalp-vessel and favor drainage, must be raised, the incision including all the tissues down to the bone. Any depressed fragments of bone must then be raised, after removing a sufficient amount of the overlapping sound bone to enable an elevator to be slipped beneath them. The removal of the overlapping edge of the external table by a Hey's saw or the widening of a fissure by the same instrument or a chisel will often render elevation possible. If the fragments are incarcerated beneath the whole thickness of the surrounding skull, some form of rongeur is the best instrument, although the trephine or chisel is preferred by some. If the inner table is extensively comminuted, sufficient sound bone must be removed to permit the removal of all spiculæ and to give exit to effused blood or exudates. Implantation of the bone fragments, the introduction of a celluloid plate or heavy gold-foil to prevent adhesions between the scalp and brain-membranes having been done or not, according to the surgeon's judgment, the wound must be closed and dressed as described under Trephining.

Methods of performing the Operations of Trephining and Osteoplastic Resection of the Skull.—The term trephining is often employed to include the elevation or removal of bone or perforation of a skull by any operative procedure. Two distinct methods are employed,—viz., one where the bone is actually removed, although sometimes replaced, the other where the bone is temporarily displaced, its chief attachments to the soft parts remaining undisturbed. This latter procedure is not trephining, but osteoplastic or temporary resection of the skull.

The special instruments required for the removal or elevation of portions of the skull, besides hæmostatic forceps, tenacula, and knives, are trephines of various sizes, a pair of Hey's saws, a pair of small cutting bone forceps, a rongeur, elevators, narrow-bladed sequestrum forceps, a bone chisel and mallet, and a flat-ended probe or sterilized tooth-pick. The surgical engine with circular saws or some form of craniotome may also be used, if the surgeon sees fit to employ these instruments. The attempt to restrain hemorrhage by the previous application of rubber tubing so rarely succeeds that I have not enumerated this among the instruments required. In this and all other operations described perfect asepsis of hands, instruments, ligatures, etc., is, of course, presupposed.

A convenient method of supporting and steadying the head is the use of a sand-bag. If the operation is being done for compound fracture, the wound by proper enlargement will give access to the damaged portions of skull, but where there is no wound the scalp should be incised down to the bone with one stroke of the knife, forming a large flap whose outline is a shallow curve so planned as to avoid the main scalp-vessels and favor drainage in the recumbent posture; this should be rapidly reflected. Any hemorrhage not likely to cease spontaneously must be arrested by hæmostatic forceps. The periosteum usually readily strips off with the rest of the flap. The trephine, with the centre-pin protruded about one-sixteenth of an inch and firmly screwed in this position, should now be applied to the portion of bone to be removed, the periphery of the trephine somewhat overlapping the depressed bone at one point if a fracture be present. The instrument should be worked with a light, sharp, quick movement from left to right and from right to left, care being taken not to press unevenly, and the pressure being chiefly exerted as the hand is carried from left to right. As soon as a sufficient groove has been cut to steady the trephine, the centre-pin should be withdrawn and fixed so as to avoid injury to the dura mater. Until the diploe is reached the bone dust is dry, then it becomes bloody; but as this structure is absent in early life, and at all ages over a large part of the squamous bone and in the occipital fossa, the sound and feel must be relied upon to warn the surgeon when he has reached the internal table.[1] The depth of the bony groove must be frequently ascertained at various portions of its circumference to determine whether the bone is being evenly divided or owing to inequalities of thickness the dura mater is bared at some points. If one segment of the groove be deeper than another, or the bone be actually penetrated, the trephine must be inclined towards the shallow side, and pressure made at that portion alone until the groove is of equal depth throughout or the partially sawn bone is completely divided. The segmented trephine may be employed where the bone is of very unequal thickness. The varying thickness of the skull in different individuals always calls for caution, but the entire absence of diploe in many of the children requiring operation, leaving it doubtful whether much or little bone intervenes between the trephine and the dura mater, demands a light hand and frequent examination of the groove by the flat-ended probe. When the sound to the ear and the sensation conveyed to the hand give warning that denser bone—viz., the inner table—has been reached, each turn must be made cautiously and with very light pressure, frequent resort being had to the probe or tooth-pick to determine at which points, if any, the bone has been completely divided, that the teeth may be so inclined as to avoid wounding the dura mater. Incipient loosening of the button can sometimes be ascertained by slightly rocking the trephine; but it is better to lay the instrument aside, and, seizing the disk in the grasp of a pair of forceps whose blades have been inserted in the groove, gently move it from side to side. If loosened at one edge, the trephine teeth must be made to cut upon the opposite, attached part, for a turn or two, when the forceps should again test the stability of the disk. When found sufficiently loose the bone disk must be removed by tilting it out with the sequestrum forceps, using a rocking movement, and always drawing it out towards the side where any portion of the inner table remains unsawn, as then the dura mater escapes injury from the other sharp, thoroughly sawn, or perhaps splintered edge of bone. If any portions of the inner table are left attached to the margins of the opening, they can be removed by the rongeur, the serrated edge of the elevator, or the lenticular. When a large disk of from one and a half to two inches in diameter is to be removed, one of the loosened edges should be gently lifted with an elevator, and the dura mater carefully stripped off with Horsley's instrument or by a smooth, blunt elevator. If replacement of the bone is contemplated, the trephine button and all osseous fragments must be at once placed in warm sterilized water, or wrapped in a warm sterilized towel.[1] Hemorrhage from the bone sometimes proves troublesome during the removal of the button, usually proceeding from one of the large diploic veins. Severe bleeding from this source is unlikely except in adolescents or older children; yet even in the youngest—who do not bear the loss of blood well—very free oozing sometimes occurs, which cannot be arrested by ordinary measures, because not accessible. Filling the groove with antiseptic wax[2] and then resuming the use of the trephine will lessen or totally arrest the bleeding, if repeated sufficiently often, the wax being forced into the orifices of the osseous vessels. Crushing of the diploe surrounding a bleeding osseous vein into the orifice by the point of an elevator or forceps, after removal of the bone, in the absence of wax, is usually sufficient; if not, plugging with a bone chip should be done. Usually firmly packing gauze against the bone edges secures permanent hæmostasis by the time the wound is ready for closure.

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[1] The former recommendation to employ a piece of perforated wood when the centre-pin of the trephine cannot be used to steady the instrument until a groove is cut seems no longer good advice, because I think the chisel, saw, or rongeur had better be employed to remove sound bone to permit elevation of fragments rather than to apply the trephine to depressed bone.

For a punctured fracture a trephine large enough to include the starring of the outer table with a small area beyond should be used, because most of the fragments of the inner table will come away with the button. When removing splinters of the inner table great care must be exercised lest the dura mater or venous sinuses be wounded. For an ordinary depressed fracture the trephine should be applied so that only about two-thirds of the circumference will be located upon the sound bone, and the crown of the instrument should be no larger than is requisite to leave an opening which will admit of the easy introduction of an elevator. In many cases of fracture elevation of fragments can be more readily effected by removing overlapping edges of bone with the chisel, saw, or rongeur. If the removal of one button will not allow of elevation of the fragments, more bone can now be readily excised by the rongeur, Hey's saw, or chisel. Possibly it may be better to use the trephine. Special precautions must be observed when the fracture is near a large sinus which may have been wounded by depressed fragments. Thus, any bone opening must be planned so as to give ready access to any bleeding point rather than merely to permit easy elevation of the fragments. The extraction of a long fragment driven some distance beneath sound bone, whose concealed extremity lies in close proximity to a large sinus and possibly penetrates it, is a most dangerous practice, because direct access to any bleeding point cannot be obtained. It is far safer to trephine over the site of the concealed extremity of bone, or remove the sound bone with chisel or rongeur up to this, when any hemorrhage following its withdrawal can be instantly checked by compression or ligature. Neglect of this rule has often been followed by prompt death, due to uncontrollable bleeding from a wounded lateral or superior longitudinal sinus or one of the sinus-like dilatations of the pial veins,[1] which could have been readily dealt with if the bleeding vessel could have been reached. When compelled to remove bone overlying these sinuses an opening had better be made on each side and the intervening bridge of bone carefully chiselled away until only a thin layer of bone covers the sinus, which can safely be broken away piecemeal by the elevator or cut away with the rongeur. The osseous bridge can also be entirely removed by the rongeur, or the covering bone gnawed away through a single trephine or traumatic opening, but to trephine directly over the groove for one of these great vessels is bad surgery, because unnecessarily complicating the operation by the almost certain wound of the sinus following an attempt to remove a disk of bone varying greatly in thickness at various portions of its periphery. Free removal of bone is usually necessary when operating for intracranial hemorrhage, cerebral tumors, or epilepsy, or after traumatisms when localizing symptoms suggest the gradual involvement of several centres in succession. This can be done by using trephines of from one and one-half to two inches in diameter. The skull can also be perforated by a small trephine at two or more points and the openings connected by the circular saw operated by the dental or electrical engine, by the chisel, or by the wire saw. Again, by carefully chiselling a groove around a portion of the skull, it can be removed by insinuating the points of two or three stout elevators beneath the inner table at points where this has been completely divided, when, by judiciously applied force, the undivided portions can be fractured and the fragments removed, the dura mater being first carefully stripped off. Temporary resection by an osteo-cutaneous flap is often the better practice in intracranial hemorrhage or when attempting to remove a brain-tumor.

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[1] See original article concerning the advisability of these procedures.

[2] Wax, seven parts; almond oil, one part; salicylic acid, one part.

After traumatisms all loose fragments of bone should be removed and depressed pieces elevated to secure *disinfection*. When depressed fragments can be effectually disinfected *in situ*, elevation is alone requisite. Any bleeding from the dura mater, if of consequence, can be arrested by the passage of fine catgut ligatures through the membrane beneath the vessels by a curved needle. Neither subdural blood nor wound of the dura being detected, after smoothing off the edges of the bone opening, the disinfected[1] bone fragments may be replaced. If too small to fill the gap, perforate the fragments with the centre-pin of the trephine and secure them, where possible, in contact with sound bone by means of catgut sutures passed through the under surface of the scalp. At least temporary osteogenesis may also be secured by carefully mincing the smaller bone fragments and dusting them over the exposed dura mater.

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[1] See original article.

Closure of Openings by Celluloid or Gold Plates.—Osseous defects may be mechanically repaired by the insertion of celluloid plates fitted to the shape of the opening, but notched at one side for drainage, or a heavy piece of gold-foil inserted between the dura mater and the bone, or between the brain and bone when the dura mater has been removed. Such procedures are only adapted for cases where all chances of infection can be excluded. The object in view is to prevent fretting adhesions forming between the brain or its membranes and the soft parts or bone. When possible, as already mentioned, the dura must be sutured with fine catgut. Since later experience seems to have demonstrated that after replacement of *detached* pieces of bone the osteogenesis is either temporary or very incomplete, and because adhesions cannot form between the soft parts and gold-foil or celluloid, heteroplasty is probably always better than replacing totally detached osseous fragments. The celluloid may be fitted to and sprung into the bone opening or be slipped between the dura and the bone, or beneath the periosteum around the opening, which must be detached for a short distance to facilitate this manœuvre. Drainage (temporary) is best secured by suturing the middle of a bunch of fine gut to the deeper parts of the wound.[2] Silkworm-gut or medium-sized silk is the best material for suturing the scalp, the stitches being placed about one centimetre apart. Between each pair three or four of the catgut threads should be brought out, cut squarely off, and kept in contact with one another (see vol. iv. p. 757), not sprayed out. Superficial sutures of horsehair or fine silk may be employed to insure more perfect coaptation. Protective to keep the catgut drains moist—without which precaution they will fail to drain—and voluminous folds of sterilized or antiseptic gauze and cotton, secured by a sterilized bandage, complete the dressing.

Hemorrhage coming from a large sinus must be combated by the means suggested in vol. iv. p. 756, and the only additional procedure to be recommended for middle meningeal hemorrhage is that of plugging the spinous foramen with the end of a narrow strip of gauze, as directed in vol. iv. p. 757.

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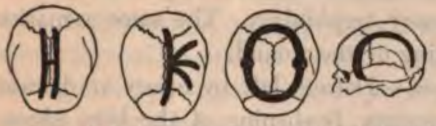
[1] By paring off with chisel or cutting forceps every particle of suspicious bone; otherwise replacing the fragments is only inviting disaster, because one of the chief modern indications for operating for compound fractures is to secure disinfection, and so avert septic or infective encephalitis.

[2] Drainage can often be secured by leaving an inch or more of one angle of the wound unsutured, but the capillary drain is equally safe and more efficient.

All antiseptic irrigations of the brain must be avoided, as already mentioned, because they lower vitality and thus favor infection by diminishing the resistance of the tissues; moreover, they are never requisite during an aseptic operation. It is possible that, when distinct infection of the brain or its membranes exists, they may prove more beneficial than simple irrigation.

Craniectomy.—It has been suggested by Park that these operations be done in two sittings. The first consists in incising the scalp, separating it from the pericranium, and checking all oozing by packing the wound with gauze, a few days later removing the bone after some one of the methods indicated below in the diagrams. It has been still further recommended that the dura mater be widely incised. When this is done, Park suggests that the bone be removed at the first operation, the wound packed with gauze, and then, a week or more later, if "the wound is absolutely free from possibility of infection," the dura can be freely incised and the wound closed. Contrary to the rule in trephining, all periosteum along the lines of bony incision must be removed, to prevent, as long as possible, the filling of the gaps with bone. Numerous bone incisions are preferable to more limited ones with manual divulsion of the cranium as practised by some,—*i.e.*, after one or more longitudinal exsections of bone, forcibly widening the gaps, which may result in fracturing of the base, and certainly risks an undesirable amount of shock.

FIG. 4.



Some of the lines of removal of bone advocated by Lannelongue, Park, and others instead of the simple antero-posterior one. (See Fig. 17, vol. i., Park's Surgery, for additional methods.)

Osteoplastic Resection, or Temporary Resection, of the Skull.—This substitute for trephining is due to Wagner, who in 1889 acted upon the suggestion of Wolff. This operation when not contra-indicated, as stated in the preliminary considerations on p. 1250, is superior to trephining in that a large exposure of the brain is secured, which leaves no material defect in the skull, while the extent of the possible adhesions is reduced to a minimum. A chisel requires more skill than the trephine, but in reasonably careful hands is as safe. Unless done with some form of circular saw, there is both a theoretical and, in a few cases, practical disadvantage in the repeated blows of the hammer when a chisel is employed. A large horseshoe or omega-shaped flap, with a base containing a good vascular supply, is cut down to the bone, which is then chiselled through all around, except at the base, by means of special chisels, as those of Hartley or Pyle, or by a fine carver's gouge or an ordinary narrow bone-chisel. Usually at one or more points the bone is actually completely divided, at which spots two or three stout elevators are introduced and the bone is fractured across the base. Some of the irregularly fractured parts of the internal table around the opening must not be removed—in fact, none—unless absolutely indicated for a special purpose, so that the flap when laid in place will not sink down upon the brain. Chiselling across the base of the osseous flap renders fracture easier, but perils the vascular supply unless Chipault's chisel (Fig. 2) be employed. When any form of saw is employed, one or more preliminary perforations with a small trephine will be requisite to determine the thickness of the skull, so as to avoid wounding the dura, which had often better be carefully separated along the proposed track with a blunt instrument and protected by a strip of metal. A gigli wire saw may be passed along a grooved director extending between two trephine openings and the line be readily divided without injury to the dura mater. As each one of the special craniotomes or saws requires a special technique to employ them safely, nothing beyond these general directions is possible. If special drainage is requisite which irregularities of the bony flap will not admit of, this must be notched with a rongeur. The manner of dealing with the dura mater, pia mater, and brain varies in no respect from that described as applicable during ordinary trephining. The same remarks are also true of the closing and dressing of the wound.

Osteoplastic Repair of the Cranium.—Defects left by injury or disease can be repaired by removal of the cicatrix, freshening of the bone edges, and transplantation from the contiguous parts of a properly planned flap consisting of skin, periosteum, and outer table of the skull, the latter being chiselled loose. The surface left by the removal of the flap should be covered with skin-grafts after Thiersch's method.

Abscess of the Brain.—The advice given in vol. iv. p. 770, to replace the bone button if a second opening is made for better drainage, seems more than questionable in the light of recent experience, since even without infection bone-implantation so seldom results in permanent union, and when this fails the bone is a constant source of irritation, which results in a dense cicatrix blending brain, membranes, and scalp into one,—a not uncommon cause of epilepsy.

OPERATIVE TREATMENT OF EPILEPSY.

Any scar in the scalp must first be freely excised if the disease has resulted from traumatism, especially if pressure on the scar produces a paroxysm commencing on the side of injury. If this fails, or during the operation depressed or thickened bone is detected,—provided the epilepsy is not Jacksonian,—the affected portions with some of the surrounding healthy bone should be removed. If a scar in or distinct thickening of the dura mater exist, the portion involved must be excised with such underlying portions of the brain-substance as are involved in the cicatrix, when possible merely dissecting the membrane off the surface of the convolutions, thus dividing the smallest possible number of pial vessels, the majority of which are terminal. A piece of gold-foil or rubber tissue placed between the dura and the brain may, by preventing the reformation of adhesions, cure the epilepsy without the removal of any portion of the tex. Any distinct cicatrix, tumor, cyst, or localized portion of diseased tex should be excised, the requisite incisions being clean-cut and, when sible, vertical to the surface of the convolutions and at right angles their long axes. If during the operation the cortex is exposed, no iseptic solution should be permitted to come in contact with it, lest its sitiveness be impaired in spots, rendering it difficult or impossible to cessfully locate any centres by electricity; there is no objection to em- ying sterilized normal (0.6 per cent.) salt solution.

Previous to any operation for Jacksonian or focal epilepsy the course of fissure of Rolando and that of the fissure of Sylvius must be marked h nitrate of silver on the shaven scalp, and their extremities indicated on skull, before reflecting the flap at the operation, by a drill or the centre- of the trephine. A large flap having been reflected, as already de- bed, or a temporary resection having been effected, nothing but sterilized solution must be used during the remainder of the operation. The a mater must be carefully incised around four-fifths of its circumference, ut three-sixteenths of an inch from the osseous opening, any vessels ing been previously secured by fine gut passed beneath them by a curved lle. The discharging lesion may now be located by a delicate electrode vol. iv.), employing as weak an interrupted current as possible. The mater should next be carefully elevated out of the sulci where the pro- d incisions are to be made, after such preliminary incisions—avoiding e vessels—as may be deemed requisite. Serrefines or immediate liga- with gut drawn just tight enough to close the lumen of any wounded ls should now be used. Next excise the proper portion of the centre ncisions made at right angles to the long axes of the convolutions. ss the dura mater be diseased,—when its excision is indicated,—after ully replacing the pia mater when all hemorrhage has been arrested, ormer membrane should be carefully sutured with fine catgut. Either e catgut capillary drain must be placed between the scalp and dura r or sutures must be omitted for about an inch from one angle of wound. Suturing and dressing, as already described, complete the tion, after the introduction of gold-foil or rubber tissue to prevent ions.

SURGERY OF THE SPINAL COLUMN.

aminectomy in Pott's disease has been recommended and employed sfully to relieve the cord from pressure, but its technique differs in no t from that recommended for the removal of tumors, except that iodo- gauze packing is indicated rather than closure of the wound with simple or tube drainage. The shock induced by repeated blows of the hammer using a chisel has proved so objectionable that those instruments should employed, but the rongeur and saw. Osteoplastic flaps are not to be mended. Access to the bodies of the vertebræ may be demanded for s of the bones composing the spinal column, for tuberculous osteitis, and for infective osteomyelitis. The requisite incisions and details of procedure vary with the region. The method as devised and employed by Treves for lesions involving the lumbar spine will be described first, because it illustrates all the principles involved in operations upon the dorsal and cervical vertebræ.

Exploratory Operations on the Lumbar Spine; Sequestrotomy.—Make a vertical incision two and a half inches to one side of the lumbar spinous processes, its mid-point being equidistant from the iliac crest and last rib; the length should be from two and a half inches upward. Expose the erector spinæ muscle by incising the aponeurosis of the latissimus dorsi for the full extent of the skin-incision. Draw forcibly towards the median line the outer border of the erector spinæ by means of a broad retractor. Search through the anterior layer of the sheath for the transverse processes of the lumbar vertebræ, the most readily detectable being that of the third. Divide the anterior layer of the sheath vertically as close to the tranverse processes as possible, thus exposing the spinal origin of the quadratus lumborum. Cautiously incise the muscle to the full extent of the skin-wound close to the tips of the spinous processes, carefully avoiding the abdominal branches of the lumbar arteries, which "often are as large as the lingual." Most of these run behind the quadratus lumborum, but "that from the first vessel runs in front, and not infrequently those from one or two of the lower arteries follow its example." The main trunks, after passing over the vertebral bodies, run between the transverse processes, where also the division occurs; hence, by hugging the tips of the processes and "reaching the spine along a transverse process" the vessels escape injury. As soon as the quadratus lumborum is incised the psoas muscle is reached, when by dividing some of its tendinous fibres close to a transverse process the finger can be introduced beneath the muscle until the front of the vertebral bodies is reached; the primary incision can then be cautiously enlarged, by blunt dissection when possible. Often as soon as the quadratus lumborum muscle is divided an abscess-cavity is reached which will serve as a guide to the diseased bone. Treves thinks that, while more inconvenient for the surgeon, there is less risk of wounding the peritoneum if the operation is done on the left side. If operating for tuberculous disease, having opened the abscess, the condition of the bones must be determined as far as possible by the finger, a probe being used with great caution, lest the anterior wall of the cavity be perforated, opening the peritoneal cavity. For the same reason, if a curette is used it must never be directed forward, but only against the posterior portion of the cavity. The finger-nail, or gauze, or a marine sponge held by long forceps is perfectly safe and usually sufficient to remove all caseated material and the tubercular lining of the cavity and to break down any septa which it is safe to rupture. Every recess and pocket must be thoroughly cleansed by scrubbing with the gauze or sponge. Free irrigations with many gallons of the selected antiseptic solution must precede and follow each scrubbing of the cavity, which must be repeated until the surgeon believes that the cavity has been practically converted into a fresh wound, when it may be closed with deep silkworm-gut sutures, including most of the muscular tissues, after leaving a proper amount of iodoform emulsion in the cavity or not, according to the conditions left. If all tubercular material has been removed, no recurrence will take place; if this is in doubt, iodoform may yet insure a cure. Voluminous dry aseptic dressings must be employed to secure as far as possible coaptation of the walls of the cavity; these dressings may be left undisturbed for many days. The recumbent posture, with or without the use of a plaster jacket, must be maintained for months. Should the abscess refill, the operation should be done as if for a previously unoperated case. When poisonous antiseptics are employed for irrigation, the cavity must be carefully flushed with sterilized water to remove all traces of mercuric chloride or carbolic acid. Sterilized water to which enough tincture of iodine has been added to render it of a light straw color is better than corrosive sublimate or carbolic acid, because more active against tubercle bacilli. Of course, operations for wounds of the vertebral bodies or for osteomyelitis will require drainage, but the indications differ in no way from those inducing the surgeon to employ this measure in other regions of the body.

Operations on the Cervical Spine.—Incise the skin along the posterior border of the sterno-cleido-mastoid muscle for a distance corresponding to the depth of the vertebræ to be reached. Saving all the superficial nerves which can be avoided, ligate all veins traversing the incision at two points, and sever the vessels. Raise the sterno-mastoid and omohyoid muscles; select the portion of longus colli bounded by the splenius, omohyoid, and posterior scalene muscles, and carefully dissect through it down to the spine, avoiding all deep vessels and nerves; proceed as advised for the lumbar spine, remembering the possibility of wounding the vertebral artery, especially when much deformity has occurred from absorption of bone.

Operations on the Dorsal Spine.—Make a sufficiently extensive longitudinal incision slightly to one side of the spinous processes; proceed as for a laminectomy until the tips of the transverse processes of the diseased vertebra are reached; divide the rib or ribs near their tuberosities; remove the transverse process, using dry dissection as much as possible. By proceeding as directed the structures liable to be injured can be most certainly avoided,—viz., the intercostal arteries, the spinal nerves, the sympathetic ganglia, and the pleura. The treatment of the diseased area after exposure must depend upon the conditions found, and has already been sufficiently indicated.

Vertebral Puncture.—Rigid asepsis being observed, enter the point of a small trocar slightly to one side of the median line at the level of the fifth lumbar intervertebral space, although the fourth or third may be punctured. Direct the instrument towards the median line so as to reach the subdural space at this point. Not more than fifty cubic centimetres should be removed in children, and even as little as two cubic centimetres has proved serviceable. The puncture after withdrawal of the trocar must be sealed with a shred of aseptic cotton and collodion. Other regions of the spine have been attacked, and the point of puncture must be at the tip of the spinous process in adults, but for children the directions given seem the best. Wiring of spinous processes after forcible straightening of antero-posterior curvatures of the spinal column resulting from caries is described in the chapter on Orthopædic Surgery.

CONVULSIONS IN INFANCY AND CHILDHOOD.

By LEO NEWMARK, M.D.

Etiology.—Convulsions may occur in infancy and childhood under the same conditions as in later periods of life, denoting, for instance, the existence of so-called idiopathic epilepsy or the presence of structural lesions in the central nervous system. They arise, furthermore, in very young individuals far more readily than in older ones, as a consequence of disturbances which the latter would tolerate without such manifestations of nervous disorder.

The term *eclampsia,* when applied to children, should be reserved for those convulsive seizures which neither depend on anatomical disease of the nervous central organs nor recur in an indefinite series like the fits of chronic epilepsy, but are due primarily to causes not residing in the nervous centres, and disappear with the removal of the cause.

Some of the conditions under which convulsions befall children will receive only collateral consideration in this article, and further details must be sought under the appropriate headings.

1. *Organic lesions of the brain,* such as tumor, meningitis, abscess, and vascular lesions, either traumatic or non-traumatic, are sources of convulsions. Convulsive seizures often accompany the onset of infantile cerebral palsies, and they announce, as a rule, the occurrence of meningeal hemorrhage when they immediately follow birth. The fits which usher in the later cerebral palsies of children are very apt to recur as a chronic affliction, while those due to meningeal hemorrhage resulting from injury during labor are much more likely to disappear, spastic paralysis, however, remaining as an effect of the injury.

2. The convulsions may be a manifestation of *idiopathic epilepsy,* which not uncommonly originates in childhood. According to Gowers,[1] one-eighth of all cases commence during the first three years of life. Starr[2] states that out of one hundred and forty-nine cases in which the age of onset was known, the epileptic attacks had begun in twenty-nine before the

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[1] Manual of Diseases of the Nervous System, vol. ii. p. 677.

[2] Familiar Forms of Nervous Disease, p. 256.

age of five years, and in twenty-two between the ages of five and ten. All cases of epilepsy require careful scrutiny as to their idiopathic nature, especially those arising in early childhood, when organic cerebral affections are common. Nevertheless, while it is true that the resulting palsy or other defect may sometimes be so slight as to escape detection, the statement[1] that so-called true epilepsy is not a neurosis, but a congenital or acquired organic disease, is not yet warranted by facts.

3. *Eclampsia* comprising infantile convulsions arising under conditions other than those included in the foregoing categories is more common than the latter. Walton and Carter[2] found that out of one thousand children taken consecutively at random from all classes of society, one hundred and eleven had a history of infantile convulsions, while the number of epileptics in a community is not much in excess of six in one thousand.

In regard to the frequency of these convulsions in relation to age, the statistics of Kassowitz[3] are instructive: out of one hundred children affected with these seizures, sixty-five were in their first year, twenty-two in their second, eight in their third, three in their fourth, and two in their fifth year. The diminution of the convulsive tendency as age advances is also evident from a further analysis of the figures: of the sixty-five children, thirty-nine were in the first and twenty-six in the second half-year of life, and twenty-three of the thirty-nine infants were less and sixteen more than three months old.

The special liability to convulsions in early life is probably determined by the immaturity of the infantile brain. At the time of birth extensive strands of fibres, particularly the pyramidal tracts, are still destitute of the myelin sheath, and such naked fibres, according to the theory, do not readily conduct inhibitory impulses from higher centres to control reflex action at lower levels, where development is further advanced. Soltmann[4] showed experimentally that the motor centres in the cortex of the new-born animal are inexcitable. But even after structural completion of the nervous system, it must be assumed that more time is required to attain functional stability, in order to explain the persistence of the convulsive tendency into the second and third years and even later.

The physiological irritability of the child's nervous system may be exalted by an *inherited neurotic disposition*, which is evinced sometimes by the occurrence of convulsions in several or even all the children of a family.

Of all acquired constitutional disturbances, *rachitis* is most frequently associated with general convulsions as well as with the localized variety known as laryngismus stridulus. Notwithstanding the frequency of this association, the etiological connection has been disputed. Loos,[1] following Escherich, considers laryngismus a symptom of infantile tetany; Rehn[2] maintains that eclampsia, laryngismus, and tetany are alike directly attributable to improper feeding, originating and disappearing independently of the rachitic condition. But other recent writers[3] on the subject abide by the view that there is a causal relation between rickets and the spasmodic phenomena. According to Kassowitz and Jacobi, cranial rachitis, by producing hyperæmia of the skull and of the subjacent meninges and cortex, renders the centres morbidly irritable and therefore liable to discharge on slight provocation. The irritation of the centres for adduction of the vocal cords and other parts of the mechanism of respiration results in those spasms of the respiratory muscles designated as laryngismus; diffusion of the irritation leads to a general convulsion. Indeed, attacks of laryngismus often expand into general convulsions; Henoch[4] observed their coincidence in forty-six out of sixty-one cases, while in only fifteen the laryngeal spasm occurred alone.

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[1] Freud, Zur Kenntniss der cerebralen Diplegien des Kindesalters, 1893, S. 165.

[2] Boston Medical and Surgical Journal, November 5, 1891.

[3] Vorlesungen über Kinderkrankheiten im Alter der Zahnung, 1892, S. 155.

[4] Article on "Eclampsia Infantum" in Eulenburg's Real-Encyclopädie, 3d ed., 1895.

The influence of *dentition* in the causation of infantile convulsions has been, and still is, the subject of much dispute, and this controversy well illustrates the statement that "experience is fallacious and judgment difficult." For ages a disturbance in the process of dentition was considered responsible for many of the diseases to which infants are liable, and barbarous measures were employed to obviate or palliate its supposed consequences. Thus, the celebrated Marshall Hall declared in 1844 that he would rather lance the gums a hundred times unnecessarily than permit the occurrence of a single convulsion by omitting this operation, and he advised that the gums be lanced daily and in urgent cases twice a day when the child is feverish and menaced by a convulsion during dentition. As long as a century ago Wichmann had opposed views, not yet universally abandoned, of the phenomena accompanying dentition and of the dangers to which children are thereby exposed. In the course of time dentition appeared less potent as a pathogenic agent in general and as the exciting cause of convulsions in particular; and Kassowitz,[5] after a prolonged and laborious investigation of the subject, recently expressed the conclusion that convulsions in teething children arise from the same causes as those which occur in children who are not cutting teeth. According to this writer, the advance of the tooth and its protrusion through the gum cause neither local irritation nor fever nor any reflex symptoms due to implication of the dental nerves, all such disturbances being purely adventitious and readily accounted for by processes other than dentition. This negative view is not universally accepted, but it is now conceded that cases of convulsions excited solely by the eruption of teeth are at any rate rare, and that this causal connection is difficult to prove.

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[1] Deutsche medicinische Wochenschrift, 1896, S. 183 of the society supplement. See also Abercrombie, Archives of Pediatrics, November, 1896.

[2] Berliner klinische Wochenschrift, 1896, S. 787.

[3] Kassowitz, loc. cit., p. 177. A. Jacobi, Archives of Pediatrics, November, 1896 Hauser, Berliner klinische Wochenschrift, 1896, S. 782.

[4] Vorlesungen über Kinderkrankheiten, 3d ed , p. 171.

[5] Loc. cit.

To *gastro-intestinal* disorders as much as eighty per cent. of the convulsions in children have been ascribed.[1] In some cases auto-infection from the alimentary canal may be assumed in order to explain the fit as toxic, but the fit is generally of reflex origin, provoked by the presence of food which is indigestible either on account of its quality or quantity. Rehn, in a paper already mentioned, published observations to show that a change to proper nourishment may almost immediately produce a cessation of laryngismus and eclampsia in rachitic children, even though the rachitis persist. Invagination, rectal polypi, and hernia[2] have been known to excite general convulsions. Intestinal parasites are credited with the power of causing violent eclampsia, but helminthic eclampsia has encountered incredulity as well as dental eclampsia. Cases have been reported on good authority, but the rarity of this etiology may be inferred from the statement of so experienced a pediatrist as Henoch[3] that he had never seen a case of eclampsia which he could ascribe with certainty to irritation by intestinal worms.

The mucous membrane of the genito-urinary tract and of the nasal cavity has occasionally been the starting-point of reflex action culminating in convulsions. Affections of the common integument, in consequence of burns, for instance, and even the slight trauma of piercing the ear[4] have been reported to have had that result.

Acute febrile diseases in children may be ushered in by an epileptiform seizure occasionally in such a manner as to divert attention from the primary trouble. Rigors are rare in children, but Baldwin[5] concludes from a study of one hundred and thirty cases of acute febrile disturbances in children, the majority of which might have been accompanied by rigors in adults, that convulsions very rarely take their place, even in infants. In five out of the one hundred and thirty cases there were rigors, and in an equal number convulsions. While it is, therefore, true that in severe and acute febrile diseases initial convulsions very often do not occur, yet they may be observed in almost any affection in children attended by an abrupt rise in temperature. Their occurrence seems to depend—besides the suddenness of the elevation in temperature—on the nature of the pathogenic agent, and sometimes also on the special irritability of the patient's nervous centres.

The onset of *pneumonia* is relatively often accompanied by epileptiform seizures. Coutts[1] noted their occurrence in about five per cent. of Eustace Smith's cases; but Osler[2] states that they usher in the disease in at least one-half the cases. Apex pneumonia is considered by some as particularly likely to be associated with convulsions, but this relation has been denied by others.

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[1] Morselli, abstract in Neurologisches Centralblatt, 1896, S. 892.

[2] Feurer, Correspondenz-blatt für Schweizer Aerzte, 1893.

[3] Loc. cit., p. 162.

[4] Hennig, quoted by Soltmann.

[5] Lancet, June 13, 1896.

Acute *pleurisy* begins with a convulsive fit far less frequently than does pneumonia.

Kassowitz[3] saw *variola* set in with convulsions in a child two months old. Hunter,[4] Hilton Fagge,[5] and B. Sachs[6] observed this occurrence in *chicken-pox,* and *vaccine fever* has also been known to produce such seizures. They may precede the eruption in *scarlatina* and *measles*. Epileptiform attacks may be produced by *malaria* in infants, in place of the rigor, and they may recur with every paroxysm of the disease, as observed by Dubrisan[7] in a child of seventeen months on eleven consecutive days. *Whooping-cough* may be complicated by eclampsia which often begins with laryngismus. The convulsions which occasionally attend the onset of *acute poliomyelitis* are probably of the same nature as those met with in other acute febrile diseases. In rare instances such comparatively mild disorders as *tonsillitis* and *parotitis* have been introduced with a single or repeated convulsive fits.

Toxæmic Convulsions.—Nephritis occurring as a complication of the acute infectious fevers is a source of *uræmic convulsions*, especially in the course of scarlatina. Eclampsia in the mother has been followed by eclampsia in the new-born child, and the fits of the infant are regarded as due to the cause which originated the disorder of the parent. In one such case[8] the autopsy on the child revealed nothing to account for the convulsions, and it was assumed that both mother and offspring had been intoxicated by abnormal products of metabolism. In another instance[9] multiple encephalitis was found, and this lesion was also attributed to the toxic influence of the mother's blood. *Alcohol* may produce epileptiform fits, either when imbibed by the child itself or when it affects the milk of the mother or nurse.[10] *Lead* has a strong tendency to affect the brain either directly or, more probably,[11] indirectly, through its deleterious effect on the kidneys, and thus excites convulsions. Tuczek[12] observed epileptiform attacks in a child from the therapeutic use of antipyrin, and the same effect has been reported from the administration of other drugs.

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[1] Quoted by Morris J. Lewis in vol. iv. p. 877 of this Cyclopædia.

[2] Principles and Practice of Medicine, p. 521.

[3] Loc. cit., p. 156.

[4] Quoted by Soltmann.

[5] Principles and Practice of Medicine, 2d ed., vol. i. p. 245.

[6] Nervous Diseases of Children, p. 61.

[7] Soltmann's article.

[8] Woyer, Centralblatt für Gynäkologie, 1895, No. 13.

[9] Wilke, Centralblatt für Gynäkologie, 1893, No. 17.

[10] Toulouse Gazette des hôpitaux, 1891, No. 98.

[11] Oliver, Lead Poisoning, 1891, p. 112.

[12] Berliner klinische Wochenschrift, 1889, No. 17.

Observations have been recorded by Soltmann and others which indicate that the emotions of fear or anger aroused in mother or nurse may evoke convulsions in the infant, probably by altering in some way the composition of the milk. The effect of fright experienced by the child itself is illustrated by the case mentioned by Henoch, of an infant who was greatly startled by the cry of its mother, whom it had bitten in the nipple while being suckled, and was immediately seized with convulsions.

Symptomatology.—Besides general convulsions, spasms confined to the respiratory apparatus are observed in infancy, chiefly between the ages of six months and two years. This form of local spasm is termed *laryngismus stridulus*, or *spasm of the glottis;* but the term is too narrow, as in the majority of cases more of the mechanism of respiration than the adductors of the vocal cords is involved. The characteristic feature of the attack is the arrest of respiration, a brief apnœa, followed by a long, crowing inspiration. According to Kassowitz, the inspiratory spasm with closure of the glottis is less common than a rapid succession of expirations terminating in expiratory apnœa; the two forms may occur in combination or independently. In the former case the pause in the breathing after expiration is ended, not by the child's freely taking a deep breath, but by its inspiration through the almost completely closed glottis, with the crowing sound. In any case, if the attack is at all severe, the child becomes livid, retracts its head and may become rigid, its arms and legs being extended and the fingers and toes flexed; in a few seconds breathing is resumed, but occasionally the apnœa is prolonged until death results. The seizures vary greatly as regards frequency; there may be long intervals between them, or they may recur as often as twenty or thirty times daily, being provoked in irritable rachitic infants whenever they cry or even when they attempt to nurse. The tendency to the paroxysms persists for several months. In Europe, where laryngismus is very common, at least in large cities, observers note that the attacks are most frequent from January to April.

The *general convulsions* are not distinguishable from ordinary epileptic seizures as far as the appearance presented by the child is concerned. Sometimes, after preliminary manifestations of discomfort due to the exciting cause, consciousness vanishes, the head and eyes may turn to one side, and then the muscles of the trunk and extremities are subjected to tonic contraction. This phase of the fit is followed by clonic contractions of the voluntary muscles, by which the head is drawn back with jerky movements, the features are distorted, and alternate flexion and extension produced in the trunk and extremities. The fists are clinched and the thumbs adducted into the palms. The pupils are dilated. In consequence of the involvement of the respiratory muscles the breathing is at first suspended and is then resumed in an irregular, explosive manner. The complexion becomes livid. Foam issues from the mouth, and in children who have teeth it may be tinged with blood. Urine and fæces may be discharged during the fit. Gradually the movements become less frequent and finally cease, the child remaining in a condition of stupor which may pass into sound, normal sleep, or may be interrupted by a renewal of the convulsions. The initial convulsions of the acute febrile diseases may occur in a single attack or be repeated several times, the patient continuing comatose during the intermissions. The seizures caused by indigestion and others of similar reflex origin sometimes recur for hours with intervals of coma, and then subside after removal of the exciting cause. In rachitic children the general convulsions may be preceded by laryngismus; they have been observed as often as twenty or thirty times daily and hundreds of times in the course of a few weeks in the same child.

Unilateral convulsions and still more limited seizures are almost always due to organic disease of the brain; but in rare cases localized spasms of an eclamptic nature have been observed without organic lesions of the nervous centres; for instance, in uræmia following scarlet fever.[1]

Diagnosis.—It is difficult to determine the nature of a general convulsive attack without taking into consideration the concomitant symptoms and the course of events, for the mere aspect of the individual seizure at its height does not afford a criterion by which eclamptic may be distinguished from idiopathic epileptic fits or from those of organic origin. When the first convulsive manifestations occur in a numerous series protracted throughout the day or longer, with intermissions of stupor, there is considerable probability that they are due to some definite disturbance. If there is high fever the fit may herald the onset of an acute infectious disease; but an increase of temperature may accompany other disorders causing convulsions; for example, indigestion. Indeed, Bridge[2] finds that high fever is present whenever the convulsive movements are prolonged beyond a few minutes, and he considers the fever as marking the distinction between eclampsia and true epilepsy. In the absence of fever, the sources of reflex or toxic irritation enumerated in the foregoing will be sought for. The occurrence of laryngismus indicates the rachitic origin, but to the predisposing influence of rachitis there is not uncommonly added the action of a special exciting cause. Toxæmic convulsions occurring in the course of febrile and other diseases of children are, as a rule, easily interpreted. If the fits are caused by organic brain disease, other symptoms of the morbid process will appear.

A fit apparently or actually excited by some accidental, eccentric cause may prove to be the forerunner of habitually recurrent epilepsy. Thus, it is credibly asserted in the histories of some confirmed epileptics that the first seizure was provoked by a derangement of digestion or by a severe mental shock. Hughlings Jackson mentions a case in which a convulsive fit at the onset of scarlet fever was the first indication of epilepsy.[1]

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[1] Dunin, Berliner klinische Wochenschrift, 1889, No. 7.

[2] American Journal of the Medical Sciences, March, 1893.

Hysterical convulsions may simulate epilepsy and eclampsia in children as well as in adults. They may recur in more or less rapid succession for hours and even days, as may the eclamptic; but this great frequency is not necessarily followed by prostration, nor is it attended with danger in hysteria, as is the case in eclampsia. The cessation of the attacks at night while of frequent occurrence during the day, the association of characteristic hysterical features with those of an epileptiform type, and often the influence of psychic treatment may also serve to distinguish the hysterical nature of the seizures.[2]

Prognosis.—The prognosis of convulsions must be considered with regard to the child's survival, to immediate sequelæ, and to the repetition of the fits in such a manner as to constitute epilepsy.

Not all children dying in convulsions are to be looked upon as dying of convulsions. The fatal termination of tuberculous meningitis, for instance, with epileptiform seizures would not be attributable to the latter.

In proportion to the recoveries the mortality from infantile convulsions is not great. Nevertheless, in every case of eclampsia the prognosis should be formed with caution. If the child is debilitated, and if numerous attacks occur with great severity, the outlook is grave. The reflex convulsions arising from irritation of the alimentary canal generally disappear after the removal of the irritant. The convulsions ushering in exanthematic diseases hardly menace the life of the patient. Those observed later in their course are more liable to prove fatal, but the prognosis in individual cases must be determined by the conditions presented by the underlying affection. Eclamptic attacks in whooping-cough are of very grave significance. Rilliet[3] lost four-fifths of his cases with this complication.

The majority of children with laryngismus stridulus survive the attacks, but deaths are sufficiently common to justify a guarded prognosis. According to Kassowitz, the expiratory apnœa is the more dangerous form of the attack.

In some cases aphasia, palsy, or mental defect, denoting a structural lesion of the brain, is observed after one or a series of convulsive fits, and it is a moot-point whether these lesions and the convulsions are coördinate effects of a primary cause or whether the convulsions caused the lesions. The former relation is probably the more frequent, but there is anatomical and clinical evidence in proof of the latter also.[4]

A prognostic question of very great importance is that which regards the subsequent development of epilepsy in those who have had infantile convulsions. The vast majority escape epilepsy afterwards. Walton and Carter[1] calculated that "the chances of a given child with infantile convulsions becoming an epileptic later in life" are "something like one in two hundred and twenty." When we consider, however, that infantile convulsions are mentioned in the antecedents of a by no means insignificant proportion of epileptics, we must admit some relation between the early and the later fits. Walton and Carter found that out of seventy epileptics only five had had convulsions in infancy followed by a period of immunity before they became continuously epileptic, but M. Allen Starr noted infantile convulsions in the histories of nearly forty per cent. of over one thousand cases of epilepsy.[2] The relation between the convulsions of infancy and epilepsy beginning at a later period in the same individual is probably that they are both manifestations of a congenital neurotic irritability. Such a congenital tendency to convulsions is evident in families in which several children have had fits in infancy without subsequently becoming epileptic, while some other child of the same family develops epilepsy without having had infantile convulsions. Instead of epilepsy, other neuroses may appear in the after-life of the subjects of early convulsive seizures.[3]

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[1] Fagge, loc. cit., p. 805.

[2] Fürstner, Archiv für Psychiatrie, 1896, Bd. xxviii. S. 494.

[3] Soltmann, loc. cit.

[4] Sachs, Nervous Diseases of Children, p. 530. Freud, Zur Kenntniss der cerebralen Diplegien des Kindesalters, S. 60.

Treatment.—When a child is seized with convulsions it is often necessary to proceed to the suppression of the fit before its etiology can be ascertained. This is generally readily accomplished by the administration of chloroform. A few whiffs may suffice for the purpose; but this treatment does not prevent the recurrence of the attacks. It must, therefore, in severe cases be repeatedly employed. Henoch strongly recommends the use of chloroform above all other means, but he found it occasionally ineffectual. Hydrate of chloral is useful, and should be given by enema. These drugs ordinarily serve the purpose of, at least temporarily, tranquillizing the convulsed child. A number of others, such as opium or morphine or nitrite of amyl, have been suggested in protracted and rapidly recurrent seizures. Bromides are of benefit only when given to combat the convulsive tendency after a fit.

The child is usually placed in a bath before the arrival of medical aid. A hot bath is likely to be injurious, and a warm one of doubtful utility unless the child has fever and the temperature of the bath is sufficiently low to reduce it. Cool affusions while the patient is in the bath increase its efficacy.

If a digestive trouble is recognized as the cause of the fit, an emetic should be administered, if possible, and also an enema. Other sources of irritation should be dealt with in an appropriate manner. From what has been said concerning the relation of dentition to convulsions, the lancing of the gums will rarely appear advisable.

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[1] Boston Medical and Surgical Journal, November 5, 1891.

[2] Personal communication.

[3] Coutts, quoted by Lewis, loc. cit., p. 888.

Attacks of laryngismus stridulus are treated by dashing cold water into the infant's face, or by some other method of peripheral irritation likely to produce an inhibitory effect. Kurt[1] obtained a curative as well as an inhibitory effect by regularly applying to the nasal mucous membrane a mixture of quinine and sugar at the onset of an attack and also in the intervals. Osler[2] recommends Ringer's method in severe cases,—that is, placing the child two or three times daily in a warm bath and then thoroughly sponging the back and chest for a minute or two with cold water. If the attacks recur so frequently as to endanger the child's life, Henoch counsels the use of morphine.

In cases of general convulsions as well as of laryngismus stridulus it is very important to treat the underlying predisposing condition. Rachitis especially requires careful attention. Mention has already been made of Rehn's experience of the striking effect produced by a change in the nourishment of rachitic children who had not been receiving adequate breast-milk. Kassowitz[3] considers phosphorus in small doses an almost infallible remedy for laryngismus stridulus. Senator also finds it beneficial in this affection, but Heubner[4] employed it without success.

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[1] Neurologisches Centralblatt, 1890, S. 446.

[2] Practice of Medicine, p. 486.

[3] Loc. cit., p. 176.

[4] Berliner klinische Wochenschrift, 1896, No. 31.

TETANUS.

By F. X. DERCUM, M.D.

THE article on Tetanus, vol. iv. p. 913 of this Cyclopædia, which was written about nine years ago, is still in many respects a statement of our present knowledge of the disease. It will then be the object of the present communication to deal only with the advances in knowledge that have accumulated since the original paper was written. Reference is made to the systematic consideration of the subject there presented. Tetanus as a whole, it should be added, is a much rarer disease than formerly. This is doubtless due to the general introduction of antiseptic measures in the treatment of even trivial wounds.

Tetanus is now definitely known to be an infectious disease. The tetanus bacillus has not only been isolated, but its life-history and the conditions necessary for infection have been largely worked out. Further, tetanus, whether making its appearance as traumatic tetanus, as tetanus neonatorum, as puerperal tetanus, or as the so-called idiopathic or rheumatic tetanus, is now considered to be one and the same disease. In traumatic tetanus, in tetanus neonatorum, and in puerperal tetanus the avenues of infection are quite evident. The so-called idiopathic and rheumatic cases consist, in all probability, of instances in which a trauma so small as to be readily overlooked is present, or in which the trauma has occurred in some concealed situation.

A more intimate knowledge of the life-history of the bacillus and of the conditions governing infection may yield an explanation of the interesting fact of the great prevalence of tetanus in hot countries and its comparative rarity in colder climes. The Calcutta Hospital reports two hundred and eighty cases in ten years, and the Pennsylvania Hospital thirty-six cases in seventeen years.

Peyraud[1] confirmed the relation existing between hay-dust and tetanus. As a matter of fact, the tetanus bacillus has an exceedingly wide distribution. It is found not only in soil, hay, straw, and manure, but on walls in rooms, in hospitals, and in other diverse situations. Schwartz[2] and Williams, of New York, have both demonstrated the occasional presence of the germs in the atmosphere.

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[1] La Semaine Médicale, 1890, p. 273.

[2] Centralblatt für Pathologie, 1892, Bd. ix. S. 697.

I have already, in my former article, mentioned the tetanus germ as a pin-headed, bristle-shaped bacillus. This description applies, however, only to the stage of sporulation. In this stage it is without motility. When not sporulating the germ consists of a slender thread of variable length with rounded ends, and has some power of movement. It is a significant fact that the tetanus germ grows more actively at the temperature of or about that of the human body,—namely, from 97° to 100° F. It grows less and less readily as the temperature of the culture falls, and finally ceases growing at 14° C. (57.2° F.). The germ is strictly anaerobic. It will not grow in the presence of oxygen. It has extraordinary vitality, and is unusually resistant to ordinary antiseptic agents.

The tetanus germ also survives relatively high temperatures; for instance, it is not destroyed by long-continued exposure to 80° C. A five minutes' exposure to the temperature of boiling water destroys it. It resists the action of five per cent. carbolic acid solution for ten hours, but succumbs when exposed to such a solution for fifteen hours. The same solution, together with 0.5 per cent. of hydrochloric acid, renders it inactive after two hours. It is killed when acted upon for three hours by corrosive sublimate 1 to 1000, and in thirty minutes by the same solution to which five per cent. hydrochloric acid has been added. Kitasato[1] showed that the filtrate of the cultures of tetanus bacilli will, when injected into animals, produce tetanus, but regarding the nature of the poison we are still in doubt. Brieger[2] obtained a number of poisonous ptomaines, the principal one of which he named tetanin. The others, which appeared to be allied in character to tetanin, he named tetanotoxin, spasmotoxin, and spasmodin. Later Brieger and Fränkel[3] obtained another substance, a toxalbumin, which also produces tetanic symptoms when injected into animals. Still later Brieger, together with Cohn and Boer, obtained what appears to be the actual tetanus poison, but which did not prove to be an albuminous body. Sydney Martin[4] believes that the poison is a proteolytic ferment developed from the tetanus bacillus, and that the albumoses also found in the bodies of patients dying from tetanus are the results of the activity of the poison. He holds, from experiments made on guinea-pigs, that the muscular spasms do not develop until such albumoses are formed. According to Nissin,[5] the poison is contained in the blood; according to Kitasato,[6] it is contained in the serum of the pleura and pericardium; according to Bruschettini,[7] it is found in urine; and according to Brunner, it is found in the saliva.

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[1] Zeitschrift für Hygiene, 1891, Bd. x. S. 267.

[2] Deutsche medicinische Wochenschrift, 1887.

[3] Berliner klinische Wochenschrift, 1890, S. 241.

[4] Report of the Medical Officers of the Local Government Board, London, 1894, 1895.

[5] Deutsche medicinische Wochenschrift, 1891, S. 775.

[6] Zeitschrift für Hygiene, Bd. x.

[7] Deutsche medicinische Wochenschrift, 1892, S. 349.

It is probable also that the blood-vessels and lymphatics are not the only channels by which the poison is conveyed. Brunner, for instance, advances the view that the nerve-fibres also carry the poison and carry it directly to the cord, and his view is supported by the fact that in experimental tetanus the part of the spinal cord nearest to the seat of the injection was always capable of producing tetanus in other animals, while other portions of the cord were not.[1]

Certain important factors relative to the infection of tetanus remain to be considered. It appears from the researches of Vaillard, Vincent, Rouget, and Klipstein that *pure* cultures of tetanus, provided the germs had sporulated and the poison had been destroyed by heat, could be injected into animals without producing tetanus. It seems as though some trauma of the tissues—for instance, a bruise, or a mixed infection—is necessary. In the experiments of the authors quoted the animals developed tetanus just as soon as the wounds became infected with extraneous germs. This fact is evidently of very great practical importance, inasmuch as the ordinary saprophytic germs are killed by modern methods of antisepsis, and while these methods do not kill the tetanus bacillus, they at least kill the germs which assist or favor tetanic infection. The degree of the bruising of the tissue is in this connection of extreme importance. It would appear that anything which lowers the vitality of the tissues and prevents the proper action of the phagocytes favors the infection.

Infection without inoculation probably does not occur. As already stated in my previous article, the so-called idiopathic and rheumatic cases of tetanus are probably to be explained by the location of the infecting wound,—that is, that the latter occurred in some concealed situation, or, as is not infrequently the case, that the original wound has healed and been forgotten by the patient by the time the symptoms of tetanus make their appearance. Guelpa's[2] suggestion that idiopathic tetanus may be due to infection from the intestinal tract appears to be negatived by the observation of Fermi and Pernassi[3] that the tetanus toxins are quickly destroyed by the intestinal mucous membrane.

Inasmuch as tetanus is beyond all doubt an infectious disease, it is not surprising that instances of contagion from man to man occasionally occur. Indeed, a considerable number of such cases are on record.

The unquestionable success which has attended the use of antitoxin in diphtheria, as well as the increasing number of cases of tetanus reported in the literature as having been treated successfully by antitoxin, estimated by Lambert at 29.57 per cent., make it necessary to give this method of treatment extended consideration.

In 1890 Kitasato attempted to make animals immune to tetanus. His attempts by means of repeated inoculations of attenuated virus failed to yield a satisfactory result. However, by applying the method which Behring had devised in experiments with diphtheria, he finally succeeded. He first injected a middle-sized rabbit with 0.3 cubic centimetre of the filtrate of tenanus culture subcutaneously. Immediately afterwards he injected into the same area three cubic centimetres of a one per cent. solution of iodine trichloride. In twenty-four hours the same dose of iodine trichloride was again administered. In forty-eight hours, some symptoms of tetanus infection having appeared, the dose of iodine trichloride was again repeated, and twice thereafter; in all, five doses were administered before the tetanic symptoms finally disappeared. The animal was entirely well in ten days. On the fourteenth, eighteenth, and twenty-fifth days large doses (from two to three cubic centimetres) of tetanus culture were administered with negative results. Two control animals which received but a moderate dose of the culture died in a few days. This method was successful in producing immunity in forty per cent. of the animals experimented upon, and this immunity persisted for two months. Experiments were further made to render other animals immune with the blood of immunized rabbits. Kitasato injected liquid blood, before coagulation, into the abdominal cavity of mice, which, with control animals, were subsequently inoculated with tetanus bacilli, and in such a dose that the control animals died in thirty-six hours; the others remained immune. The serum also revealed therapeutic properties. An animal could be infected with tetanus, and, after symptoms had appeared, successful results were obtained by injecting the serum into the abdominal cavity. Kitasato further proved that when the filtrate of tetanus culture is mixed with the serum of the tetanus-immune rabbit it fails to kill the mice, while control animals perish. Behring experimented extensively in the same field, and found that iodine trichloride added to his carbolized tetanus culture in increasing doses made the culture less and less virulent, and finally innocuous. Rabbits which survived injections of this mixture were able to stand more than the minimal dose necessary to kill control animals, and thus attained a certain degree of immunity which, through further inoculation with the culture-fluid, became greater and greater. Behring and Schuetz later demonstrated the possibility of thus immunizing horses and sheep.

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[1] Bruschettini, Centralblatt für Bakteriologie, 1891, S. 15.

[2] Journal de Médecine de Paris, January 13 and 20, 1889.

[3] Zeitschrift für Hygiene, Bd. xvi.

More interesting than all, however, are the combined researches of Brieger, Kitasato, and Wassermann. To begin with, they noted that when they attempted to grow the tetanus bacillus in a watery infusion of thymus gland they were only occasionally successful. They then made the remarkable observation that the tetanus bacillus, though it grew in thymus infusion, failed utterly to develop spores. Even when they submitted the cultures to the incubator for as long a period as fourteen days the result did not vary. However, when these sporeless bacilli were transferred to other culture-media they again developed spores,—that is, the tetanus bacillus simply loses the power of spore-bearing while in the thymus infusion. These investigators then followed up this discovery by inoculating animals with cultures made in thymus infusion, with the result that they were able to produce immunity in all of the animals experimented upon. While the trichloride method yielded forty per cent. of immunized animals with the immunity persisting for two months, the thymus method yielded immunity in one hundred per cent., and at the end of four months this immunity was as marked as ever.

Behring, Frank, Kitasato, and Tizzoni and Cattani were successful in conferring immunity upon other animals by the injection of immunized serum. They all demonstrated also that this serum possessed in addition curative properties,—that is, animals in which tetanus had been produced by inoculation, and which were afterwards treated by injections of immunized serum, recovered, while control animals perished. Further, they all found that the dose required to effect a cure was far greater than that necessary to bring about immunity.

Inasmuch as the quantity of serum which could be derived from mice and rabbits was obviously too small to be available in man, Behring experimented upon horses and sheep. Even under these circumstances it seemed at first as though the quantity of serum required would be too large to be practicable. However, it was found that the serum of the immunized animals increased in strength with time, and Behring finally succeeded in immunizing the serum of horses until it attained an immunizing value of 1 to 1,000,000 body-weight. Of this serum one gramme will be sufficient to immunize one thousand animals of one thousand grammes body-weight each against a minimal fatal dose of tetanus culture.

According to Behring and Frank, and also Tizzoni and Cattani, the healing value of the serum is from one thousand to two thousand times less than its immunizing value. Therefore a serum which has an immunizing value of 1 to 1,000,000 would have a curative value of only 1 to 1000,—that is, for every kilogramme of body-weight one gramme of serum should be used. Again, in cases in which the disease has already advanced, or in which the infection has already produced severe symptoms, a still larger quantity would have to be employed.

Of late it has been possible to raise the immunizing equivalence of the serum of the horse to a much higher degree. Thus the New York Health Department[1] has furnished serum of the strength of 1 to 400,000,000. "The tetanus antitoxin is prepared in the same manner as the diphtheria antitoxin, by inoculating the tetanus toxin in increasing doses into horses. The toxin is grown in bouillon under hydrogen, and after ten or fifteen days filtered through porcelain, and the germ-free filtrate is used for the inoculations. The horses receive half a cubic centimetre as the initial dose of toxin, and this dose is increased as rapidly as the horses can stand it, until they support seven to eight hundred cubic centimetres or more at a single dose. After some months of this treatment the blood of the horse contains the antitoxin in sufficient amount for therapeutic use. When the animals' temperatures are normal and they have recovered from the dose of toxin last given, they are bled into sterile flasks and the serum collected. The serum contains the antitoxin, and is tested on white mice or guinea-pigs." A serum of 1 to 400,000,000 is one "which will protect 400,000,000 grammes of white mice against a three-to-four-day fatal dose of tetanus toxin. Reckoning in antitoxic units, twenty cubic centimetres of serum will contain eight thousand antitoxin units, an antitoxin unit in tetanus being the amount of serum necessary to protect one million grammes of test-animal.

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[1] Lambert, loc. cit.

Administration.—"The serum is supplied in twenty-cubic-centimetre bottles, and should be injected in from ten- to twenty-cubic-centimetre doses. In severe cases the patient should receive fifty cubic centimetres in the first twenty-four or thirty-six hours, and these ten- to twenty-cubic-centimetre doses repeated once or twice each day during the following four or five days or longer, according to the course of the disease. The circular accompanying the bottles explains fully the proper method of procedure."

It is extremely probable that the antitoxin does not directly destroy the tetanus poison. It appears to prevent the destructive action of the toxin on the nerve-cells. In all probability it immunizes those nerve-cells which have not yet been destroyed or seriously damaged by the toxin. It can be readily seen that it is of the utmost importance to use the antitoxin at the earliest possible moment and in large amount. As stated above, it has been shown that the curative value of the antitoxin is from one thousand to two thousand times less than its immunizing value, and it can therefore be readily understood why the dose should be large. It is of practical importance to remember that the employment of the serum has not been found to be in any way injurious. Thus far no contra-indications to its use have been discovered.

Treatment of the Wound.—We are always confronted by the knotty problem presented by the wound. Every possible method of dealing with it seems to have been practised, with, in the vast majority of cases, a negative result. Let us analyze the condition of the wound in the light of recent developments and see what indications present themselves. Guelpa concludes that "during the first manifestations of tetanus, at least, the multiplication of the microbe is limited to the site of the infection," and that "it is only later and quite rarely that the bacillus becomes generally diffused through the organism." He also points out that the bacillus is always to be found in the deeper portions of the wound; and this fact seems to have an especial significance. It certainly suggests that from the superficial portions of the wound the bacillus is soon expelled by the defensive process of discharge and suppuration, while in the deeper portions of the wound, ready escape not being possible, the bacillus is retained sufficiently long to mature and secrete its ptomaines. The very conditions demanded by this view are presented in the kinds of wounds most frequently found in tetanus. The punctured wound permits the lodgement of the germ deep within the tissues, with but a narrow, sinuous, and readily obstructed outlet. The lacerated wound and the deep burn expose intermuscular septa, open up the sheaths of nerves, tendons, and joints, and permit of the establishment of numerous and tortuous tracts of infection. Lastly, successful inoculation in animals is made not by rubbing the virus upon an abraded surface, but by introducing it beneath the skin, into the sheaths of the nerves, and beneath the dura of the brain.

If these inferences be correct, the first indication for treatment is very clear. Upon the very earliest signs of tetanus the wound should, other things being equal, be freely incised and its innermost recesses exposed. The wound should be curetted, the tissues freely released, and, if necessary, deep excisions of tissue made. These directions, of course, cannot apply to every individual case, but must be adopted according to circumstances. Guelpa calls especial attention to the fact that it is in the thrombi of veins coming from the wound that the principal nidus of the microbe is frequently found. The operation being completed, the wound may be swabbed with Lugol's solution or, better still, with a one per cent. solution of iodide trichloride. A very strong solution of sublimate—from five to ten parts in a thousand (Guelpa)—may also be used. Abundant and frequent irrigation should be thoroughly practised. The surgical interference should, however, be judicious in the extreme, and should be limited to the free opening up of the wound, the excision of ragged edges and sloughs, the removal of clots and thrombi, and thorough washing, the wound being kept open so that the washing can be repeated at intervals. Recoveries have occasionally occurred after nerve-stretching and even amputation, but certainly in the present state of our knowledge nerve-stretching has nothing to recommend it, while amputation is probably rarely justified. It should be remembered that the average case of tetanus comes under medical care after the symptoms are fully developed and when some degree of general infection has taken place. Unless indicated upon general surgical grounds, amputation should not be practised.

Inasmuch as watery infusion of the thymus gland has such a remarkable power in inhibiting the growth and development of the tetanus bacillus, it would be perfectly proper to practise subcutaneous injections of strong thymus infusion whenever the antitoxin cannot be obtained. This remedy was originally suggested by me, and I employed it in one case, but, unfortunately, five days had already elapsed, and the trial was unsuccessful.

The injection of a one per cent. solution of carbolic acid subcutaneously (Bacelli's treatment) is useless and unphilosophical. Carbolic acid exercises little or no influence on the toxin. A five-tenths per cent. solution of carbolic acid is used to preserve the toxin in the laboratory.

As regards the excessive sweating, this does not seem to call for special interference. It is not impossible that the toxin may in part be eliminated in this way. Excretion of the poison by the kidney should also be favored by the free administration of liquids.

TETANY.

BY F. X. DERCUM, M.D.

Definition.—Tetany is a disease, probably toxic in origin, which is characterized by attacks of intermittent tonic cramp of the voluntary muscles, symmetrical in distribution, affecting especially the muscles of the extremities,—most frequently those of the hands and forearms, causing the hands to assume the position of the "writing" or "obstetric hand,"—together with abnormal response of the nerves to mechanical and electrical stimulation. Quite a number of synonyms exist for tetany, among which the following are the most important: tetanilla, intermittent tetanus, and pseudo-tetanus.

Etiology and Pathology.—Tetany is an exceedingly obscure disease. It is rarely met with in this country. In Europe it is in certain cities quite common, and not infrequently occurs in an epidemic form. It is not, however, equally distributed, nor is it always present in the same locality. For instance, frequent mention of tetany is made in the literature of Paris between the years 1830 and 1860. Subsequent to 1860, however, the subject of tetany ceased to recur in the Paris writings, and it is only fair to assume that the disease had become either very rare or had disappeared. Charcot, who had seen some cases in early life, observed none in after-years. In Vienna, on the other hand, where it has been much studied for the last fifteen years, it seems to have been previous to this period very infrequent. Again, Schulze, who had had abundant opportunity to study the disease in Heidelberg, saw in his subsequent experience at Bonn no cases for the first four years, and after this time observed it only very infrequently. The experience of Bruns is also very interesting. Of four thousand cases of nervous disease observed by him in Hamburg during a period of ten years, twelve only were cases of tetany. These occurred in an epidemic, supposedly from gastro-intestinal auto-intoxication.

As regards children it is quite well established that the lower classes and those whose hygienic surroundings are bad, are most frequently attacked by the disease. Tetany is of such varied origin as to suggest that its detailed pathology is not always the same. Thus it occasionally follows infectious fevers, such as measles, scarlet fever, and influenza. At other times it is associated with gastro-intestinal disturbances, indigestion, diarrhœa, dilatation of the stomach, and the presence of worms in the intestine. Tetany also frequently occurs associated with rickets. It is likewise met with in pregnant, puerperal, and nursing women; at times it is attributed to catching cold, to rheumatism, to emotional disturbances, and to physical over-exertion. In exceedingly rare instances it has been seen to follow poisoning with ergot, lead, alcohol, and even the administration of chloroform. Again, one of the most remarkable facts connected with the subject of tetany is that a syndrome which appears to be in every way identical with that of true tetany not infrequently follows total extirpation of the thyroid gland. That this is in reality a form of true tetany observers almost without exception admit. Finally, tetany has in very rare instances been found in association with various nervous diseases, such as Graves's disease, syringomyelia, and brain-tumor. Of seventy-two American cases collected by Griffith, thirteen occurred in children. They were ascribed to such causes as digestive disturbances, dilatation of the stomach, rheumatism, exposure to cold, diphtheria, extirpation of the thyroid gland, and adenoid growths of the pharynx.

In view of the lack of accurate knowledge, it is somewhat difficult to formulate a theory of the disease. The relation, however, which exists between tetany and total extirpation of the thyroid gland is worthy of thoughtful consideration. Without pausing to discuss the physiology of the thyroid gland in detail, it will suffice to point out that from all we know the inference is justified that the thyroid gland secretes a material which is indispensable to the maintenance of normal nutrition and normal nerve-function. How this material acts is, of course, a matter of speculation. Ewald is of the opinion that it is protective to the nervous elements by being antitoxic to the products of tissue metabolism. Whatever the method of its action, there can be no doubt that it is protective, and if protective against the products of ordinary tissue metabolism, why not also against other toxic agents? That toxin of one kind or another plays a *rôle* in tetany seems extremely probable. In what other way can we explain the relation of the infectious fevers or of gastro-intestinal diseases to tetany? Further, the *rôles* played by pregnancy, by the puerperium, and by lactation also assume a new character when viewed from the stand-point of intoxication. The association of tetany with intestinal worms will bear a similar interpretation. Albu has advanced the view that the worms excrete substances which are poisonous to their host, and there is nothing inherently improbable in this explanation.

All of the facts in regard to tetany, it appears to the writer, can be reconciled by supposing that tetany occurs by reason of some impairment of the function of the thyroid gland, so that the latter is no longer able to protect the organism against tetany-producing toxin. It does no violence to the facts to further suppose that this impairment of function is directly related to insufficient and improper food and bad hygiene. Certainly the frequent association of rickets with tetany seems to bear out such a view.

Symptoms.—The disease is ushered in, as a rule, by vague pains, a general sense of uneasiness, and a feeling of weakness and stiffness in the muscles, generally most marked in the arms. The duration of these symptoms, which constitutes a prodromal period, is usually short, sometimes only a few hours. The stage of muscular contraction begins almost always in the upper extremities, especially in the fingers, and thence spreads up the forearm and arm. Later the lower extremities also show signs of rigidity. The muscles of the flexor groups are most affected, thus causing characteristic positions to be assumed. In many cases the spasm is limited to the hands, and no symptom of involvement elsewhere may be noted, save, perhaps, that the patient may complain of vague drawing pains in the legs. The fingers become flexed, generally assuming the position of the writing hand; Trousseau compared the position to that of the obstetric hand. The writing or obstetric hand does not, however, invariably obtain. The hand may be clinched, or the fingers, on the contrary, may be opened out and spread apart. This latter position is, however, extremely rare. In exceptional cases the cramp may be limited to a single digit.

The fingers having assumed the position in the writing hand, the wrist next becomes flexed. Then the forearm may be flexed upon the arm and the arm adducted to the shoulder. If the spasm involves the foot, the toes become flexed, the foot arched, and the legs extended upon the thighs. The attack comes on gradually. It may last a few minutes only or may persist several hours or even days. It may recur on the same, the next day, or after several days. During the interval between the attacks the patient feels comparatively well, save that he may complain of slight pain and stiffness in the muscles. During the height of the spasm the affected muscles become very firm and hard and are somewhat sensitive to pressure. The degree of the spasm varies in different cases. The contraction of the muscles is at times so slight that the patient can overcome it temporarily by his own voluntary efforts. At other times it is so severe as to resist the efforts of the physician to extend the joints during his examination. In addition to the muscles of the extremities, those of mastication may in rare cases be involved, and more rarely still the muscles of expression may be affected. The spasm has also been known to invade the muscles of the trunk and neck. Even the tongue may be involved, and the fact that some patients complain of difficulty in swallowing would suggest involvement of the pharyngeal constrictors. Involvement of the orbicularis palpebrarum has also been met with, and this is likewise true of various muscles of the eyeball. The frequent occurrence of severe spasm of the larynx leaves no doubt as to the involvement of the muscles of this organ. Indeed, laryngo-spasm is a not infrequent and serious accompaniment of tetany.

The involvement of the muscles in tetany is almost always symmetrical. However, cases in which the spasm has been most marked or limited to one side have been reported, though they are very unusual. As a rule, an attack of spasm lasts from a fraction of an hour to two hours, and sometimes to three hours. The duration is very variable. Even in the same case successive attacks may differ greatly in this particular. Cases are occasionally met with in which the attacks last only a few minutes. On the other hand, instances in which the duration is very long—a day or more—have been reported; but they are very rare. Fibrillary twitching is occasionally observed in the contracted muscles, but clonic movements almost never occur. Tremor, however, is quite common. In the intervals of the attacks muscular weakness more or less marked is present. The length of the interval between the attacks is also quite variable, sometimes only a few hours; at other times several days and often weeks intervene.

In addition to the attacks of spasm just described, several other exceedingly important symptoms are, as a rule, present. A remarkable symptom is one that was discovered by Trousseau, and consists in the fact that the spasm may be brought on, if absent, or may be made more pronounced, if present, by pressure upon or constriction of an affected extremity. For instance, if during a passive interval the arm be grasped in such a way that the finger-tips of the physician forcibly compress the nerves of the inner aspect of the arm, the characteristic cramp sooner or later makes its appearance. This symptom can be elicited more readily at certain times than at others. The pressure should be forcible, and should be persisted in for several minutes if necessary. When present, this symptom is pathognomonic of tetany. It is, however, by no means always present, being absent in from about one-fourth to one-third of the cases. When it can be elicited during an interval it is of great value, inasmuch as it at once enables us to decide the nature of the case. It is also of value in demonstrating the existence of latent tetany,—that is, a form of tetany in which there is at no time a clearly marked contracture. It is, perhaps, worthy of mention that the spasm produced by this artificial means sometimes lasts longer than the attacks which come on spontaneously. The explanation of this symptom—the Trousseau symptom, as it is called—is somewhat difficult. It is probable, however, that the spasm of the muscles is excited in a reflex manner by the pressure made upon the great nerve-trunks of the arm. It certainly cannot be explained by interference with the circulation of the arm or by mechanical irritation of the muscles.

Another remarkable symptom has been discovered by Chvostek. This is the extraordinary susceptibility of the nerves in tetany to mechanical impressions. It is an entirely different symptom from that just described, and may be present when the Trousseau symptom is absent. It is best demonstrated in the facial nerve. If we strike the face below the zygoma and in front of the ear a blow, sometimes lightly, sometimes forcibly, with the finger or the percussion hammer, we observe twitching of the angle of the mouth, or it may be of all the muscles of the facial distribution; most frequently the reaction consists merely of movements of the angle of the mouth. It is comparatively rare to have extensive facial response.

Like Trousseau's symptom, Chvostek's symptom is not invariably present. Indeed, it is important to bear in mind that in children it is very frequently absent. At times it may be demonstrated in other nerves than the facial; for instance, in the nerves of the extremities. It is not apt, however, to be as pronounced in these situations as in the face. Exceptions to this rule are said to occur. Unlike Trousseau's symptom, Chvostek's symptom is not pathognomonic of tetany. It is occasionally observed in children who present no evidence of the disease whatever. Chvostek's symptom is further limited to the nerve-trunks. The response to a blow upon the facial nerve may be exceedingly pronounced and yet the muscles remain unchanged,—that is, their mechanical excitability is not increased.

A third important symptom of tetany remains to be described. It consists in a greatly exaggerated electrical excitability of the nerves. This symptom was first thoroughly studied by Erb. Exceedingly weak currents produce muscular contractions out of all proportion to the normal. Cathodal closure tetanus (CaClTe) is induced with remarkable readiness. Anodal closure contraction (AnClC) and anodal opening contraction (AnOC) are both easily obtained. It is also an easy matter to elicit anodal closure tetanus (AnClTe). Cathodal opening contraction (CaOC) is not infrequently observed, and even cathodal opening tetanus (CaOTe) has been met with.

There are no disturbances of cutaneous sensibility in tetany, nor are there any other nervous symptoms except headache, which occurs not infrequently. There is no involvement of the sphincters, except in rare instances, when spasm of the vesical sphincter may be observed. Both in the intervals and during the attacks the mind is, as a rule, quite clear. Mental aberration has, however, in rare instances been observed. For example, Fränkel-Hochwart has reported three cases of confusional insanity occurring in tetany. Kraepelin has also reported several similar instances. Examination of the special senses does not, as a rule, reveal any striking anomaly. Thus, the eye-symptoms are usually unimportant. As already stated, however, spasm of the ocular muscles has been reported. This is true also of dilatation of the pupil; even loss of light reaction has been met with. Nystagmus likewise has been observed. The eye-ground in tetany is normal, though in rare instances hyperæmia has been noted. No anomalies are revealed by the ear or by the senses of taste and smell.

Pains variable in degree are present. They are referred by the patient to the muscles, especially those affected by the contractures. They may be exceedingly slight, and may consist of mere sensations of tension and pulling. On the other hand, they are exceptionally so severe as to demand relief by morphine. In some cases the pains are so widely distributed as to be quite pronounced in muscles which are not involved in the spasm. Frequently, again, they are so insignificant that the patient makes no mention of them. Paræsthesia, such as formication and slight numbness, is often complained of. Anæsthesia is not present. The nerve-trunks, as a rule, are somewhat sensitive, and in some instances even painful to pressure. Tender points not related to nerve-trunks are sometimes found upon the limbs and upon the trunk. Spinal tenderness has also been noted.

During the height of an attack the patient may become quite warm, but, as a rule, the temperature in tetany remains unaffected. However, it occasionally shows variations; both rise of temperature and subnormal temperature have been noted. Febrile conditions when present can sometimes be explained by the existence of complications, but there is a residue of cases in which rise in temperature is found which cannot be thus accounted for. The respiration is, as a rule, unaffected. Very infrequently, however, dyspnœa may be produced by the involvement of the chest muscles or perhaps by the involvement of the diaphragm. In uncomplicated cases the pulse is at times decidedly increased. Strangury has also in rare instances been noted, due doubtless to participation of the bladder and its sphincter. The urine may not reveal any decided change. Polyuria, however, transient in character, has been observed. Albumin has in a few instances been discovered, the albuminuria being in such cases generally independent of nephritis. Nephritis, it should be remembered, occasionally occurs in tetany, and when albumin is detected in a given case the possible involvement of the kidney should be considered. Among the less frequent conditions present are acetonuria, phosphaturia, and transitory glycosuria. The perspiration is in some cases decidedly increased.

Œdema and redness in the neighborhood of the joints have occasionally been noted, as have also herpes and urticaria. Rarely, falling out of the hair and of the nails or transverse markings of the nails accompany the disease. When present, such symptoms as these denote grave nutritive disturbances. Muscular atrophy deserves to be mentioned as among the unusual symptoms. The reflexes in tetany show nothing characteristic; thus the knee-jerks may present almost any condition. As a rule, they are normal. They may, however, be exaggerated, or, on the other hand, though infrequently, they may be absent. Ankle clonus has in a few cases been observed.

Pathological Anatomy.—Autopsies in tetany have at times yielded negative results; at other times, when changes have been discovered, these have been unessential and variable. Among them may be mentioned serous exudation in the cervical cord and in the ventricles of the brain, sclerotic changes, spinal extra-dural hemorrhage, atrophy in ganglion cells and nerve-fibres, and proliferation of the neuroglia. Definite changes have never been observed in the peripheral nerves or in the brain, while in the spinal cord such as have been noted have been found by preference in the cervical enlargement and most frequently in the anterior horns of the gray matter. However, such symptoms as epilepsy, polyuria, and glycosuria, and the occasional occurrence of mental symptoms, such as confusional insanity, prove to us that the affection cannot be limited to the spinal cord. Some authors, because of the peculiar reaction of the nerves during life to mechanical and electrical excitants, have regarded tetany as a peripheral disease. A more conservative view, and perhaps the only one which will reconcile the scanty facts in our possession, is to regard the symptoms of tetany as the result of the action of a toxin or toxins upon the entire nervous system.

Duration, Course, and Prognosis.—The duration of an attack of tetany is very variable. It usually lasts several weeks. Cases have, however, been described in which the entire disease was characterized by but a single attack of spasm. In such cases the paroxysm may make its appearance, last several hours, and then subside without any further signs of the disease being manifested. On the other hand, there are cases which are exceedingly prolonged, extending over several months; but we must bear in mind that such long-continued cases are characterized by more or less long remissions, in which the symptoms are practically in abeyance for days and even weeks. It is a question whether such cases should not be described as those of recurring tetany, and not of chronic tetany.

Infrequently epileptiform convulsions complicate tetany. It is not improbable that in some cases the two diseases, epilepsy and tetany, are coincident. In others, however, there can be no doubt that the epileptiform seizures are directly dependent upon and closely associated with the tetany.

The prognosis of tetany is, on the whole, favorable. The majority of cases recover. Indeed, fatal ones are rare. An exception to this statement must, however, be made in tetany associated with dilatation of the stomach. Here, for some unknown reason, the disease assumes a grave character and frequently terminates in death. Out of eleven cases, for instance, collected by Fränkel-Hochwart, there were ten deaths. An exception must also be made as regards tetany which is the result of thyroid extirpation. Here a fatal outcome is also by no means infrequent. Cases of this kind are, moreover, especially unpromising because they are apt to be associated with symptoms of myxœdema.

The shortest and mildest cases of tetany are those forms which follow infectious diseases. In children the prognosis is largely based upon the condition of general health. It should be remembered that children not infrequently die, not of tetany, but of an associated disease; for example, rickets or bowel complaint. Not infrequently persistent laryngeal spasm is the factor which determines the fatal outcome. As ordinarily observed in children, however, let us repeat, a fatal outcome in tetany is rare.

Diagnosis.—The diagnosis of tetany is exceedingly simple. The characteristic position of the hands, the muscular pains, the Trousseau, the Erb, and the Chvostek symptoms, leave no room for doubt. Tetany is readily distinguished from epilepsy, as the attack comes on less suddenly, the spasm of the muscles is tonic, and there is no impairment of consciousness. Now and then hysteria simulates tetany. Here, however, the Trousseau or the Erb symptom will enable us to differentiate between them; besides, in a case of hysterical contracture simulating tetany it is improbable that the attacks would come and go as do those of tetany, and in addition various hysterical stigmata would in all probability be present and enable us to at once determine the nature of the case. Organic brain and cord affections in rare instances present symptoms superficially resembling tetany. A close study of the cases, however, should leave no doubt as to the actual conditions present.

In a case in which tetany is suspected, but in which the physician sees the patient only in the intervals and has not the opportunity of observing him during the spasm, the presence of the Trousseau, the Erb, and the Chvostek symptoms will enable him to make a diagnosis; indeed, latent tetany—that is, tetany in which a history of spasm is absent—can not infrequently be thus discovered.

Treatment.—In the treatment of tetany general measures are first to be applied. Absolute rest in bed and a nourishing and readily digestible diet, with occasionally tepid sponge-bathing, should be instituted. In addition, a mild, constant galvanic current may be applied to the affected muscles once or twice daily. We should devote our attention especially to the maintenance of the general nutrition, and to this end give tonics and, if rickets be present, cod-liver oil. Inasmuch as we are aware that many cases of tetany have their origin in gastro-intestinal troubles, these, if present, should receive prompt attention. The bowels should be emptied, and we should aim as much as possible at intestinal antisepsis. This is the plan advocated by Oddo. If there be reason to suspect worms, an effective anthelmintic should be given.

To control the muscular spasm various remedies have been from time to time administered,—*e.g.*, bromide, chloral, belladonna, and hyoscine. Antipyrin deserves to be especially mentioned because it appears to be of real value, and tends also to give relief to the laryngo-spasm, if present. Evans recommends Calabar bean as efficacious. In bad cases chloroform has been resorted to.

Because of the *rôle* which the thyroid gland appears to play in tetany, Bramwell, Gollstein, and others have administered thyroid gland to a few cases with benefit. Thomas, however, who gave this remedy a trial in a child, abandoned it, as the patient appeared to get steadily worse. Notwithstanding, it is worthy of a more extended trial. In children it should, of course, be given in relatively small doses.

MARANTIC THROMBOSIS:

PRIMARY THROMBOSIS OF THE CEREBRAL SINUSES IN CONNECTION WITH GENERAL MALNUTRITION.

By PEARCE BAILEY, M.D.

Marantic thrombosis of the cerebral sinuses, as distinguished from septic or inflammatory thrombosis, occurs as the result of such exhausting conditions or diseases as impair the propulsive power of the heart or as render the blood liable to coagulation within the vessels. After death the veins which empty into the sinuses are unusually prominent and filled with dark blood, and the sinus or sinuses contain firm, dense, partially organized clots which lie free in the lumen of the vessels.

The symptoms of the condition fall naturally into three categories: 1, symptoms of the conditions which have determined the thrombosis; 2, symptoms referable to disturbances of circulation in the particular vessel which is occluded; 3, symptoms of associated intra-cranial lesions.

The symptoms of the first class are those of the diseases which have caused the marasmus. Pulmonary diseases and protracted diarrhœa figure most prominently in this respect. The symptoms of the second class are only rarely pronounced, being most frequently masked by those of the cachexia. They are most common in the first six months of life, although they are sometimes seen in children of the age of five or seven or even more years. They are ushered in by general convulsions and rise of temperature. Subsequent symptoms are focal, and vary according to the vessel which is the seat of the thrombosis. If it is a vein of the cortex which is affected, there is monoplegia or hemiplegia associated with the ordinary evidences of cortical lesions. If one sinus alone is affected, it is almost unexceptionally the superior longitudinal. As into this sinus empty the veins of the nose, forehead, and occiput, its obstruction results in an œdema and congestion of these parts, of which the clinical manifestations are pain, bleeding from the nose, and swelling of the forehead, temples, and back of the head. When other sinuses are occluded, the occlusion occurs as an extension from the superior longitudinal sinus, and the symptoms of obstructed venous return from parts drained by them are added to the symptoms of superior longitudinal sinus thrombosis. Thus from stasis in the cavernous sinuses result various ocular conditions, such as pain, œdema of the eyelids, exophthalmos, optic neuritis, or palsies of muscles supplied by the oculo-motor nerves. If the thrombosis reaches the lateral sinus there may be pain and œdema over the mastoid process. Associated cerebral lesions are not at all uncommon in marantic thrombosis of these sinuses. They consist in general œdema of the brain, ventricular distention, softening, and meningeal hemorrhage. The occurrence of these complications renders the clinical picture still more obscure, and their exact diagnosis is ordinarily impossible.

The diagnosis of marantic thrombosis of the cerebral sinuses depends upon the consideration of general symptoms rather than upon the local effects of obstructed venous circulation. If at the end of an exhausting disease the patient develops convulsions and an increase of temperature, the possibility of thrombosis must at once be thought of. If unequivocal signs of local obstruction to the return of venous blood are present, the diagnosis becomes reasonably sure. In the absence of such evidences it is impossible to distinguish the trouble from cerebral abscess or ear disease unless the symptoms of these latter conditions are unmistakable. In many cases thrombosis is found after death, although during life no symptoms existed by which it could have been recognized. The prognosis is always serious and directly dependent upon the prognosis of the cachectic condition, of which the thrombosis is only a secondary result. The treatment must be directed towards the underlying cause. Surgical measures, which are sometimes effectual in thrombosis of septic origin, are of course unavailing when the vascular condition is dependent upon malnutrition.

ACROMEGALY.

By SOLOMON SOLIS-COHEN, M.D.

Historical.—Acromegaly (ἄκρος, at the extremity, and μέγας, great), pachyacria, or Marie's disease, was first described by P. Marie[1] in 1885, from cases studied in the wards of Charcot at the Salpêtrière. Its literature has been steadily growing, among the most important communications being the theses of Souza-Leite, published in 1890,[2] and of Hinsdale,[3] published in 1898.

From the very nature of the affection it is not likely to be seen or recognized in children save in rare instances, and its special pediatric literature is therefore scanty.

Moncorvo[4] records a case associated with microcephalus in a female infant of fourteen months. Rake[5] described a case of doubtful diagnosis occurring in a negro boy aged ten years. McGregor's[6] case of unilateral hypertrophy of the extremities, with spinal curvature and facial deformity, is likewise doubtful. Hinsdale alludes to four published cases in which the age at the onset of the disease was under ten years, and nineteen cases in which it was between eleven and twenty years. The cases of macrodactylia accumulating in literature are of various pathologic characters, and no relation between them and acromegaly has been established.

General.—Acromegaly is a trophic disorder, slowly developing and persistent, of unknown etiology and pathogenesis, but probably belonging to a group of atavistic or reversionary tropho-neuroses. It has a close association, not yet satisfactorily explained, with morbid alterations of the thyroid gland and of the pituitary body, with, in some cases, persistence of the thymus. It has been conjectured that some internal secretion may be at fault; by excess causing toxæmia, or by deficiency permitting it through failure to neutralize deleterious metabolins, but as yet this is merely guesswork. The affection is characterized by extensive tissue-changes, chiefly hyperplastic or sclerotic, the most evident being hardening of cartilage and overgrowth of bone and of related soft parts. The principal changes affect the face, the spinal column, and the extremities, giving rise to a characteristic physiognomy, to cervico-dorsal kyphosis with compensatory lordosis, and to enlargement, chiefly broadening, of the hands and feet; but any portion of the bony or cartilaginous skeleton, with its overlying tissues, may be involved. Vascular and other growths are often present. The skin of the affected parts is coarse, thick, hard, and greasy; pigmentation is not infrequent. Macroglossia is a common accompaniment. The heart and vessels are frequently affected, the latter becoming hardened, enlarged, and tortuous. Venous varicosities are more marked and more frequent than arterial changes. In some instances the lymphatic system is likewise involved, both glands and vessels participating; the visceral (sympathetic) nervous system sometimes exhibits marked lesions, polyneuritis has been demonstrated, and visceral changes in general have been found in advanced cases. Secretions are disturbed, but not always in the same manner. Pain, disturbances of special senses, and other functional disorders arise in accordance with the degree and distribution of special lesions.

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[1] Marie, Revue de Médecine, 1886, t. vi p. 297 *et seq.*

[2] J. D. de Souza-Leite, De l'Acromégalie, Thèse de Paris, 1890.

[3] Guy Hinsdale, Acromegaly, Boylston Prize Essay, Medicine, Chicago, June, July, August, and September, 1898.

[4] Revue mensuelle des Maladies de l'Enfance, t. x. p. 549, 1892.

[5] British Medical Journal, March 11, 1893, p. 518.

[6] Glasgow Medical Journal, 1894, vol. xli. p. 189.

Symptomatology.—At first acromegaly was supposed to be a comparatively rare affection, but in the last ten years instances have been recorded in such number that reports of single cases no longer attract attention, unless for some unusual feature, clinical or post-mortem. I observed in my own practice within a period of five years three undoubted cases and two doubtful ones, and I have seen probably twice as many in the practice of others. The early stages of the affection rarely attract attention, and even advanced cases may escape notice and record unless one is on the lookout for them. The characteristic alterations of form and feature come about so gradually that they are usually looked upon as simple ugliness, and neither the subject nor his friends consider them a matter for medical advice. When medical advice is sought, it is for some complication, such as headache or polyuria. One of the most marked cases that I have seen is in the person of a physician whom I met at a medical gathering in the interior of one of the Eastern Atlantic States, and neither he nor the other members of the society seemed to have the slightest suspicion that his condition was pathologic. My first case was in the person of a big man twenty-five years old,[1] who had been selected on account of his size and supposed strength as a member of the foot-ball team of a New England college, a post from which his awkwardness and the ease with which he could be withstood by much smaller men soon caused his dismissal. The students had noticed in the man what may be considered one of the most characteristic symptoms,—

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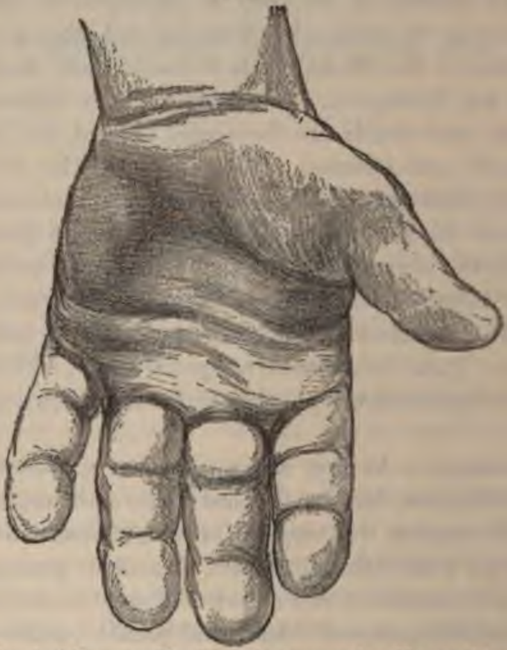
[1] Transactions of the College of Physicians, Philadelphia, third series, vol. xiv, 1892, p. 156.

namely, the disproportionate breadth of the hand (Fig. 1),—and graphically described it by saying that "X. could not play base-ball because, if he forgot to turn his hands edgewise when running the bases, the wind would blow him back." This unaccountable growth of the hands, and similar changes in the feet, are usually the first symptoms to attract attention.

FIG. 1.



Spade-shaped hand with sausage-like fingers in acromegaly. Man aged twenty-five years.

Larger and larger sizes of gloves and of shoes become necessary, and then a larger hat must be got on account of the growth of the head. The bones and cartilages of the face increase in size, the big nose first attracting attention. The vertical enlargement of the chin, with protrusion of the lower jaw, the lateral projection of the enlarged cheek-bones, and the prominence of the superciliary ridges, due both to external thickening and to enlargement of the frontal sinuses, cause a characteristic physiognomy, the outline of the face being an irregular oval (hexagon) with angles replacing the curves, thus forming a marked contrast with the round, moon-shaped face of myxœdema or cretinism and the triangular outline of osteitis deformans (see Fig. 2), with which it is most likely to be confounded. The tongue and lips are thickened, and the overhanging of the lower lip adds to the repulsiveness of the patient's appearance.

Usually the patient grows taller, too, but cases are on record in persons of small size, as the woman whom I exhibited to the Philadelphia County Medical Society.[1] Whether the patient be tall or short, giant or dwarf, the thickening of the vertebræ and intervertebral cartilages and the weakening of the muscles lead to cervico-dorsal kyphosis, with compensatory lordosis. At first it may simply be noticed that the patient carries his head thrust forward between his shoulders, as if always looking down upon the ground in front of him; afterwards the hunching of the back and the protrusion of the abdomen (like Punchinello) become evident, and these may progress so far that the ribs and the crests of the ilia may overlap.

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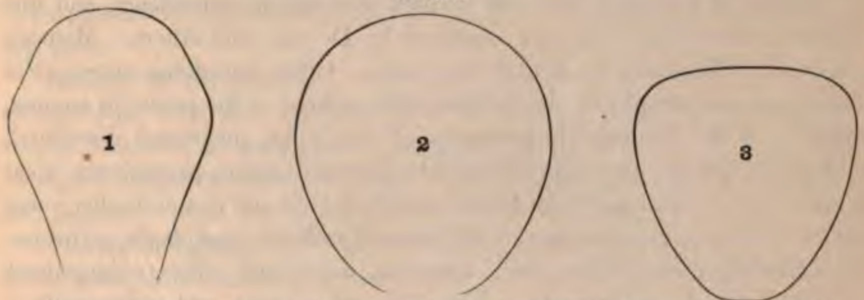
[1] Vide infra.

FIG. 2.



Outlines of face in (1) acromegaly, (2) myxœdema, and (3) osteitis deformans. (After Marie.)

In some cases the ribs themselves become much thickened, so that the intercostal spaces become nearly, if not quite, obliterated, and much restriction of the movements of the chest, with consequent dyspnœa and vertical or abdominal respiration, ensues. Enlargement of the turbinate bodies may still further impede respiration. The larynx usually enlarges and the cartilages thicken, harden, and calcify prematurely. The voice may be deepened and roughened in consequence; usually it has a peculiar monotone. The auricles also undergo thickening, and the ears frequently protrude from the sides of the head. The thyroid gland may be enlarged or may be atrophied. The skin becomes thick, tough, and coarse; the finer markings are lost, the coarser deepened. Where motion causes wrinkles, as on the forehead and across the abdomen, there are developed great rounded ridges with intervening furrows. Pigmentation, general or local, is common. The nails are thickened, flattened, broadened, striated. The hair usually grows coarse and stubbly, and in a man the arms and the chest may be covered with such hair. Varicosities of the veins, especially in the legs, become more and more evident. The arteries, less evidently, become enlarged and fibroid. The pulse-rate is usually accelerated, but the action of the heart is feeble, and this is made more evident upon exertion. While the enlargement of the muscles that accompanies the other changes gives the patient the appearance of strength, this, as in the case of the foot-ball player referred to, is deceptive. There is really weakness. Perspiration is usually excessive and occurs with undue readiness. Polyuria is common, the urine sometimes containing sugar, less frequently albumin. Phosphaturia and albumosuria have been observed. As a rule, digestive disturbances are not complained of, though usually there are polyphagia and polydipsia.

Headache is one of the most frequent and distressing symptoms. It becomes more and more constant and intractable, and when, as often happens, visual disturbances (restriction of the fields; hemianopsia, often bitemporal) become superadded, one is justified in referring this to enlargement of the pituitary body. Optic atrophy has been observed. Cases with exophthalmos and with exophthalmic goitre[1] have been reported.

In some cases there have been impaired hearing, loss of smell, and disorder of taste.

In two of my cases there was decided tendency to somnolence, and this excessive drowsiness has been observed by Dercum and others. Memory may fail. There may be mental depression. Other symptoms, more or less inconstant, are atrophy of the testicles, enlargement of the penis (in women, atrophy of the mammæ, hypertrophy of the labiæ, menstrual disorders), epilepsy, migraine, neuralgia and pseudo-rheumatic pains, paræsthesia, great sensitiveness to heat and cold, blueness of the hands and feet and other vasomotor disorders, exaggeration or abolition of reflexes, paraplegia, arthropathies,[2] enlargement of the heart, lipomata, moles and warts, enlargement of lymphatic glands, impotence, loss of sexual appetite, and melancholia.

In a few instances there has been observed an impairment of the percussion note (Erb's dulness) over the upper portion of the sternum, supposed to indicate persistence of the thymus. It has been wanting in cases in which persistent and enlarged thymus was found.

Syringomyelia[3] has been associated with acromegaly, and in one of my cases, presenting bitemporal hemianopsia, glycosuria, and pigmentation, myxœdema seemed to be present as well.[4]

Pathology.—The changes found in the bones and other structures are essentially those of overgrowth, not of inflammation. In the bones it seems to proceed from the periosteum, affecting connective tissue and adipose layers, and from the inner surface giving rise to osseous new formation, —"an intense peripheral histogenesis."

The hypertrophy affects specially the bones of the extremities and the extremities of the bones. (Marie.) The sutures of the skull may have disappeared; its air-cavities are enlarged; the facial bones, especially the zygoma and inferior maxillary, are thickened and increased in length and breadth. The sella turcica may be much enlarged. The prognathous skull with its great air-cavities presents marked resemblances to those of the Neanderthal man and of the anthropoid apes.

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[1] Lancereaux, La Semaine médicale, February 16, 1895, p. 61. Also others are cited by Hinsdale.

[2] A. A. Eshner, Medical News, Philadelphia, October 26, 1895, p. 458. In one of my cases there was rheumatoid arthritis.

[3] F. Peterson, Medical Record, New York, September 23, 1893, p. 391.

[4] Proceedings of the Philadelphia County Medical Society, 1895, vol. xvi. p. 163; Philadelphia Polyclinic, September 7, 1895, p. 372. The patient died in coma. Autopsy was refused.

The skin of the affected parts shows hyperplasia of all the component structures,—papillæ, derma, connective tissue, sweat-glands, sebaceous glands, vessel-walls, and nerve-sheaths. In the visceral nervous system are found sclerotic changes, which may especially affect the lower cervical ganglion. The semilunar ganglion is sometimes enlarged and hardened. Optic neuritis is common. The lymphatic vessels and glands show hyperplastic and sclerotic changes. There may be sclerosis and fatty changes in the liver; the kidneys may be sclerotic; the spleen may give evidence of hyperplasia and degenerative changes. Blood-changes are not commonly reported.

Lesions of the pituitary body are frequent, and vary from simple enlargement to cystic degeneration and tumor-growth, vascular hypertrophy, sarcoma, adenoma, and glioma having been found. On the other hand, acromegaly has been observed without change in the pituitary body,[1] and pituitary tumor has been reported without acromegaly.[2]

The thyroid gland has been found normal at some autopsies; at others it has been absent or atrophied; at others, enlarged, and then simply hyperplastic, or fibroid, with or without cystic or colloid degeneration. Persistence of the thymus is common, but not invariable. The persistent thymus is usually enlarged, but no definite lesion has been demonstrated; nor is there any constant association to be made out as to the presence or absence of thyroid, thymus, or pituitary lesions with relation to one another. Nevertheless, the consensus of opinion looks to further researches upon the functions of these organs to throw light on the strange disorder of nutrition under consideration. I have elsewhere recorded my opinion that there is a compensatory relation between the thyroid and the pituitary, and an antagonistic relation between the thyroid and the thymus.

Etiology.—No definite statement can be hazarded as to causation. Marie declares the disease not to be congenital; yet I must believe that it is the result of congenital tendencies. Heredity does not seem to play a part, although Bonardi observed the disease in father and son,[3] and in many reported cases there has been noted a neurotic ancestry.

Diagnosis.—Most of the cases reported have been in persons past middle age. Some have been in young adults, a score in adolescents of both sexes, two in boys of ten years, and one in an infant girl. It is probable that in most cases the changes begin to be noticeable between the twentieth and thirtieth years, and that to detect the disease earlier will require either very marked lesions or excessively close scrutiny upon the part of the physician. A well-developed case in an adult is unmistakable. In children the chief sources of error would be in mistaking for acromegaly irregular hypertrophy of hands, feet, or face, pulmonary hypertrophic osteo-arthropathy, and cretinism.

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[1] Bonardi (vide infra). But Hinsdale (op. cit.) and M. Sternberg deny the occurrence of true acromegaly without pituitary lesion.

[2] F. A. Packard, American Journal of the Medical Sciences, June, 1892, p. 657; Hinsdale, Medicine, August, 1898, p. 687; Burr and Riesman (personal communication). The latter calls attention to the fact that adequate studies have not been made of the entire structure in the reported cases. In the case studied by Dr. Riesman a small portion of fairly healthy pituitary gland structure was found.

[3] Archivo Italiano di Clinica Medica, 1893. Cited in the Medical News, February 17, 1894, p. 186.

It may be that when greater light is thrown upon the true pathology of acromegaly, cases of macroglossia[1] and of irregular hypertrophy of the face and of the extremities may be properly affiliated therewith. For the present, however, I feel that such cases ought to be excluded, and that to justify the diagnosis of acromegaly the enlargements should be symmetrical, progressive, and associated in the characteristic manner described under symptomatology.

Gigantism, general or special, is not acromegaly, and is to be distinguished either by the isolation of the part affected or by the general proportionate growth of all the parts and in all directions. In acromegaly the enlargement of the hands, feet, and facial bones is disproportionate to the growth of the rest of the body and disproportionately great in the transverse direction in the structures affected. Thickening exceeds elongation. Thus, as in the case of the student before cited, the great breadth of the hands is noticeable. They enlarge in length also, but not in the same proportion. The fingers are round, deeply creased at the joints, but with the markings rather obliterated elsewhere. Thus the descriptive terms of "spade-like" hands with "sausage-shaped" fingers are justified. In the feet there is to be noticed the "negro heel," or backward projection of the os calcis, and fleshy pads laterally around the great toes. Skiagraphy shows hyperostosis of the heads of the phalanges in hands and feet. In myxœdema the hands are likewise spade-shaped, but the enlargement is distinctly of the soft parts only, not of the bones as well. The characteristic doughy sensation to the touch present in myxœdema is absent in acromegaly. Myxœdema and acromegaly may, as in one of my cases, be associated. Cretins resemble closely the subjects of myxœdema. They are undersized, but not necessarily affected with spinal curvature. The physiognomy is unlike that of acromegaly. Acromegalic patients do not necessarily exhibit the intellectual deficiencies of cretins.

In pulmonary hypertrophic osteo-arthropathy there may be spinal curvature, but usually a dorsal or dorso-lumbar kyphosis, the cervical region escaping. The lower jaw may enlarge and project, but the cheek-bones are unaltered. The nose, if affected, is pinched rather than thickened. The wrists and ankles are enlarged, while in acromegaly they are, as a rule, little changed. In the hands, as will be seen from the accompanying illustration (Fig. 4), it is the terminal phalanges alone that are affected, giving to the ends of the fingers a somewhat bulbous shape, and not producing either the spade-like alteration of the hands or the sausage-like condition of the fingers characteristic of acromegaly (Fig. 1). In acromegaly (Fig. 3) the nails are flattened, broadened, and striated either longitudinally or horizontally, the finger-pads usually projecting beyond the nails. In the alteration of the fingers associated with pulmonary disease the nail is

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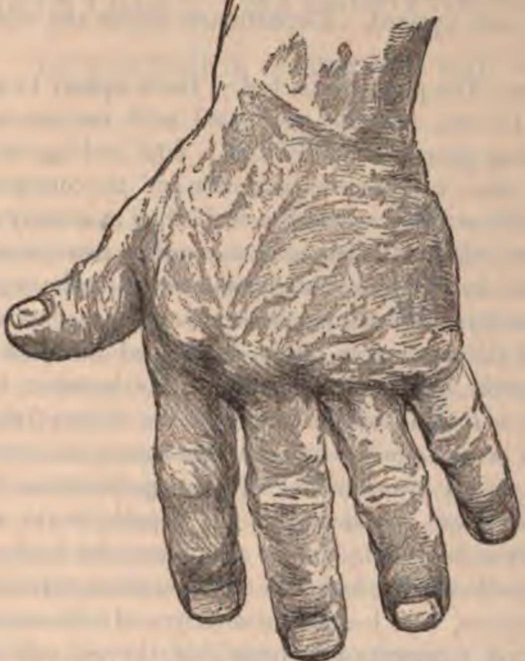
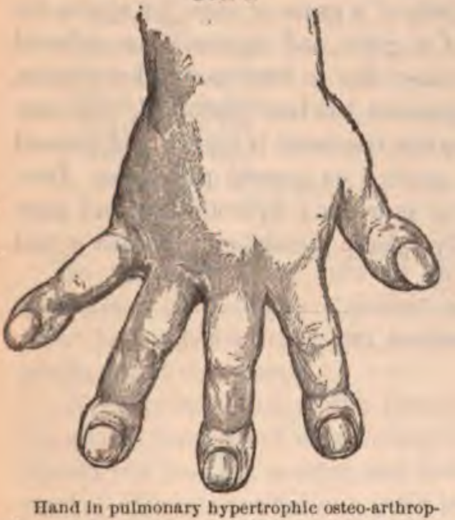
[1] Cf. Chauffard's case; Le Mercredi Médical, 1895, No. 29, p. 345.

FIG. 3.



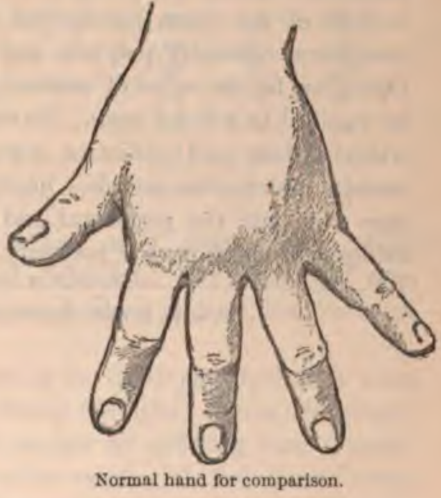
Hand in acromegaly, showing enlarged veins and flattened nails. Man aged fifty-two years.

FIG. 4.



Hand in pulmonary hypertrophic osteo-arthropathy. From a boy aged eleven years, with non-tuberculous empyema.

FIG. 5.



Normal hand for comparison.

curved in both directions and projects beyond the finger-pad, giving to the finger in profile the outline of a parrot-beak. Then, too, in the latter case there is empyema, bronchorrhœa, or other septic affection of the chest, which is absent in acromegaly.

In osteitis deformans it is the brain-case rather than the bones of the face that is affected, giving the countenance the outline, already described, of a triangle, base upward. Elephantiasis affects the skin only, not the bones.

Prognosis.—The prognosis is bad. There appear to be two types of the disease. In one, probably associated with sarcoma of the pituitary body, the lesions progress rapidly and a fatal ending occurs within five years; in the other, the progress is slower and the continuance almost indefinite, one patient being recorded as still alive at seventy years of age.

Treatment.—Palliation has been obtained in some cases by administration of preparations of the thyroid gland or of the pituitary gland, or of both together. Latterly, difference has been shown to exist between the physiologic action of the anterior or pituitary lobe and the posterior portion, or hypophysis cerebri, of the pituitary body. As, however, in some cases of acromegaly it is the one and in other cases the other of these lobes that is affected, while in still others both structures have been involved, no preference can be given *a priori* to one of these preparations over the other; although embryologic considerations might point to the anterior lobe as the more likely to be useful. In one of my cases the headache was entirely relieved by thyroid extract, and the other symptoms, especially the polyuria and the somnolence, were lessened in severity. In the case associated with hemianopsia and symptoms of myxœdema, thyroid extract removed the myxœdematous symptoms, but utterly failed to relieve the headache, which, indeed, could only be controlled by morphine narcosis. Picrotoxin in as large doses as can be borne (one-thirtieth of a grain or more for adults, for a child of ten years, one-sixtieth of a grain and upward) has relieved symptoms, especially polyuria and those due to vaso-motor disturbance. Operation for the relief of cerebral pressure has been attempted,[1] and may be justified in selected cases. No curative treatment is known, and, beyond what has been said, palliation must proceed on general principles. Iron, arsenic, and similar remedies improve nutrition; hydrotherapy and massage stimulate the peripheral and lymphatic circulation. Sedatives and analgesics may be used if necessary.

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[1] Caton, British Medical Journal, 1893, vol. ii. p 1421.

SYRINGOMYELIA (MYELOSYRINGOSIS).

BY FREDERICK PETERSON, M.D.

SYRINGOMYELIA may be defined as a chronic disease of the spinal cord characterized by the formation of cavities in the central gray matter, and giving rise to peculiar sensory symptoms, together with various trophic disorders. The name myelosyringosis proposed by Foster is a better word philologically, but its synonyme has the advantage of common usage. *Hydromyelia*, or hydromyelus, is a congenital dilatation of the central canal of the spinal cord, and is not to be confounded with the disorder under discussion, which is of pathological origin. We owe the name syringomyelia to Ollivier (1837), though the existence of cavities in the spinal cord was known to pathologists as long ago as the time of Morgagni. It is only within some fifteen years that the symptomatology and the relations of the symptoms to the characteristic lesions have been thoroughly understood. The literature is now very extensive.

Etiology.—The age at which the disease develops is best shown by Schlesinger's table:

AGE.	MALE.	FEMALE.	TOTAL.
One to ten	4	1	5
Eleven to twenty	36	8	44
Twenty-one to thirty	53	25	78
Thirty-one to forty	30	12	42
Forty-one to fifty	4	7	11
Fifty-one to sixty	3	3	6
Sixty-one and over	3	1	4
	133	57	190

In the table of one hundred and ninety cases, forty-nine, or about twenty-five per cent., were children and adolescents. Of these forty were males and only nine females; so that, in children at least, the male sex predominates enormously.

Heredity has usually little influence in its development, though there are several instances of its assuming a family character. Thus, Ferrannini reports two brothers, a sister, and their mother all suffering from syringomyelia. Trauma is cited as a cause in a few cases, while infectious diseases seem to predispose to its development in many.

Pathology and Pathological Anatomy.—Cavities are sometimes found in the cord and in the brain as the result of vascular lesions, but true syringomyelia is now looked upon as cavity-formation following upon an infiltration of the substance of the cord with gliomatous cells. The gliosis begins about the central canal, and as the infiltration of cells proceeds there may be a slight or considerable tumefaction of the cord. Degeneration and breaking down take place in the newly formed tissue, resulting in the creation of a cavity or cavities. The disorder is nearly always situated in the cervical or upper dorsal region, but has been observed in the lumbar region of the cord. The cavity, while usually limited in extent, may reach upward into the medulla and even into the pons. When there are several cavities, there are frequently communications between them. As a rule, the cavity is irregular in outline, filled with fluid, with a delicate lining membrane, and surrounded by gliomatous tissue. The gray matter is always earliest and most affected, but invasion of the white matter to a greater or less extent takes place in most cases.

Symptomatology.—The clinical manifestations of syringomyelia are such as one would expect from lesions affecting the central gray matter of the cervical cord at different levels, associated with invasion of the lateral and posterior columns, when we remember the important functions of these centres and tracts. The symptoms may be classified as follows:

I. Lesions of the gray matter.
   1. Anterior horns (paralysis with muscular atrophy according to the levels affected; loss of reflexes).
   2. Central gray matter (trophic disorders of skin, nails, joints, bones, vaso-motor symptoms, cilio-spinal disorders, scoliosis).
   3. Posterior horns (analgesia; thermo-anæsthesia).

II. Lesions of the columns.
   1. Lateral columns (stiffness or spastic rigidity of legs; exaggeration of knee-jerks; ankle clonus).
   2. Posterior columns (tabic symptoms, such as incoördination, pains, loss of knee-jerk).

There are two which may be looked upon as cardinal symptoms,—viz., progressive muscular atrophy, with loss of the temperature and pain sense in areas innervated by fibres from the level of the lesion. The amyotrophic symptoms are almost always precisely like those of the Aran-Duchenne type of progressive muscular atrophy, with degenerative reaction and fibrillary tremor. Doubtless many cases of syringomyelia were in former years diagnosticated as progressive muscular atrophies. The peculiarity of the sensory disturbance is, therefore, the chief distinctive feature. Owing to the different paths taken by different sensory impulses, we usually find in this disease a disassociation of sensory perceptions. Thus the pain and temperature senses are lost, while tactile and muscular sensibilities are preserved.

But in addition to muscular atrophies, we observe frequently other trophic disorders, together with vaso-motor symptoms, such as cyanosis, bullous eruptions, ulcers, glossy fingers, gangrene, fragility of the bones, Charcot joints (in the upper extremities almost always). Scoliosis, slight or marked, is a very common symptom. It is partly due to paralysis and atrophy of spinal muscles, and probably also in part caused by trophic changes in the vertebral joints and their ligaments.

In over twenty per cent. of the cases inequality of the pupil has been noted. Nystagmus is sometimes seen.

Whenever the disease extends to the bulbar region, we have, in addition to the symptoms described, others characteristic of lesions at this point, such as laryngeal paralysis, the bulbar-palsy syndrome, and anæsthesias of the mouth, throat, and larynx.

In more than half of the cases there is exaggeration of the knee-jerks, and in some fifteen per cent. spastic symptoms.

Syringomyelia is sometimes associated with other diseases, such as hysteria, hydrocephalus, hydromyelia, tumors and syphilis of the central nervous system, and more rarely acromegaly. (See case of writer, *Medical Record*, September 23, 1893.)

Diagnosis.—In cases of progressive muscular atrophy and of amyotrophic lateral sclerosis the distinguishing symptom must be the peculiar disassociation in the sensory perceptions. But in patients presenting sensory disassociation we must remember that we nowadays recognize such disassociation in a number of other disorders, as, for instance, in certain cases of neuritis and in anæsthetic leprosy.

Anæsthetic leprosy is differentiated by anæsthetic discolored plaques on various parts of the body (instead of the large areas of anæsthesia of syringomyelia), by irregular thickening of nerve-trunks, and by atrophic paralyses of facial muscles.

The main difficulty of diagnosis will always be between syringomyelia and other lesions which may occupy the same location in the cervical cord, such as central hemorrhage and tumors of one kind or another. A sudden onset characterizes hemorrhage. With tumors of specific nature the presence of positive syphilitic signs or syphilitic disease elsewhere would be helpful, and the same may be said of tubercle. The progress of syringomyelia is very slow, much slower than that of ordinary neoplasms.

Course and Prognosis.—The disease is exceedingly chronic in its course, some patients having been known to live as long as forty years. Recovery never takes place, of course, but death is usually due to intercurrent maladies or to disorders incidental to some of the manifestations of the disease itself, such as bed-sores, ulcers, phlegmon, or bulbar symptoms.

Treatment.—The chief indications in treatment are to alleviate so far as possible some of the more trying symptoms, and to build up the system with tonics, good air, and good food. The relief of pain is frequently demanded, and naturally the exhibition of drugs liable to lead to habitual use should be avoided. The use of morphine should be a last resort. Combinations of phenacetine or antipyrin with codeine are effectual and fairly safe. Counter-irritation, the cautery, hot applications, and the like are generally contra-indicated because of the danger of trophic lesions. Massage and electricity should be employed for muscular atrophies in about the same manner as in other forms of amyotrophy.

GYROSPASM AND SPASMUS NUTANS IN INFANTS.

Definition.—Gyrospasm is a term given by the writer to the rotary movements of the head observed in early infancy as a result probably of concussion. Spasmus nutans has the same clinical history, etiology, and pathology, and is a nodding movement of the head. Gyrospasm is much more common than spasmus nutans.

History.—Henoch described these morbid movements long ago in his work on diseases of children. Hadden, however, was the first to describe the disorder in minute detail in a paper on "Head-Nodding and Head-Jerking in Children, commonly associated with Nystagmus."[1] The writer's paper on "Gyrospasm of the Head in Infants," in which were reported five cases, appeared in the *Medical News*, Philadelphia, October 1, 1892. In the St. Thomas Hospital Reports for 1892, Hadden published "A Second Series of Cases of Head-Jerking in Children," with details of nine cases. Gordon Norrie has written upon the subject,[2] and Caillé has reported two cases.[3]

Etiology.—The disorder is one essentially of early infancy, most of the cases being considerably under ten months of age when brought for notice. The age of onset varies from three to fifteen months. Henoch has reported a case at three years of age. Osler[4] reports one at five years of age when examined, but the disorder had begun in earliest infancy. The sexes are about equally affected. In a seventh of the cases a history of antecedent convulsions will be obtained. In a somewhat larger percentage rickets is present. Henoch entertained the opinion that dentition bore a close relation to these morbid movements, but the evidence accumulated since his publication tends to show no interdependence between the two, save the possibility of aggravation of the symptoms by the nervous disturbances sometimes incidental to the physiological process. The writer has observed several cases beginning at the age of three or four months, Hadden records one at six weeks of age, and Gee one at a still earlier period. The evidence at the present time would tend to show that the condition is one of traumatic origin, a species of traumatic neurosis, using this latter phase in its true generic sense to include all neuroses developed by accident or injury. In over one-third of the cases a history of a fall upon the head has been obtained. In one of Hadden's cases the fall upon the head was so severe as to produce a large hæmatoma of the scalp. The nystagmus was first noticed two weeks afterwards.

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[1] Lancet, June 14, 21, 28, 1890.

[2] Centralblatt für prakt. Augenheilkunde, 1888, S. 229.

[3] Transactions of the American Pediatric Society, 1889.

[4] Montreal Medical Journal, 1896.

Pathology.—The process underlying the development of this neurosis is obscure, but as most of the cases recover perfectly, there is probably no permanent organic lesion. It is interesting to note that the spasm is concerned with muscles which are the earliest to acquire purposive movements (head and eyeballs). We know that in adults a rapid and temporary nystagmus may be set up by severe concussion of the brain. In lieu of the actual facts to guide us, we may suppose that in these infantile cases an arhythmic discharge of nervous force is induced by concussion in the centres of the spinal accessory and oculo-motor nerves. Snell, in his work on miners' nystagmus, observes that tremors of the eyelids and even muscles of the neck with slight nodding movements are often met with in connection with the nystagmus of miners. This is the only condition in adults which bears any analogy to the gyrospasm and spasmus nutans of infants.

Symptomatology.—The great majority of the cases exhibit a lateral movement of the head, while the nodding spasm is extremely rare, so that the term gyrospasm includes most examples of this neurosis. The motion is not rapid, the number of excursions per second seldom exceeding two or three. There is complete cessation of the movement during sleep, and often when the patient lies down. Often the motion ceases temporarily when the attention of the child is fixed. Sometimes it develops only, or is exaggerated, upon attracting the patient's attention. Occasionally the excursion of the head is greater to one side than to the other. Nystagmus is frequently associated with gyrospasm. The nystagmus may be lateral, vertical, or rotary, or may vary from one form to another at different times. A curious fact is that the nystagmus is limited to one eye in about half of the cases in which it is present as an associated symptom. The movements of the eyeball are usually twice as rapid as those of the head. Occasionally nystagmus is discovered only upon fixation of the eye. A very few cases have been reported with strabismus. The pupils are always normal, though in one patient there was hippus (Norrie). The media of the eye are normal and the fundus almost always so. In one of Hadden's cases there was deficiency of retinal pigment, and in another slight crescentic atrophy. Two patients of the same author presented nictitation in conjunction with nystagmus.

The general health of the patients is nearly always excellent, and usually we find no other evidences of central nervous disease than the symptoms above described. The mental state is good, and, indeed, it is the rule to find these patients rather above the average in intelligence.

Diagnosis.—The age of the patient and the absence of all nervous symptoms save gyrospasm and nystagmus render the diagnosis easy. It is only in rudimentary forms, as, for instance, when the latter only is present, that any difficulty in diagnosis arises. In a case with nystagmus solely, it is necessary to exclude the multiform causes of nystagmus in children before arriving at the conclusion that a case is a species of traumatic neurosis. Among the causes of nystagmus in children are blindness, various diseases of the cornea, lens, and retina, multiple sclerosis, cerebellar tumor, and, indeed, a number of disorders and defects of the central nervous system.

Prognosis.—The outlook for these cases is always favorable. Most patients recover in from three months to a year. Only one instance is recorded in literature (Osler's) in which the symptoms that had begun in infancy were still present at the age of five years. Relapses occasionally occur after apparent cure, but only when there is general debility from some intercurrent affection.

Treatment.—There is no doubt that the morbid movements described are favorably affected by moderate doses of bromide of potassium (three or four grains three times daily); but cases recover without any treatment. If the general health should be undermined, the usual tonics are indicated.

AUTOMATIC MOVEMENTS IN DEFECTIVE CHILDREN.

There are certain peculiar disorders of motility to be observed in feeble-minded, imbecile, and idiotic children. Besides the morbid movements found among them common to other pathological conditions, such as epilepsy, athetosis, chorea, tremor, ataxia, nystagmus, and associated movements, there is a class of mobile disorder which we may term automatic, impulsive, or imperative.

Normal children and young animals exhibit numerous spontaneous movements, undoubtedly due to impressions received at some time during their lives, or, it may be, to inherited impressions; and while such spontaneous movements are more or less similar in their nature to the automatic mobile disorders observed in defective children, they rarely present the rhythmical character of the latter. It is probable that in the feeble or enfeebled mind, upon which nerve stimuli seldom make much of an impression, some of the simple old motor expressions are retained, repeated, and ultimately become habitual and automatic. Automatism of movement, then, is a sign of little aptitude or impressionability so far as great mental stimulation is concerned.

On going through a large institution for defectives of the class here considered, one of the most striking symptoms immediately observed is the continual motion of a large proportion of the inmates. The most common form of rhythmical movement noticed is an antero-posterior oscillation. The patient in a sitting attitude sways his whole body slowly or rapidly forward and backward. Often this oscillation of the trunk is from side to side. Occasionally the hands and fingers are flexed and extended slowly or quickly. On the other hand, there are patients who walk to and fro, turn rapidly around, or dance about, these being more elaborate forms of the same motor phenomena. They are spontaneous movements, but under the control of the will, so that they may be easily stopped at command, to be resumed again immediately afterwards. They have no relation seemingly to any stimulation of the brain giving rise to a motor expression. Any sensory impression, such as that of seeing a stranger enter the room, or being spoken to, generally induces cessation of the movement for a brief period by temporarily altering the feeble current of thought or disturbing the mental blankness which has originated it. This same phenomenon so frequently observed in defective children has its analogue in the automatic motions of adult dements in our asylums for the insane.

The smiles and grimaces of mentally defective children belong in the same category of infantile spontaneous motor expressions.

The impulsive rhythmical movements above described, so far as the feeble-minded, imbecile, and idiotic are concerned, are to be looked upon, therefore, as the habitual motor expression of the simplest and oldest stimuli. But the analogous automatic motions of secondary dements are reversions to the spontaneous motor expressions of infancy.

NEURAL AND MENTAL DISORDER IN CHILDREN.[1]

BY FRANCIS WARNER, M.D. (LOND.), F.R.C.P., F.R.C.S. ENG.

As many forms of neurosis and mental disorderliness in children appear to be reversions to modes of brain-action normal in the infant, it is convenient to commence by a study of the healthy infant brain.

In the healthy new-born infant we see a well-developed body, complete and well-proportioned in all its parts. When he is awake and the brain is in full functional activity, we see movements in all parts; these movements are seen in the limbs, the fingers and toes specially, which move separately; they are slower than most of the movements in adults, but not dissimilar in the combinations of parts coacting. They are neither inhibited nor coördinated through the senses; hence we may assume that they are spontaneous or the outcome of the uncontrolled action of the nerve-centres acting separately and spontaneously as the result of their nutritional life. Such spontaneous action in the body I described[2] under the term Microkinesis in 1888; it appears to indicate that the nerve centres, or loci of brain-tissue, act separately and spontaneously; this microkinesis is absent in many imbecile infants, and in others with ill-nourished brain and sunken fontanelle. A little later temporary inhibition of movement through the senses may occur. At four or five months temporary inhibition of movements may result from the sight of an object, while this period of inhibition is followed by coördinated action; the head and eyes turning to the object, the hands are placed over it with the fingers extended, then the object is clutched and conveyed to the mouth. The inference is that at birth the nerve-centres act slowly and independently of one another, and that the time and order of their action, as determined through the senses, is a faculty of subsequent evolution. As the so-called voluntary or intellectual act of seizing the object followed upon a period of inhibition of movement, it may be inferred that during the period of no visible action the nerve-centres were being arranged functionally for the coördinated act; this functional arrangement among the nerve-centres (diatactic action[1]) during the period of no visible action appears to correspond to a neural mental act (act of psychosis). Such phenomena are absent in imbeciles.

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[1] For an account of the clinical methods of observation see article on page 122.

[2] Proceedings of the Royal Society, vol. xliv., and Journal of Mental Science, April.

We shall see that in some of the disordered conditions of older subjects the spontaneity characteristic of healthy babyhood may remain or return without good mental action. The period of inhibition of movement previous to the coördinated action may apparently be too short or absent, while in some cases the coördinated action is long delayed after the stimulus which produced it.[2]

In the normal child of school age the phenomena of imitation are of the highest importance, as this inborn faculty affords the first means of training the brain to a normal condition of coördinative power, while it is the physiological basis of methods of control by what is commonly termed "moral influence," and of implanting new mental processes.

The really useful aspect of school competition is when pupils of nearly equal capacity imitate that which is best in action as seen in their companions, and establish a good tone in the school. The sight of an object may produce a certain impression on the child's brain; the sight of action in another person tends to produce similar action in the child,—*i.e.*, similar muscular acts are seen to follow in the child, and we infer that the same nerve-centres are thrown into coördinated action in the child as produced the gestures in the person imitated. Sight of the gesture or movement imitated coördinates the child's brain-centres which correspond to those in action in the person imitated. These phenomena of imitation may be called instinctive, as depending upon inborn make of brain due to the antecedent common inheritance of man. The phenomena of imitation as characters of the brain imply impressionability, retentiveness, and potentiality for some forms of coördination.

Almost all our clinical knowledge of neural conditions is by study of movements in the body and the corresponding action among the nerve-centres,[3] while all expression of mental states and mental action is by movements and results of movement, whether such expression be effected by gesture, speech, or writing. We have to observe the part moved, while the time and the quantity of the movement indicate what is going on in the nerve-centre corresponding. Movements may be the direct result of stimulation through the senses, or they may occur without any known circumstances stimulating them, as when we see the spontaneous movements of infancy. Movement may be observed in a single part of the body or in many parts at once; each should be the subject of observation. When action in several parts is observed we may have a combination and a series of combinations of acts making up a complex phenomenon. Such series of acts may be classified as,—

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[1] "Diatactic Action among Nerve Centres." See Dr. Hack Tuke's Mental Science, Churchill & Sons, London.

[2] The Study of Children, The Macmillan Company, New York. See p. 125.

[3] See "Mental Faculty," a course of lectures on the growth and means of training the mental faculty, delivered in the University of Cambridge, published by the Cambridge University Press and Macmillan Company, New York.

(1) Uniform series of acts.
(2) Augmenting series of acts.
(3) Diminishing series of acts.
(4) Movements controlled or coördinated by circumstances.

Let us briefly consider each kind of series or mode of action.

A uniformly repeated set of movements is seen when the fingers are all opened and closed together; in the face when the frontal muscles and corrugators act together over and over again as a habit without variation, forming an athetoid series of acts; the same is seen in habitual grinning. Similar gestures may be often repeated.

An augmenting series of movements corresponding to a spreading area of nerve-action may be seen in a spreading smile or facial expression. In a burst of laughter the area of movement spreads over the face to the hands and the movements of respiration. When a question is asked of the pupil, the tongue may be protruded and the head held on one side, while the fingers fidget. Such spreading action, not coördinated, is antithetical to good intellectual function.

A diminishing series of movements is seen as the child quiets down after laughter or passion and is controlled to intellectual work.

In *coördinated action* adapted by circumstances we have a high-class function; but though work is accomplished thereby, we may see fewer movements and infer fewer nerve-acts than in the case of an augmenting series of acts which, while useless, is followed by more nerve-exhaustion than the good coördinated action; hence training the nervous, excitable children often lessens their exhaustion and weariness.

These modes of movement have been briefly defined; they will be referred to in giving descriptions of various classes of cases. Uniformity of action is often characteristic of brain-deficiency or defect; augmenting action characterizes the nervous and excitable children.[1]

Children with some neural defect or disorder may be classed as cases of neural defect or deficiency, or as nervous without known structural defect. Dull or backward children may be deficient in mental function from organic conditions, or, without being constitutionally defective in make of brain, they may present abnormal modes of neural action corresponding to modes of mental disorderliness. Various classes of defect may be combined in the case. Children who are dull or backward pupils may be either delicate or nervous, or both. Besides these classes, we meet with cases of disease and gross lesion of brain, as well as of disease of the throat, the heart, and other parts of the body which space will not allow me to deal with here.

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[1] The Study of Children. See cases.

Children of the Nervous or Neurotic Type.—Among the children commonly classed as "nervous children" are included those who sleep badly, are long in getting to sleep, talk at night and grind their teeth, are tired in the morning and fail in appetite for breakfast. These children are sometimes somnambulists, or suffer from night-terrors; those who suffer from headaches will be referred to separately. Such children often have a varying appetite, sometimes being most difficult to feed, at other times becoming voracious, needing extra meals. They often have a hacking cough and emaciate without lung disease; the urine is frequently dense but clear, with specific gravity 1025 to 1035, and laden with urea. In temperament they are usually affectionate and pleasing, but irritable and passionate at times, and often suffer from headaches; they are quick at learning, often precocious in their thoughts and expression. These nervous children are gregarious in social habits. In school they tend to congregate, and, being mentally quick, often finish their lessons before the others and play, while duller pupils plod on. They are imitative and excite one another. The amount of incoördinated mental activity and motor action often leads to exhaustion, and is best overcome by continuous quiet training. The individual is better at school, but their interaction on one another needs to be watched.

In bodily development the nervous child is usually well made, with a good head and good features, but tall and thin, more thin in the limbs and body than in the face. Various nerve-signs may be present, such as asymmetrical balance of the body, the head not erect, the spine not quite symmetrical, the feet unequally planted; when the hands are held out lordosis may appear, while the limbs are held at unequal height and neither on a level with the shoulder, the left usually being lower. The hands balance in the "nervous posture,"[1]—*vide ante,*—especially marked on the left side, the fingers twitching spontaneously. Facial expression is usually bright unless there be great exhaustion, when there is fulness of the lower lids. The eyes are well directed to objects, but go through many spontaneous movements like the fingers. Some explanation of the pathology of these cases may be found by considering the significance and the interaction of the points observed. The nervous child is usually below the normal weight. It is well known in biological study that a condition of low nutrition predisposes to reversion or a return to earlier modes of growth or action. The number of spontaneous or incoördinated movements in nervous children appears to me a reversion to the spontaneity of infant movements. The condition "nervousness" is a certain degree of weakness or lowered status of action in the nerve-centres, with spontaneity and lessened coördination. The weakness is indicated by lordosis and weaker power over muscles, and the spontaneous discharges of the nerve-centres produce finger-twitches and uncontrolled eye-movements. Certain extra movements or an irregular spreading area of action following a slight stimulus may be seen in facial grimaces, swaying of the body, or much movement of the head; too many nerve-centres are put in action in place of the few exercised in well-coördinated action. By producing a better condition of general nutrition and training the child to imitate good action in the teacher, much improvement may be effected. Finger-twitches may be flexor and extensor, produced by the muscles of the forearm, while the lateral twitches of the fingers are produced by the small intrinsic muscles of the hand, and appear more directly suggestive of disturbance in the highest centres. These twitching movements are to be distinguished from tremor, in which the displacement of the part in movement is much less, while the tremulous movement is regular in its time and rapid, often up to two hundred movements per minute. Athetosis, which indicates organic defect of the brain, is characterized by slow and uniformly repeated series of movements whatever the stimulus producing it, the movements not being coördinated by impressions through the senses, and therefore useless or purposeless.

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[1] See former article, p. 132.

Children who suffer from headaches are numerous among those of the neurotic type, especially among girls, and they mostly present some of the signs described as characterizing such cases, or else show signs of exhaustion. When a child says he has headache, but shows no nerve-signs or exhaustion, his general life and school work need not be greatly interfered with on account of his complaining of head pain. The most distinct point in causation is inheritance. Among the family of parents who have both suffered from migraine for many years some members are almost sure to develop the neurosis, and it is important to explain this in early life, that the children may, if possible, be brought up in the country and also to bear the pain which is their inheritance. These children should be protected from great fatigue; their feeding and management should be much the same as that described for nervous and exhausted children. Errors in refraction of the eye should be looked for and corrected when found. Children who suffer from headaches, as well as other nervous children, not uncommonly see spectral illusions. It may be distinct objects, as a man's head or an animal. I think that with children these illusions are generally stationary. They point out the place where they see them and say what they see, but do not often indicate them as moving. Such spectra may be taken by the child to be a real object, and he may be frightened at the sight.

Among my notes of fifty-eight cases of headaches in children, I find: seven years and under, boys fifteen, girls eight; eight to ten years last birthday, boys three, girls ten; eleven to fifteen years, boys seven, girls fifteen: total, boys twenty-five, girls thirty-three.[1]

Brain-fatigue and exhaustion are conditions of great importance in children, and are also very commonly met with. Weariness and inertness in action may be due to many circumstances. Much might be said as to the effects of muscular exercise, defective ventilation and diet, conditions of the blood and circulation, etc., in producing fatigue, but I am obliged to confine my remarks mainly to the status of brain-fatigue, its diagnosis and treatment. The lessened power of brain is indicated in lessened force and a lower power of coördination both in motor and in mental action, while irritability is manifested in certain fidgety extra movements, as in children of the nervous type. The signs of exhaustion are a lessened degree of facial expression, relaxation of the orbicularis oculi muscles with fulness under the eyes, drooping of the jaw with the mouth open without naso-pharyngeal obstruction, asymmetrical balance of the body and lordosis, unequal balance of the arms when held out, the hand falling in the weak posture of flexion with the metacarpus contracted or folded together. The exhausted child, as in slight cases of chorea, may present some spontaneous twitching movements of the fingers and eyes, while if of a low type of brain make there may be overaction of the frontal muscles with corrugation or over-smiling, together with fidgety extra movements when spoken to or required to perform some action. Response in action is slower than normal, and imitation of movements less exact in its coördination. Mental action is comparable to the defect in movement; it is slow and uncertain both in processes of thinking and in expression, while the tone of voice becomes low and monotonous. It is important to seek the sources of such exhaustion. The most frequent cause is want of regular and sufficient hours of sleep or deficient rest during sleep. Tooth-grinding is usually an indication that sleep is defective; usually the canines or the incisors are the teeth found flattened at their tips by trituration produced by coincident action of the masseter and temporal muscles with the pterygoids, all of which are supplied by the motor division of the fifth cranial nerve, whose sensory division supplies fibres to the meninges. Children of the neurotic type sometimes become exhausted by a great amount of spontaneous unguided thinking, constantly asking questions on points they cannot understand and puzzling over their own thoughts. They may appear to be precocious and clever children, but are too little occupied with a child's natural life and games. Regular school life, wisely directed, may be of the greatest benefit in removing such causes of exhaustion.

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[1] See International Clinics, 1895, vol. ii.

Chorea in children is a disorder frequently met with in well-developed but ill-nourished brains under the stress of circumstances. The main facts concerning this form of brain disturbance are:

1. The association with rheumatism and heart disease. These well-known points in etiology I will not consider further than to point out that impurity or irregularity in supply of blood to the brain often leads to head pain, want of sleep, mental distress, or mania, as well as delirium in fevers, uræmia, etc.

2. Chorea occurs in well-developed, highly evolved brains, and not at the youngest ages, and brain health may be recovered.

3. It is more frequent among girls than boys.

4. Almost all cases of chorea are under the normal weight for their age; the body weight continues to fall for a time, but commences to rise before the movements subside, and they usually cease soon after body weight becomes normal.

5. The condition is characterized by weakened force of the nerve-centres on the muscles and by spontaneous action.

6. The movements seen are but seldom coördinated by impressions from without, but the combinations of movements are the normal. Can any explanation be given why a well-developed child's brain, when ill-nourished, should under stress of the environment fall into a condition of disorder among the nerve-centres, resulting in their becoming weak in motor action and liable to constant spontaneous discharge of force? Such appears to be the problem. In 1887 I suggested the view, which I continue to hold, that chorea is a condition that may occur in a well-developed brain analogous to the mode of action described as normal in the infant brain.[1] In a normal brain at twelve years old the mode of function has changed greatly from the infantile condition; the separate spontaneous (motor) action of many nerve-centres has been replaced by quietness and adaptability for coördinated action If the larger mass of brain at twelve years resumes the infantile mode of action, the individual nerve-centres becoming as weak as in the infant, and again acting separately and spontaneously, the movements of chorea will appear. The choreic brain appears to me to be one in which the mode of action is for the time infantile; the greater number of movements seen in chorea, as compared with the microkinesis of infancy, corresponds to the greater number of motor nerve-centres in the larger brain. We have a condition of reversion to the infantile mode of action, and the coördinative power previously acquired is for the time being lost; there is a dissolution of the acquired evolution; hence the loss of speech, which is an acquired coördinated mode of action. Speech may be lost independent of choreic movements of the tongue. These facts closely correspond with what has been described in conditions of "nervousness" which are lesser degrees of dissolution of acquired evolution. We might arrange a graded group of fifty children,—at the one end those simply "nervous and over-mobile," then those with manifest twitches, succeeded by children convalescent from chorea but not steady, up to all forms of well-marked cases of the affection: paralytic chorea, hemichorea, or general affection of all parts of the body.[2]

Mental Examination of Children.—It is shown in Table I. of the former article that, of the children with abnormal nerve-signs, forty per cent. of the boys and forty-two per cent. of the girls were dull and backward pupils; while of the children reported by the teachers as dull, 61.8 per cent. of the boys and sixty per cent. of the girls presented nerve-signs. In these facts we see evidence of the connection of brain-disorderliness in motor action and adaptability for mental function. It follows that in the clinical examination of a case with any abnormal brain-condition, or any defect in development of the body, the mental status should be looked to. In conducting the mental examination of a child it is convenient to commence with some general conversation on simple subjects,—*e.g.*, his life at school or at home, in play and at work; what he reads, his companions and amusements. The faculty of speech is thus ascertained and some idea of the extent of his vocabulary. Various defects of speech may be found; there may be ill-regulated intonation, the voice at times almost dying away; there may be thickness of utterance, often in part due to naso-pharyngeal obstruction; a few words may be spoken in reply to a question without affording an answer; the question may be simply repeated without any reply. In many other particulars speech or utterance may be defective. Conversation with the child may show whether, according to his age, he can give a fair account as to how he would travel from one place to another, as to how he went to the sea for his holidays, what he must get to write a letter and how it gets to its destination, where articles of food come from. Particularly notice in conversation whether the child makes comparisons or exercises the faculty of judgment. "Did you get to the sea-side in the same way as the letter that said you were coming?" The behavior of the child with common objects may show much as to his modes of dealing with his surroundings. Does he turn over the leaves of a picture-book and speak of the objects, or point to them? Is he destructive? Does he turn over page after page, or soon throw the book away from him? Let him have your purse and count the money, first the number of coins, then count their value. A child who has been taught may add sums set on paper, and cast up the value of money, though he is unable to count objects presented to him or find the coins corresponding to the written sum. In children with little or no speech it is more difficult to ascertain whether a judgment is formed. A boy liked to play with a ball, and called it "ball;" when shown the ball and a spherical glass held out towards him, one at a time, he spontaneously said, "ball," "glass," "glass-ball," which indicated a mental process. A one-ounce weight was placed in his right hand, and two ounces in his left; he moved them up and down and said, "heavy," holding out the latter weight; such a judgment was determined by his muscular sense. Thus both the faculty of muscular sense and counting are important as tests; the two are much allied, and each individual defect detected suggests a line on which methods in training should be directed. Place similar coins on the table, let the child move each in turn with his hand, and thus count them; again, place the coins at equal distances in a line and let him count them with his eyes. In the former case his brain appreciates (counts) his hand or his hand- and eye-movements, in the latter case it is only his eye-movements as they turn to each coin that he counts; the former method is easier for the child. He may similarly count objects in the room, pointing to them, or only by looking at them.

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[1] Anatomy of Movement, The Macmillan Company, New York. See p. 98.

[2] International Clinics, 1897, vol. i.

The appreciation of weights has been referred to; it is well to use metal weights whose weight is proportional to their size, and also other objects of similar size whose weight varies, such as pill-boxes, empty or filled with plaster.

Another useful test is the naming of colors or matching those that are similar. The sense of temperature should be tested. Some children of defective brain-power are very anæsthetic, and allow a tooth to be pulled out without any expression of pain.

The clinical examination of dull and backward pupils shows the close association between the mental and the physical conditions described. Of the main classes of defect of childhood[1] brain disorderliness, indicated by abnormal nerve-signs, is a more frequent accompaniment and apparent cause of mental dulness than defect in bodily development. Comparison of schools when good physical training is provided with others where there is none shows that in the latter there is a higher proportion of pupils with "nerve-signs" and dull pupils. All means available for removing these motor indications of brain incoördination are useful in preparing the way for mental culture. These dull children must appear under different aspects to the parents who see the whole life of the child, and whose interests lie in their future success as well as their present well-being; the teachers have to train in the school-room; the physician is consulted as to some physical ailment in the child; our business is to look at the child from every aspect and give prognosis and advice accordingly.

Inaccuracy in writing and spelling may be due to not looking with exactness; if the eyes are not trained to fix well, if the child always looks towards an object and its parts by turning the head only and not moving his eyes in the orbits, the mental impressions received will be too few. Such children are bad observers of the printed page, an object demonstrated, or the black-board; in the latter mode of instruction the boy not trained to look well may fix his eyes on the face of the teacher instead of the black-board.

Motor action and mental action in response to a command or a question may be too rapid as a habit, not allowing sufficient time for the impression received to prepare the brain for the reply. Thus nervous children sometimes make absurd mistakes when those of a slower habit answer correctly. A moment of quietness is needed before the reply. On the other hand, expression may be long delayed after the question, so long as to form part of the reply to a subsequent question, without any real failure of mental action. An expression of intelligence passing over the face may indicate mental action, and a second question may elicit a correct reply.

Children without any mental dulness may be backward and troubled with their lessons from inaptness, not from want of real ability, their habits of neural action not yet being fully evolved or coördinated to intellectual action. It often happens in children, such as were described as of the nervous type, that they have much spontaneous movement, or microkinesis, remaining from infancy,—brain-incoördinated action expressed both in motor and in mental processes. A question asked of the quiet child just produces the answer; but addressed to the nervous child with much spontaneous brain-action, the thoughts present run on and intermingle with the expression of a reply. This kind of brain-action has been referred to as a source of fatigue. These are the most hopeful among the backward pupils; their real mental status must be judged of when they are at their best, from the expression of the best judgments they make, not from their mistakes.

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[1] See Statistical Journal, March, 1896.

Teachers tell us that a principal cause of dulness is inattention; the child who stands motionless and without many passing thoughts is more ready to answer questions, read, or prepare a correct exercise or sum than the child with many spontaneous movements, which are increased on any mental stimulus; the latter is called fidgety. These extra movements indicate a wide area of brain-action, augmented rather than coördinated by any stimulus, which is inimical to mental precision and may soon exhaust the child. It is found in many cases that such extra movements occur when the child is spoken to and subside when he is controlled through his eye instead of his ear. Cases as here described, if otherwise normal, may be of good prognosis; the girls of this type are more likely to do badly if neglected and allowed to become exhausted. It is necessary to watch the conditions of health, feeding, rest, and, while pursuing a quiet training, wait for further evolution of brain-power and meet difficulties as they may arise.

Conditions of chronic exhaustion in children not ill developed may lead to much mental dulness, as in school-children who work long at home or keep late and irregular hours. Deafness may also be a cause of dulness without brain-defect.

In the former article certain groups of children are defined, graded as to mental dulness or defect of brain and body. Children of the nervous type, those exhausted, and those who suffer from headaches may be of normal construction, but have fallen into a condition of neural disorder from which they may recover under care. In minor cases of defect the relative importance of the signs observed is fairly indicated in Tables II., III., while it is shown in Table IV. that such relations vary with sex in the age-groups, nerve-signs being most associated with mental dulness under seven years of age. Some combinations of defectiveness are of grave significance; when abnormal points in development of the body are accompanied by low nutrition and abnormal nerve-signs, we find that 53.7 per cent. of the boys and 50.6 per cent. of the girls were dull pupils. Cases are also met with in which the degree of defect of body and brain leads to a bad prognosis for the mental and social life of future years, which can only be combated with any hope of success by well-directed training, commenced early and long continued. The following brief notes are of cases seen in Poor Law institutions (district and separate schools), 1888–91:

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(387.) A girl aged ten years. Standard I. Head normal, but palate very narrow; veins on forehead and bridge of nose large; expression of face not good; frontal muscles overacting finely, making the forehead dull; mouth kept open; hands when held out balance in the "weak posture" with lordosis of the spine; was reported to have learnt but very little and that but slowly. The nose and throat should be examined.

(1842.) A girl aged twelve years. Head very small, circumference nineteen and one-half inches; bridge of nose wide; expression of face dull and over-smiling, looks lacking in intelligence; mouth open; attempts to make replies to questions, but her speech is unintelligible. She appears to be a small-headed girl of weak mental power.

(1691.) A girl aged thirteen years. Head of sufficient size; palate narrow; lips thick; a big, heavy, strong girl; lacking in intelligence of expression; frontal muscles overacting; eyes not well directed to objects; healthy, but never did any work in school, and interfered with the other pupils; works well in the laundry and kitchen.

(791.) An infant girl aged three. Head small and conical, circumference eighteen inches, transverse measurement eleven and one-half inches; ears outstanding; face large in proportion to the small head; a fat child with cold blue limbs; no facial expression; could walk and say a few words; the nasal bridge was sunken with discharge from the nose; a very low type of development and brain-power.

A boy attending a day school and an out-patient at hospital, age eight years, in infant class. Head large, coarsely built, not lumpy, circumference twenty-two inches; palate high and arched; right ear defective in make, the left normal; chronic chorea from birth, affecting the face, eyes, and limbs. He could walk, but sometimes fell down. His nutrition was good, heart normal, and he had never been laid up with illness. His mother wished him to attend day school; he had never learnt his letters, but had only been at school three weeks.

A boy fifteen years of age; very low down in one of our public schools; very short; fairly muscular; weight six stone; head rather small, twenty inches in circumference; tonsils large; no nasal obstruction; expression rather blank; full under the eyes; frontal muscles overacting, sometimes with corrugation; he stands well; eye-movements good; no rigidity; knee-jerks normal; speech fair, monotonous, and some delay before replying; general movements and imitation of movements good; cannot distinguish weights; writes fairly, but slow; good at gymnastics; likes foot-ball; is generally liked in the school; has a poor memory; takes both Latin and French at school, but is very backward. It was advised that he should give up Latin, and that he was probably unfitted for the high position of commercial responsibility which had been prepared for him.

I shall now dwell in more detail on some of the indications of brain deficiency and the clinical examination of children feeble and wanting in mental power. The worst among the abnormal nerve-signs described are those classed as repeated uniform movements, such as athetosis, frontal muscles overacting with or without the corrugators, repeated grinning, together with absence of facial expression and slow, feeble, inaccurate modes of response. In examining a case of defective brain all pathological conditions should be looked for. Examine each side of the body separately; there may be indications of hemiplegia or some defect in power or in coördination on one side only, or one side more than the other. Let the child pick up a pin with either hand or button and unbutton his glove. Let him imitate the movements of your fingers in either hand and in both together. The child's imitation may be good with both hands in imitation of both of yours, but not equally good in moving one side only in correspondence to one of yours. See if there be any rigidity or hypertonicity in the limbs at the wrist or ankle as you make passive movements. In the lower extremities does the leg swing freely at the knee like a pendulum without muscular restraint? Hypertonicity is usually found in congenital hemiplegia. In congenital hemiplegia at ten years and upward the trophic effect of the lesion is often indicated by shortness of the radius and ulna, as compared with the opposite side, and the limb may be altogether smaller. Inquire carefully for indications of epilepsy and *petit mal;* casual observation is not enough; those in charge of the child should be instructed to observe him constantly. In these cases it is desirable to examine the eyes, the hearing, the throat, and the nose; also the heart. In cases of congenital defectiveness it is not uncommon to find a hernia. In social habits these defective children are solitary, not playing with other children or joining in their games; they sometimes form low companionships, tease animals, and may be uncleanly, due to want of good training.

The treatment and management of children with neural or mental disorder must in part fall as a responsibility upon the physician, who may well take his part in consultation with the teacher in the mental guidance and culture of the child. In the former article I dwelt on the duties of the medical officer of a school or institution; the recommendations in care of individual children may now engage attention.

Nervous children and those with brain defect are often delicate and dull. (See Table I.) The rooms they occupy, both for living and in school, should be light and well ventilated. The child's clothing should be appropriately arranged, securing a uniform layer of warm air over the body and the arms and feet, while not constricting his movements. Uniform times for rising and sleeping should be adhered to with regularity, while some children require to lie down some portion of the day, and those who suffer at times from bad headaches or signs of exhaustion need an occasional day devoted to complete rest, while active at other times.

It is convenient to draw up a time-table for the child showing the hours of each kind of work, occupation, play, rest, and meals. This adds accuracy to the management adopted, so that results can be seen, and the method may be altered from time to time. When nerve-signs are more prevalent, some temporary relaxation may be needed. The feeding of nervous children is often a great difficulty; they need much food, but are capricious in appetite and dawdling at meals, so that they eat insufficiently unless urged on and watched at meals. The diet should be varied; fat foods aid increase of weight when this is deficient; well-boiled suet pudding, plenty of milk, —say two pints a day,—or cocoa or coffee, mostly made with milk, may be used as beverage; all alcohol should be avoided. At lunch, biscuit, chocolate, or milk may be given; bacon, eggs, fish, etc., at breakfast; soup, meat, pudding at dinner, and a small portion of meat after the school-day are usually needed for the nervous child twelve years of age. Cod-liver oil, iron, and malt may be useful.

Treatment and management should be guided by diagnosis and description of the points in the case. The individual nerve-signs should be pointed out to the teacher in charge of the child, requesting that in daily training care may be taken to avoid increasing them, and to remove them in detail, mainly by the influence of imitation and cultivation of perfect balance and movement. We should not simply direct that fatigue be avoided, but point out the altered appearance in the face, the fulness under the eyes, the extra-movements and signs of excitement which express the commencement of fatigue.

Children of the nervous type need guidance and control rather than stimulus, while those of the defective class need stimulation and guiding throughout the day; hence their care is more laborious, as they need active help equally in work and in recreation as well as in their social life. Neglect or delay in the education and training of defective children leads to an inferior status, mental, moral, and social. It is a mistake only too common to wait and do nothing. The physician, in seeing the child, at least at the commencement of each school term after the holidays and at the conclusion of a term of work, can compare the conditions of the child from time to time, pointing out improvements effected and further points for attention, whilst guarding against signs of exhaustion as the result of the training given.

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